ONLINE - ISSN : 2583-813X
P - ISSN: 2581-8201

GMC PATIALA

JOURNAL OF RESEARCH AND MEDICAL EDUCATION

2025
January to June
Volume: 08 | Number: 01
Publication: Half Yearly

Editor-in-Chief
Dr. Balwinder Kaur Rekhi
Professor
Department of Anaesthesia
Govt Medical College Rajindra Hospital
Patiala

Official Publication of
Journal Club Government Medical College Patiala Punjab India
Place of Publication: Patiala (Punjab) India
Website:http://gmcpatiala.edu.in

GMC PATIALA JOURNAL OF RESEARCH AND MEDICAL EDUCATION
An official Publication of Journal Club, Government Medical College Patiala Punjab India
P-ISSN: 2581-8201 | ONLINE-ISSN : 2583-813X
Volume: 08 Number: 01 January to June 2025 Publication: Half Yearly

Contents

AIMS AND SCOPE

Journal of Research and Medical Education is an official publication of Journal Club, Government Medical Collage, Patiala, Punjab, India. The journal is open access, high quality, peer reviewed and published at half yearly intervals. The journal aims to demonstrate high quality research and knowledge in medical sciences. It also aims to promote evidence based medicine and provide a global platform for clinicians, researchers and healthcare professionals to share their findings, experiences and perspectives. The journal publishes original research papers, review articles, case reports/case series, editorials and reviews on various aspects of medicine, such as clinical practice, research and education, ethics, innovation and policies.

This journal covers the various medical and surgical specialties and super specialties of the medical field along with other Allied branches dedicated to human health, Medical education and research and administration.

Subscription Information

Members of the journal club will receive the journal free of cost

Annual subscription rates for Non-Members and institutions:-

Personal: India Rs. 1000/-, Rest of the World US$ 150 or equivalent

Institutional: India Rs. 3500/-, Rest of the World US$ 300 or equivalent

Subscription orders and payment should be drawn in favour of journal Club Government Medical College Patiala payable at Patiala by Demand Draft / Bank Cheque (Add Rs. 100/- for outstation cheque)

Claims for Missing Issue

A copy will be sent free to the member/ subscriber provided the claim is made within 2 months of the publication of the issue & self-addressed envelope of the size 9" X 12" is sent to the Editor (Those who want the journal to be dispatched by Registered Post, must affix Rs. 60/- worth postage stamps.

Dr. Balwinder Kaur Rekhi, Editor-in-Chief, Publisher Dr. Raja Paramjeet S. Benipal and Mohit Roy Mangla, Printer of "GMC Patiala Journal of Research and Medical Education", Patiala, English, Half Yearly, printed at Phulkian Press Pvt. Ltd., C-165, Focal Point, Patiala, on behalf of Journal Club Government Medical College Patiala Punjab India and published at Department of Radiation Oncology, Rajindra Hospital, Govt. Medical College, Patiala-147001

Issuance :
Half Yearly

First Volume of the Journal Published in 2018

Published by:
Dr. Raja Paramjeet S. Benipal on behalf of Journal Club Government Medical College Patiala Punjab India at Government Medical College (Rajindra Hospital) Patiala-147001 (Punjab) India
Phone : 96469-12340

Printed by:
Mohit Roy Mangla on behalf of Journal Club Government Medical College, Patiala Punjab India-147001

Printed at:
Phulkian Press Pvt. Ltd.
C-165, Focal Point, Patiala-147001 (Punjab) India
Phone: +91-9914430345
Email: pressphulkian@gmail.com

ISSN Number : P 2581-8201
Online No. : 2583-813X
GMC Patiala Journal of Research and Medical Education (Online)

Volume of Distriubution:
200 Copies of each volume

Funding Bodies:
Journal Club Government Medical College Patiala Punjab India.
Donations from Philanthropists and advertisements in the journal

Address for submission of articles:
Online Soft Copy:
gmcpatiala.edu.in
Address request for reprint or further information relating to any article may please be made with Editor.

Submission of all papers to the journal is understood to imply that it is not being considered for publication elsewhere.

Submission of multi-author paper implies that the consent of each author has been taken in this journal.

Researchers/Authors should adhere to publication requirements that submitted work is original, is not plagiarized and is not unethical, and has not been published elsewhere.

Every effort has been made not to publish any Inaccurate or misleading information. However, the Editor-in-Chief and the Editorial Team accept no liability in consequences of such statements.

Submission Information:
Members of the Journal Club will receive the journal free of cost.

Non-Members Personal Rs. 1000/-
(Abroad US$ 150 or Equivalent)

Institutions:
Rs. 3500/- (Abroad
US$ 300 or Equivalent
Subscription should be addressed to Journal Club GMC Patiala Payable at Patiala.

We accept bank drafts and cheques (for outstation cheques add Rs. 100/- as bank Charges.) A copy will be sent free of cost to the members/ subscriber provided the claim is made within 2 months of the publication of the issue & self-addressed envelope of the size 9"x12" is sent to the Editor (Those who want the journal to be dispatched by registered post, must affix Rs. 60/- worth postage stamps)

Editorial

ARTIFICIAL INTELLIGENCE IN MEDICAL SCIENCE

Dr. Balwinder Kaur Rekhi

Artificial Intelligence (AI) was first conceptualized in 1956; however, significant advancements have occurred primarily over the past twelve years. In the medical field, AI has become instrumental in reviewing vast numbers of medical records, thereby enabling faster treatments and improved patient outcomes. AI simulates human intelligence using computer systems, allowing machines to learn, predict, analyse, draw conclusions, and even self-correct over time. This technology has been developed to address various medical challenges, including planning, imaging, speech recognition, and learning specific traits. AI systems are trained on large datasets to generate accurate predictions and assist in solving complex problems with high precision.[1,2]

AI helps healthcare professionals reduce the time spent on documentation by digitally storing patient data and creating comprehensive databases. These databases can then be used for diagnosis, treatment planning, and ongoing patient care. Depending on specific healthcare needs, medical experts collaborate with software and hardware professionals to develop customized platforms for data collection and routine healthcare tasks. Generic software systems are being tailored for specialized applications, and modules for diagnosis, treatment, and post-treatment care are being developed accordingly. However, the effectiveness of these AI systems largely depends on the quality and analysis of the collected data.

AI enhances the creativity and capabilities of doctors and surgeons. Intelligent machines can understand human language and efficiently process various types of data—including text, images, bioinformatics, and even financial transactions—to make informed and accurate decisions.[3] AI supports precision surgery by providing critical information during operations. It also aids in gathering and analysing sufficient patient data to predict outcomes and reduce risks associated with surgeries such as joint replacements, as well as shorten hospital stays and improve recovery rates.[4]

Advancing Healthcare through Artificial Intelligence-

Artificial Intelligence (AI) is driving a wave of disruptive innovation across the medical field, transforming traditional practices through efficient data analysis, streamlined workflows, and digital automation. It enables faster, more consistent outcomes—from digital consultations to medication management—enhancing both patient care and clinical efficiency.[5]

Medicine:

AI supports advancements in diagnosis, personalized treatment, and drug development. By accelerating clinical trials and improving patient monitoring, it enhances decision-making and streamlines the often time-consuming medical processes.

Surgery:

AI-assisted surgeries enable data-driven precision at every stage of the procedure. It supports clinical decision-making, improves surgical outcomes, and enhances surgeon workflow, particularly in complex cases.

Radiology:

In radiology, AI improves the accuracy and consistency of diagnostic imaging. Real-time learning and interpretation of complex data lead to better outcomes and faster recovery, with AI systems capable of adapting to procedural variables.

Hospital Administration:

AI is revolutionizing hospital operations by digitizing medical records, automating data management, and improving real-time access to patient information. This results in more accurate diagnoses, efficient hospital workflows, and better coordination among staff.

Cardiology:

AI plays a crucial role in early detection and prevention of cardiac conditions. It helps identify risks such as valve blockages and provides real-time insights into heart function, reducing the likelihood of sudden cardiac events. From diagnosis to discharge, AI is becoming integral to patient care, offering smarter, faster, and more effective healthcare solutions.

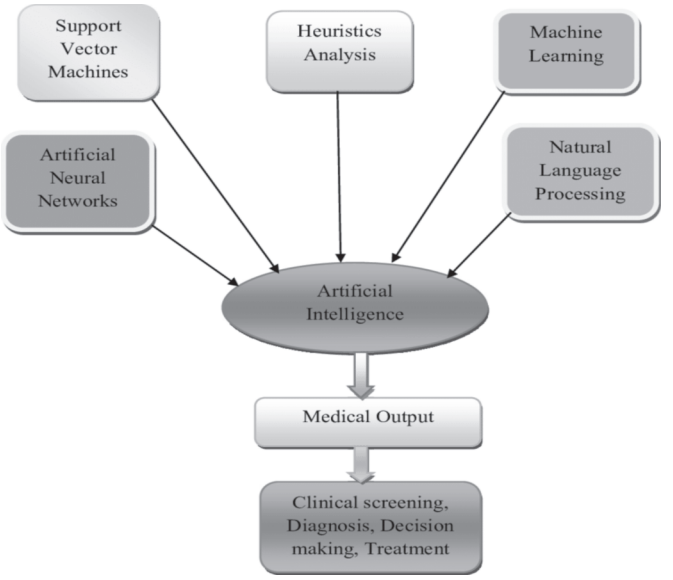
AI in Critical Care:

Machine learning (ML) is driving innovation in critical care through tools like predictive models, early sepsis detection, automated documentation, and AI-assisted imaging. These technologies analyze vast clinical data to support early intervention and reduce clinician workload. Despite some regulatory approvals, adoption remains limited due to concerns over transparency, reliability, and integration into clinical workflows. Most ICU applications are still experimental and narrowly focused. AI-powered systems show promise in detecting

patient deterioration and improving imaging analysis, especially in pulmonary care. However, clinician trust, data variability, and workflow compatibility remain key hurdles. For widespread use, AI must enhance—not replace—clinical judgment.

Process chart of Artificial Intelligence in the medical field-

AI is effectively used in ECG analysis, cardiac and respiratory monitoring, lab tests, imaging, and anaesthesia. By processing patient data, AI aids diagnosis, decision-making, and treatment. Combining techniques like neural networks and natural language processing, AI understands clinical data and human communication to support doctors in real-time, improve surgical outcomes, foster innovation, and enhance efficiency with minimal risk. Following figure shows the process chart of Artificial Intelligence in the medical field.[6]



Future Prospects and Limitations of Artificial Intelligence in Medical Science-

Artificial Intelligence (AI) is rapidly advancing as a transformative force in medical science, promising significant improvements in diagnosis, treatment, and healthcare delivery. Future prospects include enhanced personalized medicine through predictive analytics, early disease detection using large-scale data, and AI-driven robotic surgeries that increase precision and reduce human error. AI-powered telemedicine and virtual health assistants are set to improve healthcare accessibility, especially in remote and underserved regions. Moreover, continuous learning algorithms will adapt to evolving medical knowledge and patient variability, enabling more dynamic and responsive care.

Despite its potential, AI faces notable limitations. The "black box" nature of many AI models restricts transparency and interpretability, causing reluctance among healthcare providers to fully trust these systems.[7] Data privacy and security concerns are paramount given the sensitive nature of medical information. Furthermore, AI models may inherit biases present in training data, potentially perpetuating health disparities.[8] Integration into existing clinical workflows remains a challenge, complicated by regulatory hurdles and the need for robust validation across diverse patient populations. Importantly, AI is designed to augment rather than replace clinical judgment, necessitating ethical deployment to maintain patient trust and ensure equitable healthcare outcomes

In conclusion, while AI offers groundbreaking opportunities for advancing medical science, addressing these technological, ethical, and regulatory challenges is crucial for its responsible and effective adoption.

References-

1. Haleem A, Javaid M, Vaishya, R. Industry 4.0 and its applications in orthopaedics. J ClinOrthop Trauma. 2019: 10(3): 615-616.
2. Jha S, Topol EJ. Information and artificial intelligence. J Am CollRadiol. 2018; 15(3 Pt B): 509–511.
3. Murdoch TB, Detsky AS. The inevitable application of big data to health care. JAMA. 2013; 309(13): 1351–1352.
4. Caocci G, Baccoli R, Vacca A, Mastronuzzi A, Bertaina A, Piras E, et al. Comparison between an artificial neural network and logistic regression in predicting acute graft-vs-host disease after unrelated donor hematopoietic stem cell transplantation in thalassemia patients. Exp Hematol. 2010;38(5): 426–33.
5. Haleem A, Javaid M, Khan IH. Current status and applications of Artificial Intelligence (AI) in medical field: An overview. Curr Med Res Pract. 2019;9(6): 231–7.
6. Hai TS, Thuy NT. Image classification using support vector machine and artificial neural network. Int. J. Inf. Technol. Comput. Sci. 2012; 4: 32–38.
7. Samek, W., Wiegand, T., & Müller, K.-R. (2017). Explainable Artificial Intelligence: Understanding, Visualizing and Interpreting Deep Learning Models. arXiv preprint arXiv:1708.08296.
8. Obermeyer Z, Powers B, Vogeli C, Mullainathan S. Dissecting racial bias in an algorithm used to manage the health of populations. Science. 2019;366(6464): 447–53.

Original Research Article

COMPARATIVE EVALUATION OF INTRAVENOUS LABETALOL, DEXMEDETOMIDINE AND LIGNOCAINE FOR ATTENUATION OF HEMODYNAMIC RESPONSE TO PNEUMOPERITONEUM: A RANDOMIZED CONTROLLED STUDY

BalwinderKaur Rekhi, Parmod Kumar, Harsimran Kaur

Corresponding Author : Dr. Harsimran Kaur
manocha.harsimran@gmail.com

Abstract

Background and Objective:

Laparoscopic cholecystectomy, though minimally invasive, is associated with significant hemodynamic stress responses due to pneumoperitoneum and anaesthetic interventions. Effective attenuation of these responses is crucial, particularly in patients with cardiovascular risks. This study compares the efficacy of intravenous labetalol, dexmedetomidine, and lignocaine in controlling these responses.

Material and Methods:

In this prospective, randomized controlled study, 150 ASA I–II normotensive patients aged 16–60 years undergoing elective laparoscopic cholecystectomy under general anaesthesia were randomized into three groups (n = 50 each). Group L received labetalol 0.25 mg/kg in 200 ml NS over 10 minutes before induction, Group D received dexmedetomidine 0.5 µg/kg in 200 ml NS over 10 minutes before induction, and Group Lox received lignocaine 1.5 mg/kg as bolus 3 minutes before induction. Hemodynamic parameters (HR, SBP, DBP, MAP) were recorded at baseline, induction, laryngoscopy, and at 3-minute intervals up to 30 minutes post-intubation.

Results:

Dexmedetomidine demonstrated the most consistent and significant control of heart rate and blood pressure throughout the intraoperative period. Labetalol effectively maintained blood pressure but was less effective in controlling heart rate. Lignocaine showed moderate efficacy in attenuating hemodynamic responses. Adverse events were minimal: hypotension occurred in 2% of patients in Group L, and bradycardia occurred in 10% of Group D. No adverse events were noted in Group Lox.

Conclusion:

Dexmedetomidine was superior in maintaining hemodynamic stability during laparoscopic cholecystectomy, with labetalol serving as an effective alternative for blood pressure control. Lignocaine, while safe, was less effective in managing acute hemodynamic fluctuations. Dexmedetomidine may be preferred in patients at risk of exaggerated sympathetic responses.

Keywords:

Hemodynamic response, Laparoscopic cholecystectomy, Pneumoperitoneum

Introduction

Advancements in surgery and anaesthesia, including the adoption of minimally invasive techniques and faster-acting drugs, have made day care surgeries like laparoscopic cholecystectomy increasingly common. This procedure offers numerous benefits over open surgery, such as reduced postoperative pain, shorter hospital stays, faster recovery, and fewer complications like ileus.[1]

However, laparoscopic cholecystectomy, typically performed under general anaesthesia, is associated with significant haemodynamic stress responses due to laryngoscopy, intubation, and pneumoperitoneum. The insufflation of carbon dioxide to create pneumoperitoneum raises intra-abdominal pressure (IAP), leading to systemic effects:

- Cardiovascular: IAP <15 mmHg may increase cardiac output via splanchnic autotransfusion, whereas IAP >15 mmHg can reduce venous return and cardiac output. Vagal stimulation can also trigger bradyarrhythmias or cardiac arrest.[2,3]
- Respiratory: Elevated IAP reduces lung volumes and increases airway pressures.[4]
- Neurological: Hypercapnia and elevated intra-abdominal pressures may raise intracranial pressure, compromising cerebral perfusion.[5]
- Positioning: Head-up tilt lowers venous return and BP, while head-down improves preload but may impair ventilation.[6]

These transient but unpredictable haemodynamic changes are generally well tolerated in healthy patients but pose a risk in those with cardiovascular comorbidities. Therefore, blunting the stress response is crucial to ensure intraoperative stability and improve outcomes.

Various pharmacological agents such as beta blockers, opioids, calcium channel blockers, benzodiazepines, and alpha-2 agonists are used to attenuate these responses.[7] However, each has limitations, and no single agent has proven ideal. This study aims to compare the efficacy of intravenous labetalol, dexmedetomidine, and lignocaine in attenuating the haemodynamic response to pneumoperitoneum during laparoscopic cholecystectomy.

Material and Methods

After obtaining ethical committee clearance, this prospective, randomized controlled study was conducted in the Department of Anaesthesia at Government Medical College, Rajindra Hospital, Patiala. A total of 150 ASA I–II normotensive patients aged 16–60 years undergoing elective laparoscopic surgery under general anaesthesia were enrolled and randomized into three equal groups (n = 50) using the closed-envelope method.

Sample size was calculated to detect a mean heart rate difference of 6.9 bpm with a standard deviation of 12.3, 95% confidence interval, 80% power, and $\alpha = 0.05$, resulting in 50 patients per group.

- Group L received IV labetalol 0.25 mg/kg in 200 ml normal saline over 10 minutes before induction.
- Group D received IV dexmedetomidine 0.5 µg/kg in 200 ml normal saline over 10 minutes.
- Group Lox received IV lignocaine 2% 1.5 mg/kg as a bolus 3 minutes prior to induction.

Patients were preloaded with 10 ml/kg of crystalloid and monitored with ECG, NIBP, and SpO_2. Standard premedication included glycopyrrolate 4 µg/kg and butorphanol 20 µg/kg. Anaesthesia was induced with propofol 1.5 mg/kg and succinylcholine 1.5 mg/kg, and maintained with isoflurane, nitrous oxide, oxygen, and vecuronium.

Hemodynamic parameters (HR, SBP, DBP, MAP, SpO_2, $EtCO_2$) were recorded at baseline (T0), at induction (Ti), laryngoscopy (TL), and every 3rd minute up to 30th minute post-intubation. In Groups L and D, values were also recorded every 2 minutes during the 10-minute infusion period. Hypotension or bradycardia (≥20% decrease from baseline) was noted and treated accordingly.

Statistical Analysis

Data were analyzed using descriptive statistics (mean, SD, median, and percentages) and inferential tests. Categorical variables were compared using the Chi-square test, while differences between group means were assessed using one-way ANOVA, followed by Tukey's post-hoc test for multiple comparisons. Paired t-tests were used for within-group pre- and post-intervention comparisons. A p-value < 0.05 was considered statistically significant.

Results

This study compared demographic and preoperative characteristics (age, gender, weight, ASA status) across

three intervention groups (L, D, Lox) undergoing laparoscopic cholecystectomy. No significant differences were found, indicating comparable baseline profiles and minimizing selection bias as indicated in Table 1

Table1: Demographic Data

Parameter	Group L (n=50)	Group D (n=50)	Group Lox (n=50)	p-value (Overall)
Age (years)	42.72 ± 12.32	40.90±12.08	39.74±11.02	>0.05 (NS)
Gender (% Female)	84%	82%	74%	>0.05 (NS)
Weight (kg)	63.28±6.91	64.02±7.26	63.38±5.67	>0.05 (NS)
ASA Grade I (%)	86%	82%	92%	>0.05 (NS)
ASA Grade II (%)	14%	18%	8%	>0.05 (NS)

Time of Creation of Pneumoperitoneum (Pn)-

Table2: Demographic Data

Time (Pn)	Group L		Group D		Group Lox		Overall	p value		
	Patients	%	Patients	%	Patients	%		L vs D	L vs Lox	D vs lox
T6	2	4%	1	2%	1	2%	0.341 (χ^2= 2.151) NS	0.935 (χ^2= 3.617) NS	0.397 (χ^2= 9.449) NS	0.988 (χ^2=2. 2.213) NS
T9	7	14%	7	14%	3	6%				
T12	38	76%	36	72%	41	82%				
T15	3	6%	6	12%	5	10%				
Total	50	100%	50	100%	50	10%				

Table 2 shows that in our study, the number of patients in whom pneumoperitoneum was established at 6, 9, 12, and 15 minutes after laryngoscopy were as follows: 2, 7, 38, and 3 in Group L; 1, 7, 36, and 6 in Group D; and 1, 3, 41, and 5 in Group Lox. There was no statistically significant difference in the timing of pneumoperitoneum in three groups (p>0.05) and in majority of patients pneumoperitoneum was created at 12th minute after induction.

Comparison of Vitals at T12 (Pneumoperitoneum)

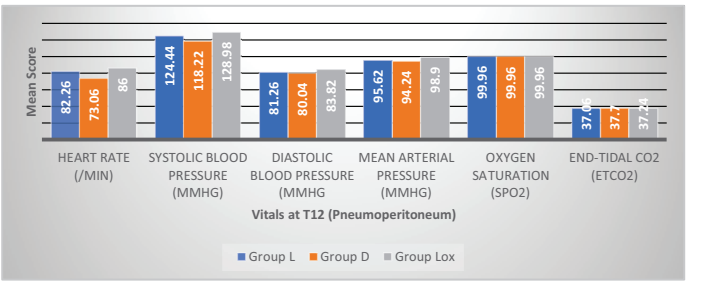
Table 3: Comparison of Vitals at T12 (Pneumoperitoneum)

	Group L	Group D	Group Lox	Overall	p value		
					L vs D	L vs Lox	D vs lox
Heart Rate (/min)	82.26±7.50	73.06±7.78	86.00±9.37	0.001 (F=32.527) HS	0.001 (t=6.020) HS	0.030 (t=2.205) S	0.001 (t=7.514) HS
Systolic BP (mmHg)	124.44±11.02	118.22±8.83	128.98±8.57	0.001 (F=16.039) HS	0.002 (t=3.114) S	0.024 (t=2.300) HS	0.001 (t=6.184) HS
Diastolic BP (mmHg	81.26±7.78	80.04±7.53	83.82±6.50	0.033 (F=3.499) S	0.428 (t=0.797) NS	0.077 (t=1.784) NS	0.008 (t=2.687) S
Mean Arterial Pressure (mmHg)	95.62±8.20	94.24±7.70	98.90±6.88	0.008 (F=4.944) S	0.388 (t=0.867) NS	0.033 (t=2.167) S	0.002 (t=3.193) S
Oxygen Saturation (Spo2)	99.96±0.20	99.96±0.20	99.96±0.20	0.816 (F=0.203) NS	1.000 (t=0.000) NS	0.562 (t=0.581) NS	0.562 (t=0.581) NS
End-tidal CO2 (EtCO2)	37.06±2.21	37.70±2.25	37.24±2.51	0.368 (F=1.005) NS	0.154 (t=1.435) NS	0.704 (t=0.381) NS	0.337 (t=0.964) NS

At the 12th minute (T12) post-pneumoperitoneum, dexmedetomidine (Group D) demonstrated significantly better control of heart rate (73.06 ± 7.78 bpm) compared to labetalol (82.26 ± 7.50 bpm) and lignocaine (86.00 ± 9.37 bpm) (p = 0.001). Systolic blood pressure was also significantly lower in Group D (118.22 ± 8.83 mmHg) than in Group L (124.44 ± 11.02 mmHg) and Group Lox (128.98 ± 8.57 mmHg) (p = 0.001).

For diastolic BP, Group D (80.04 ± 7.53 mmHg) showed significantly lower values than Group Lox (83.82 ± 6.50 mmHg) (p = 0.008), though differences with Group L were not significant. Mean arterial pressure (MAP) was lowest in the dexmedetomidine group (94.24 ± 7.70 mmHg), followed by labetalol (95.62 ± 8.20 mmHg), and highest in the lignocaine group (98.90 ± 6.88 mmHg), with a statistically significant overall difference (p = 0.008). Notably, MAP differences were significant between Group D and Group Lox (p = 0.002) and Group L and Group Lox (p = 0.033), but not between Group L and Group D.

SpO_2 remained stable at 99.96 ± 0.20% across all groups (p = 0.816), and $EtCO_2$ showed no significant variation (p = 0.368), confirming no adverse respiratory effects of the study drugs during pneumoperitoneum.



Comparison of Vitals at T12 (Pneumoperitoneum)

Discussion

Laparoscopic cholecystectomy is a widely performed surgical procedure associated with minimal invasiveness, shorter hospital stays, and faster recovery.

However, it is not without its physiological challenges, particularly during the creation of pneumoperitoneum. The insufflation of carbon dioxide (CO_2) into the peritoneal cavity leads to an increase in intra-abdominal pressure, which, in turn, stimulates the sympathetic nervous system. This sympathetic surge can result in significant hemodynamic changes such as tachycardia, hypertension, and increased systemic vascular resistance. These effects are generally well tolerated in healthy individuals but may pose serious risks in patients with underlying cardiovascular disease, such as coronary artery disease, hypertension, or arrhythmias.

In this context, pre-emptive pharmacological modulation of the autonomic response becomes essential to enhance intraoperative stability and improve patient outcomes. The present study was designed to compare the efficacy of three pharmacologic agents—dexmedetomidine, labetalol, and lignocaine—in attenuating the hemodynamic responses to pneumoperitoneum during laparoscopic cholecystectomy. Each of these drugs exerts its effects through distinct mechanisms, which were reflected in their differing hemodynamic profiles observed in this study.

Dexmedetomidine:

Dexmedetomidine, a highly selective α2-adrenergic receptor agonist, demonstrated the most effective attenuation of both heart rate (HR) and blood pressure (BP) responses in our study. Even at a lower dose of 0.5 µg/kg, it consistently maintained stable haemodynamics throughout the perioperative period. This can be attributed to its central sympatholytic action, which results in decreased norepinephrine release and blunted stress response. Additionally, its sedative, anxiolytic, and analgesic properties contribute to overall hemodynamic stability by reducing perioperative anxiety and nociceptive stimuli.

The findings of this study align with those reported by Gaiwal et al. (2025), Bhutia and Rai (2017), and Alam et al. (2023), who noted similar improvements in intraoperative haemodynamics and postoperative recovery parameters with dexmedetomidine administration.[8,10] Moreover, the 10% incidence of bradycardia in our dexmedetomidine group is consistent with previously reported rates in the literature, such as Basar et al. (2008) and Kewalramani et al. (2016), where similar dosing regimens were used.[14,7] While bradycardia may be viewed as a side effect, in many cases it reflects the desired reduction in sympathetic tone and is easily manageable with appropriate monitoring and intervention.

Labetalol:

Labetalol, a mixed α- and β-adrenergic antagonist, was effective in maintaining systolic and diastolic blood pressure but was comparatively less effective in controlling heart rate fluctuations. Its pharmacodynamic profile—blocking β1 and β2 receptors in combination with selective α1 antagonism—results in decreased peripheral vascular resistance and moderate reductions in cardiac output. These effects make it particularly suitable for patients with labile or borderline hypertension.

In our study, labetalol provided stable hemodynamic parameters with only a 2% incidence of hypotension, and no reports of bradycardia or arrhythmias. These findings reinforce the safety and utility of labetalol at a dose of 0.25 mg/kg. Similar observations were made by Singla et al. (2019), who advocated the use of labetalol in patients where blood pressure control was paramount, especially those with borderline or established hypertension.[11]

Lignocaine:

Lignocaine, primarily known as a local anaesthetic and antiarrhythmic agent, has gained interest in recent years for its systemic effects when administered intravenously. Its mechanism involves sodium channel blockade, which can stabilize neuronal membranes and attenuate the transmission of pain signals and stress responses. Additionally, lignocaine has mild sympatholytic and anti-inflammatory properties that may contribute to intraoperative hemodynamic modulation and postoperative pain relief.

In the present study, lignocaine demonstrated moderate effectiveness in attenuating intraoperative stress responses. It was less efficacious than both dexmedetomidine and labetalol in suppressing acute elevations in HR and BP. These findings are consistent with those of Karan et al. (2021) and Hegazy et al. (2019), who also reported modest hemodynamic benefits and enhanced postoperative comfort following lignocaine infusion.[12,13]

Clinical Implications and Risk-Benefit Considerations:

The comparative analysis of these three agents offers important clinical insights. Dexmedetomidine, though associated with a higher incidence of bradycardia,

provided the most comprehensive attenuation of the hemodynamic stress response and may be especially beneficial in patients where both HR and BP control are critical. However, it requires close intraoperative monitoring due to its potent sympatholytic effect.

Labetalol emerges as a useful agent in scenarios where hypertension is the primary concern, especially in patients with preserved cardiac function and minimal risk of bradycardia. Its dual α- and β-blocking activity allows for effective BP control without significant cardiac depression at lower doses.

Lignocaine, although less potent in immediate hemodynamic modulation, demonstrated value through its analgesic benefits and exceptional safety profile. It may be particularly beneficial as an adjunct in multimodal analgesia or in patients who cannot tolerate β-blockers or α2-agonists.

Limitations and Future Directions:

While the findings of this study provide valuable insights, certain limitations must be acknowledged. The sample size, while adequate for detecting significant differences in primary outcomes, may not fully capture the variability in patient responses, especially in subgroups with severe comorbidities. Additionally, the duration of follow-up was limited to the intraoperative and immediate postoperative period; future studies may explore long-term outcomes such as postoperative recovery time, pain scores, and incidence of complications like nausea, vomiting, or delayed emergence.

Further research should also investigate optimal dosing strategies, combination therapies, and patient-specific factors (e.g., age, comorbidities, and ASA status) that may influence the choice of agent. Multicentric trials with larger populations would help validate these findings and contribute to more individualized perioperative care protocols.

Conclusion:

Among the agents studied, dexmedetomidine demonstrated superior efficacy in attenuating the hemodynamic responses to pneumoperitoneum during laparoscopic cholecystectomy, offering consistent control of heart rate and blood pressure with a favourable safety profile. Labetalol effectively maintained blood pressure with minimal adverse effects, making it a suitable alternative. Lignocaine, while safe and beneficial for postoperative analgesia, was less effective in controlling intraoperative hemodynamic fluctuations. These findings support the use of dexmedetomidine as a preferred agent for hemodynamic stability in laparoscopic procedures, particularly in patients at risk of exaggerated sympathetic responses.

References:

1. Cunningham AJ, Brull SJ. Laparoscopic cholecystectomy: anesthetic implications. Anesth Analg. 1993 May;76(5):1120–33.
2. Gutt CN, Oniu T, Mehrabi A, Schemmer P, Kashfi A, Kraus T. Circulatory and respiratory complications of carbon dioxide insufflation. Dig Surg. 2004;21(2):95–105.
3. Sprung J, Abdelmalak B, Schoenwald PK. Recurrent complete heart block in a healthy patient during laparoscopic electrocauterization of the fallopian tube. Anesthesiology. 1998 May;88(5):1401–3.
4. Rauh R, Hemmerling TM, Rist M, Jacobi KE. Influence of pneumoperitoneum and patient positioning on respiratory system compliance. J Clin Anesth. 2001 Aug 1;13(5):361–5.
5. Bajwa SJS, Kulshrestha A. Anaesthesia for laparoscopic surgery: General vs regional anaesthesia. J Minim Access Surg. 2016 Jan–Mar;12(1):4–9.
6. Hirvonen EA, Poikolainen EO, Pääkkönen ME, Nuutinen LS. The adverse hemodynamic effects of anesthesia, head-up tilt, and carbon dioxide pneumoperitoneum during laparoscopic cholecystectomy. Surg Endosc. 2000 Mar;14:272–7.
7. Kewalramani A, Partani S, Sharma NP, Sharma V. Comparison of labetalol versus dexmedetomidine to assess the haemodynamic responses to laryngoscopy and intubation during induction of general anaesthesia–A prospective, randomized, controlled study. Indian J Clin Anaesth. 2016 Jun;3(4):512–7.
8. Gaiwal S, Palep JH, Mirkute R, Prasad N, Kush M. Low-pressure pneumoperitoneum with intraoperative dexmedetomidine infusion in laparoscopic cholecystectomy for enhanced recovery after surgery: a prospective randomised controlled clinical trial. J Minim Access Surg. 2025 Apr 1;21(2):147–52.
9. Bhutia MP, Rai A. Attenuation of haemodynamic parameters in response to pneumoperitoneum

during laparoscopic cholecystectomy: a randomized controlled trial comparing infusions of propofol and dexmedetomidine. J Clin Diagn Res. 2017 May 1;11(5):UC01–4.

10. Alam AF, Mahbub-Ur-Rahman M, Ahsan MN, Fatema K, Matber MS. Esmolol hydrochloride and dexmedetomidine on pressure response during laryngoscopy, intubation and pneumoperitoneum in laparoscopic surgery. J Patuakhali Med Coll. 2023 Jul;2(2)
11. Singla D, Parashar A, Pandey V, Mangla M. Comparative evaluation of dexmedetomidine and labetalol for attenuating hemodynamic stress responses during laparoscopic cholecystectomy in borderline hypertensive patients. Rev Esp Anestesiol Reanim (Engl Ed). 2019 Apr 1;66(4):181–8.
12. Karan P, D'souza N, Patil R. A prospective randomized study to evaluate the analgesic efficacy and quality of recovery of perioperative intravenous lignocaine infusion in laparoscopic surgeries. Res Innov Anesth. 2021 Nov 26;6(2):36–43.
13. Hegazy AA, El-Sayed MM, Al Wakel AM. Attenuation of pneumoperitoneum-induced hypertension by intraoperative lidocaine infusion in laparoscopic cholecystectomy. Egypt J Hosp Med. 2019 Jul 1;76(6):4436–44.
14. Basar H, Akpinar S, Doganci N, Buyukkocak U, Kaymak Ç, Sert O.The effects of preanesthetic, single-dose dexmedetomidine on induction, hemodynamic, and cardiovascular parameters. J Clin Anesth. 2008 Sep 1;20(6):431–6.

Original Research Article

PREOPERATIVE PREDICTORS OF MORTALITY IN ADULT PATIENTS WITH GASTRIC AND SMALL BOWEL PERFORATION

Parth Dhamija, Tejinder Paul Singh,[1] Ashwani Kumar[2], Jaswinder Singh, Dinesh Kumar Pasi[3]
S.R.[1], Professor & Head[2], Assoc. Professor[3],
Deptt of General & Minimal Access Surgery, GMC & Rajindra Hospital, Patiala

Corresponding Author : Dr. Parth Dhamija
S.R., Deptt of General & Minimal Access Surgery, GMC & Rajindra Hospital, Patiala
E-mail: parthdhamija04@gmail.com

ABSTRACT

Background: Perforation peritonitis is one of the most serious conditions encountered by a surgeon in the emergency. As a result, it becomes important to diagnose this condition early and intervene at the earliest. Higher mortality rates have been implicated with this disease as a result of which understanding of preoperative predictors of mortality becomes necessary to lower down these rates. **Methods:** A prospective study of 60 cases diagnosed with perforation peritonitis who underwent laparotomy was undertaken at Government Medical College and Rajindra Hospital, Patiala and data was collected in terms of age, gender, signs and symptoms, presentation to hospital, surgery timing and hematological investigations and was evaluated. **Results:** Distribution of cases showed a bimodal pattern where both younger and middle age group patients were present, majority of which were males (53/60). Ileal perforation was the most common intraoperative finding in the younger population whereas 50% (7/14) of cases in the middle age group had gastric perforation. 41/60 patients presented within 72 hrs from the onset of symptoms out of which only 3 patients died. 53/60 patients underwent exploratory laparotomy within 24hrs of admission. Higher mortality was observed in patients having raised total leucocyte count(TLC), deranged renal function tests (RFT) and dyselectrolytemia. **Conclusion:** Through our study we aimed to evaluate the importance of certain preoperative factors that can play a role in predicting mortality in patients of gastric and small bowel perforation.

Keywords : Peritonitis, Perforation, Predictors, Mortality

INTRODUCTION

Peritonitis is the inflammation of the peritoneum which may be primary where the source of infection is outside the peritoneal cavity, or secondary which occurs due to a perforated hollow viscus, or tertiary which develops following treatment of secondary peritonitis.[1] Peritonitis is mainly a clinical diagnosis with tenderness, rigidity, guarding and vomiting being the important presenting features. A plain chest X-ray showing presence of free air under the domes of diaphragm confirms the diagnosis for further surgical intervention.[2] Occurrence of gastroduodenal or ileal perforations is relatively common in our country due to excessive smoking, alcohol or coffee consumption, increased use of NSAIDS like aspirin, ibuprofen etc., and prevalence of infections such as H. pylori and typhoid fever.[3] Majority of the patients present to the emergency in late stages (septicemia, shock) where despite advancements in surgical techniques, anti-microbial therapy and intensive care, morbidity and mortality rates are very high. In addition, various risk factors such as old age, co-morbid conditions, deranged liver and renal function, coagulopathies etc. affect the treatment outcome.[4-8] Interaction of patient related factors, disease-specific factors and diagnostic and therapeutic interventions determine the prognosis and

outcome of peritonitis. Thus categorizing the patients based on preoperative risk factors would help predict the outcome, select patients for intensive care and determine operative risk ultimately deciding on final therapeutic intervention.[9]

A delay in presentation with subsequent septic shock was considered as the most important determinant of mortality in study done by Desa et al[10] which is corroborated by studies done by Simmen et al[1], Singh R et al[4], Jhobta RS et al[5], Moller MH et al[6] and Afridi SP et al[11] where further delay results in setting of dyselectrolytemia, acute respiratory distress syndrome (ARDS) and toxemia. This is relevant in our region as most of the population lives in semi-urban and rural areas where proper hospital services are not available and presence of quacks further causes delay in provision of authentic treatment. Poor physiologic reserves along with presence of co-morbid conditions in old age leads to inability of the frail body to counter stressful measures such as setting of peritonitis.[4,11-13]

Studies done by Gupta SK et al[3], Testini M et al[14], Sunil K et al[15] and Tanveer A et al[16] have recommended to stabilize the patients of perforation peritonitis with intravenous(IV) fluids and antibiotics at least two hours prior to surgery as it has to shown to have a better outcome as compared to patients going for immediate surgery. Correction of dyselectrolytemia and deranged RFT's is advisable as they are associated with poor operative outcomes.[4,15,17-18]

This study was done with the aim to highlight the preoperative factors resulting in mortality in 60 patients admitted with provisional diagnosis of perforation peritonitis of small bowel in Government Medical College and Rajindra Hospital, Patiala.

MATERIALS AND METHODS

A prospective study of 60 patients of perforation peritonitis who underwent exploratory laparotomy was done at GMC and Rajindra Hospital, Patiala where a detailed symptomatic history along with history of comorbid conditions and treatment history was taken. Monitoring of vitals was done and recorded. Abdominal examination was done in a detailed manner and patients were checked for signs and symptoms of perforation peritonitis. Following radiological investigations in the form of chest X-ray and ultrasound abdomen and resuscitation with intravenous fluids and administration of IV antibiotics, patients were taken up for exploratory laparotomy and intraoperative findings were noted.

RESULTS

Table 1 : Age and Sex Distribution

Age Group (Years)	Male		Female	
	No. of cases	%age	No. of cases	%age
16-25	14	23.33	4	6.67
26-35	11	18.33	2	3.33
36-45	8	13.33	0	0
46-55	13	21.67	1	1.67
56-65	5	8.33	0	0
66-75	1	1.67	0	0
>75	1	1.67	0	0
Total	53	88.33	7	11.67
Range	17-85		20-50	
Mean	40.53 ± 15.92		29.57 ± 9.81	

Table 2 : Age wise Distribution of Site of Perforation

Site of Perforation	Age groups (Years)							Total
	16-25	26-35	36-45	46-55	56-65	65-75	>75	
Gastric	2	4	2	7	3	1	0	19
Duodenal	2	1	1	3	0	0	0	7
Jejunal	2	1	0	1	0	0	1	5
Ileal	12	7	5	3	2	0	0	29
Total	18	13	8	14	5	1	1	60

Majority of the patients in our study population were males (53 out of 60) with a bimodal distribution where the number of patients was almost equal in the age groups of 16-25 and 46-55 (14 and 13 respectively). Out of 14 cases in the younger age group, 12 cases had ileal perforation while 7 cases out of the 13 cases of 46-55 age group had gastric perforation. This is attributable to the fact that typhoid disease is prevalent more in the younger population and is one of the most common causes of ileal perforation. Similar results can also be seen in the female group where 6 out of 7 cases belong to the younger population. Whereas increased incidence of smoking and alcohol consumption in addition to NSAID abuse is more common in middle to old age population which results in peptic ulcer disease with complications as gastric and duodenal perforation.

Abdominal pain was the most consistent presenting symptom in our study (60 cases, 100%), followed by

nausea/vomiting (38 cases, 63.34%). Guarding was the most common sign seen on abdominal examination (59 cases, 98.33%) followed by abdominal tenderness (56 cases, 93.34%). In addition, 9 cases had bilateral chest crepitations which may be attributed to development of concurrent respiratory infections in the setting of septicemia or due to pre-existing COPD especially in smokers.

Table 3 : Time Difference between onset of Sypmtoms and Presentation to the Hospital

Duration	No. of patients	Percentage (%)	No. of deaths	p- value	Significance
≤ 24 hrs	23	38.33	2	0.036	Significant
2-3 days	18	30.00	1		
4-5 days	13	21.67	0		
6-7 days	2	3.33	1		
8-9 days	1	1.67	1		
10th day	3	5.00	2		
Total	60	100	7		

Majority of patients in our study presented within the first 72 hrs of the onset of symptoms (41 out of 60) resulting in timely resuscitative measures and surgical intervention which is evident by highly low levels of mortality (3 cases) which signifies the importance of early presentation to the surgical emergency. Mortality rates were higher in patients presenting a week or later (4 out of 6 cases) where the patients usually land up with severe septicemia and multi organ dysfunction (MODS).

Table 4 : Time delay between Hospital admission and Surgery

Duration between admission and surgery	No. of cases	Percentage	No. of deaths	p-value	Significance
Within 24 hrs	53	88.33	7	0.634	Non-significant
Day 2	6	10.00	0		
Day 3	0	0	0		
Day 4	1	1.67	0		
Total	60	100	7		

Majority of the patients (88.34%) were operated within 24 hrs of admission. One patient was operated on Day 4 as the patient did not give consent for surgery initially. All the 7 deaths that have occurred in the study were in those patients who were operated at the earliest. This may be due to the maximum number of patients falling under this group which suggests a multimodal array of factors responsible for surgical outcome. Maximum patients being operated within first 24 hrs gives an impression that the surgeons are well acquainted with the benefits of early intervention after prompt diagnosis and adequate resuscitation.

Table 5 : VITALS

Heart Rate (/min)	No. of cases/No of deaths	p-value and significance	Blood pressure (mmHg)	No. of cases/No of deaths	p-value and significance	Respiratory rate (/min)	No. of cases/No of deaths	p-value and significance
<100	37/1	0.045 Significant	≤ 90	19/6	0.019 Significant	<20	19/0	0.044 Significant
100-120	22/6		91-139	30/1		20-24	19/1	
>120	1/0		≥ 140	11/0		>25	22/6	
Total	60/7		Total	60/7		Total	60/7	
Range	78-130		Range	90-160		Range	15-34	
Mean ± SD	98.62±11.08		Mean ± SD	109.23±20.46		Mean ± SD	22.98±5.10	

Tachycardia is one of the earliest signs to depict the presence of infection in the body. Its presence indicates timely antibiotic administration and fluid resuscitation. 23 patients in our study had heart rate above 100/min. Similar to tachycardia, low BP is a feature of septicemia as endotoxins released by bacteria cause peripheral vasodilation superadded by third space volume loss resulting in hypotension and poor tissue perfusion. 49 patients in our study has SBP <140 mm Hg. Sepsis results in metabolic acidosis where body tries to compensate by increasing the respiratory rate (Tachypnoea). 41 patients had RR >20/min. All the 7 deaths had tachycardia, hypotension and tachypnoea in different intensities.

Table 6 : Hematological Investigations – Part 1

Total Leucocyte Count (TLC) (/mm³	**No. of cases/No of deaths**	**p-value and significance**	**S. urea (mg%)**	**No. of cases/No of deaths**	**p-value and significance**	**S. Creatinine (mg%)**	**No. of cases/No of deaths**	**p-value and significance**
< 4000	0/0	0.038 Significant	21-40	49/3	0.047 Significant	≤ 1.5	39/6	0.048 Significant
4000-11000	41/2		41-60	7/1		1.6-1.9	20/0	
> 11000	19/5		> 60	4/3		≥ 2.0	1/1	
Total	60/7		Total	60/7		Total	60/7	
Range	4000-16000		Range	20-108		Range	0.7-3.5	
Mean ± SD	10163.58 ± 2685.57		Mean ± SD	38.20 ± 16.82		Mean ± SD	1.32 ± 0.44	

Table 7 : Hematological Investigations – Part 2

S. Sodium (mEq/L)	**No. of cases/No of deaths**	**p-value and significance**	**S. Potassium (mEq/L)**	**No. of cases/No of deaths**	**p-value and significance**
< 136	31/3	0.659 Non-significant	< 3.5	7/1	0.926 Non-significant
136 -144	29/4		3.5 -5.2	52/6	
> 144	0/0		> 5.2	1/0	
Total	60/7		Total	60/7	
Range	122 -142		Range	3-5.3	
Mean ± SD	134.07 ± 5.25		Mean ± SD	4.33 ± 0.56	

19 cases had TLC count > 11000/mm3 out of which 5 deaths (of total 7) were recorded. This can be explained by the fact that body activates inflammatory cascade in response to bacterial load resulting in raised TLC. 11 patients had blood urea levels >40mg% where out of total 7 deaths, 4 were recorded. This may be attributed to the setting of poor renal function as a result of decreased tissue perfusion in septicemia.

Serum sodium and potassium levels in all of our patients were on the lower side of the spectrum probably due to inflammatory cascade and dehydration and the values were found to be statistically non-significant.

DISCUSSION

Secondary peritonitis due to perforation of a hollow viscus organ primarily small bowel is a common occurrence in surgical emergency setting. This prospective study of 60 patients at GMC and Rajindra hospital, Patiala was undertaken to evaluate preoperative predictors of mortality in small bowel perforation where mortality was taken as the final outcome.

Age is an important prognostic factor in predicting the mortality in patients of perforation peritonitis as evident by reviews done by Sanjay G and Robin K et al[13] where higher mortality was observed in patients aged 50 years and above. Barut et al[17] in their study observed 8 deaths in 26 cases aged above 60 years where they concluded that age is an important predictor of mortality in such patients. Older patients tend to have a reduced physiological reserve as a result of which the body is not able to handle stressful events such as septicemia leading to prolonged hospital stay and a delayed recovery resulting in higher mortality rates. In our study as well, out of the total 7 deaths that occurred, maximum mortality was seen in the 56-65 years age group.

53 out of the total cases in our study were males. This heavy preponderance could be due to alcohol addiction, excessive smoking, outdoor life and consumption of irregular meals along with lack of exercise. Similar results were obtained in studies done by Choudhary J et al[19], Memon AA et al[20] and Jhobta RS et al[5].

Table 8 : Comparison of Studies in regards to age

Study	Male	Female
Choudhary (2010) [19]	85.5%	11.6%
Memon AA et al (2012) [20]	77%	23%
Jhobta RS et al (2006) [5]	84%	16%
Present Study	88.34%	11.67%

A delay in presentation to the hospital is one of the most important factor in contributing to higher morbidity and mortality rates in cases of perforation peritonitis. This is usually due to lack of health awareness especially among the rural population, lack of proper health care facilities in the vicinity and dependence upon local quacks/chemists for the treatment. Studies done by Gupta SK et al[3], Afridi SP et al[11], Tanveer et al[16] and Robin K and Sanjay G[13] have observed higher mortality rates in patients presenting late to the hospital.

Chalya LP et al[21] in their study observed that majority of patients in their study presented in poor general condition and concluded that early presentation equals lower morbidity and mortality rates. Paryani JJ et al[22] stated that delayed presentation leads to septicemia leading to lower survival rates. In our study as well, 16 out of 60 cases presented after 4 days from the onset of symptoms with 4 out of total 7 deaths occurring in such group. Therefore it is imperative on part of health department and administration to make the population aware about the importance of seeking early specialized treatment from the professionals for their diseases.

The timing of surgical intervention is as important as is the presentation to the hospital. Early intervention is associated with lower mortality rates as evident by study done by Singh R et al[4] where 75% patients were operated within 24hrs of admission out of which only 3 patients died. However, in our study, 53 (88%) cases underwent laparotomy within 24 hrs but all 7 deaths occurred in this group only. This was attributed to the fact that majority of these were of old age and had

presented late to the hospital.

Tachycardia, Hypotension and tachypnoea are the important signs with which the patients of perforation peritonitis usually present. Monitoring and understanding of these vitals is important in predicting the overall outcome for the patient. Studies done by Barut I et al[17], Samuel CJ et al[18] and Singh R et al[4] have recognized that patients having heart rate > 100/min had poor operative outcomes. 6 patients in our study had heart rate > 100/min who later on passed away. Barut I et al[17] and Samuel CJ et al[18] also observed hypotension (<90 mm Hg SBP) as a poor prognostic factor. Singh R et al[4] considered > 20/min RR to be associated with higher mortality whereas Paryani JJ et al[22] mentioned > 30/min RR to be related to increased mortality.

Paryani JJ et al[22] observed raised TLC count to have poor outcome after surgery. Singh R et al[4] in his study observed septicemia in patients having TLC count > 11000/mm3. He also mentioned RFT's to be independent predictors of mortality. Deranged levels were associated with increased mortality. Jhobhta RS et al[5] in his study reported hyponatremia in 29% cases whereas hypokalemia was seen in 9% of the patients, although no relation to mortality was associated. Tan KK et al[23] observed deranged serum potassium levels to be associated with worse outcomes in perforation patients. Evaluation suggests that correction of the electrolytes is imperative for better surgical outcomes and subsequent reduction in mortality rates.

CONCLUSION

Perforation peritonitis is one of the most common presentations to the hospital emergency and is associated with significant morbidity and mortality. Through our study we have come to a conclusion that it is possible to predict mortality in perforation peritonitis patients via a number of factors, of which age, early presentation to the hospital along with early intervention and deranged hematological interventions are significant ones. A large sample size would be required for evaluation of certain other intrinsic factors which may play a role in occurrence of mortality in such patients.

LIMITATIONS

Larger sample size may be required to increase the statistical power of the study and therefore form detailed correlations between various variables. This being a single centre study, its findings may not be applicable to other settings.

CONFLICT OF INTEREST

The authors report no conflicts of interest related to this study

FINANCIAL SUPPORT

The authors received no financial support for the research, authorship and/or publication of this article.

REFERENCES

1. Simmen HP, Heinzelmann M, Largiader F. Peritonitis: Classification and causes. Digestive Surgery. 1996 Nov 6;13(4-5):381-3.
2. Yıldırım M, Engin Ö, İlhan E, Coşkun A. RISK FACTORS AND MANNHEIM PERITONITIS INDEX FOR THE PREDICTION OF MORBIDITY AND MORTALITY IN PATIENTS WITH PEPTIC ULCER PERFORATION. Nobel Medicus Journal. 2009 Sep 1;5(3).
3. Gupta SK, Gupta R, Singh G, Gupta S. Perforation peritonitis: A two year experience. Jk Science. 2010 Jul 1;12(3):141.
4. Singh R, Kumar N, Bhattacharya A, Vajifdar H. Preoperative predictors of mortality in adult patients with perforation peritonitis. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine. 2011 Jul;15(3):157.
5. Jhobta RS, Attri AK, Kaushik R, Sharma R, Jhobta A. Spectrum of perforation peritonitis in India-review of 504 consecutive cases. World journal of Emergency surgery. 2006 Dec;1:1-4.
6. Møller MH, Shah K, Bendix J, Jensen AG, Zimmermann-Nielsen E, Adamsen S, Møller AM. Risk factors in patients surgically treated for peptic ulcer perforation. Scandinavian journal of gastroenterology. 2009 Jan 1;44(2):145-52.
7. Külah B, Gülgez B, Ozmen MM, Ozer MV, Coşkun F. Emergency bowel surgery in the elderly. The Turkish Journal of Gastroenterology: The Official Journal of Turkish Society of Gastroenterology. 2003 Sep 1;14(3):189-93.
8. SEO JH, PARK HK, PARK YH, LEE HK, LEE WG, CHO SY, LEE JN, LEE YD. Prognostic factors in duodenal ulcer perforation. Journal of the Korean Surgical Society. 2001:425-31.
9. Malik AA, Wani KA, Dar LA, Wani MA, Wani RA, Parray FQ. Mannheim Peritonitis Index and APACHE II-Prediction of outcome in patients with peritonitis. Turkish journal of trauma and emergency surgery. 2010 Jan 1;16(1):27-32.
10. Desa LA, Mehta SJ, Nadkarni KM, Bhalerao RA. Peritonitis: A study of factors contributing to mortality. Indian J Surg. 1983;45:593-604.
11. Afridi SP, Malik F, Ur-Rahman S, Shamim S, Samo KA.

Spectrum of perforation peritonitis in Pakistan: 300 cases Eastern experience. World Journal of Emergency Surgery. 2008 Dec;3:1-5.
12. Hiyama DT, Bennion RS. Peritonitis and Intraperitoneal Abscess: Maingot's Abdominal Operation Micheal J. vol.;1:634-53.
13. Gupta S, Kaushik R. Peritonitis–the Eastern experience. World journal of emergency surgery. 2006 Dec;1:1-6.
14. Testini M, Portincasa P, Piccinni G, Lissidini G, Pellegrini F, Greco L. Significant factors associated with fatal outcome in emergency open surgery for perforated peptic ulcer. World Journal of Gastroenterology. 2003 Oct 15;9(10):2338.
15. Kumar S, Gupta A, Chaudhary S, Agrawal N. Validation of the use of POSSUM score in enteric perforation peritonitis-results of a prospective study. Pan African Medical Journal. 2011;9(1).
16. Ahmad T, Khan MI, Hussain N, Siddiqui E, Islam ZU. Perforation operation interval as a prognostic factor in typhoid ileal perforation. J Surg Pakistan (International). 2009 Jan;14:11-4.
17. Barut I, Tarhan OR, Cerci C, Karaguzel N, Akdeniz Y, Bulbul M. Prognostic factors of peptic ulcer perforation. Saudi medical journal. 2005 Aug 1;26(8):1255-9.
18. Samuel JC, Qureshi JS, Mulima G, Shores CG, Cairns BA, Charles AG. An Observational Study of the Etiology, clinical presentation and outcomes associated with peritonitis in Lilongwe, Malawi. World journal of Emergency surgery. 2011 Dec;6:1-5.
19. Choudhary J. Spectrum of perforation peritonitis-260 cases experience. Journal of NAMS. 2010 Jul;10(2).
20. Memon AA, Siddiqui FG, Abro AH, Agha AH, Lubna S, Memon AS. An audit of secondary peritonitis at a tertiary care university hospital of Sindh, Pakistan. World journal of emergency surgery. 2012 Dec;7:1-5.
21. Chalya PL, Mabula JB, Koy M, Kataraihya JB, Jaka H, Mshana SE, Mirambo M, Mchembe MD, Giiti G, Gilyoma JM. Typhoid intestinal perforations at a University teaching hospital in Northwestern Tanzania: A surgical experience of 104 cases in a resource-limited setting. World journal of emergency surgery. 2012 Dec;7:1-1.
22. Paryani JJ, Patel V, Rathod G. Etiology of peritonitis and factors predicting the mortality in peritonitis. National Journal of Community Medicine. 2013 Mar 31;4(01):145-8.
23. Tan KK, Bang SL, Sim R. Surgery for small bowel perforation in an Asian population: predictors of morbidity and mortality. Journal of Gastrointestinal Surgery. 2010 Mar 1;14(3):493-9.

Original Research Article

SEROPREVALENCE OF VIRAL HEPATITIS IN A TERTIARY CARE HOSPITAL

Rupinder Bakshi, Karshdeep Kaur, Jaspreet Kaur, Palika, Satinder Kaur, Tarnum Gupta

Corresponding Author : Dr Arunita Ghoshal
drarunita.ghoshal018@gmail.com

Short running title: Viral Hepatitis

Abstract

Background: Viral hepatitis remains a significant global public health concern, accounting for over one million deaths annually. Acute hepatitis is often caused by the hepatitis A and E viruses, and it can progress to acute liver failure. Chronic hepatitis, primarily due to Hepatitis B and C viruses, often remains asymptomatic until it leads to serious complications such as cirrhosis and hepatocellular carcinoma.

Aim and Objective: This study aims to assess the seroprevalence of viral hepatitis among clinically suspected patients presenting to a tertiary care hospital.

Materials and Methods: A retrospective analysis was carried out on probable viral hepatitis cases, based on blood samples received from RH Patiala and processed at the Virus Research and Diagnostic Laboratory (VRDL), Department of Microbiology, over a period of three months. Serological testing of all the samples was performed, by using rapid card tests and confirmation is done by ELISA for HEV IgM antibodies, HBsAg of HBV and anti HCV IgG antibodies.

Results: The seroprevalence of hepatitis markers observed in our study was as follows: HCV – 7.48%, HBV – 1.69%, HEV – 3.13%, and HAV – 15.6%. Among cases of acute hepatitis, HAV positivity was notably higher than HEV, whereas in chronic hepatitis, HCV positivity exceeded that of HBV. Gender-wise distribution showed a higher proportion of males testing positive across all hepatitis markers: HEV (58.33%), HCV (51.83%), HBV (50.74%), and HAV (56.86%). Age-wise analysis revealed that the majority of positive cases for HCV (69.59%), HBV (63.33%), and HEV (66.66%) occurred in the 20–60-year age group, while HAV positivity was predominantly observed in the 0–20-year age group (60%).

Conclusion: Viral hepatitis poses a substantial public health burden; however, its transmission can be effectively reduced through the implementation of comprehensive control strategies. These include timely laboratory diagnosis, appropriate therapeutic interventions, and widespread vaccination efforts. Strengthening these measures is essential to limit disease spread and reduce associated morbidity and mortality.

Introduction:

The global population has contended with infectious hepatitis for decades, enduring both acute and chronic forms that continue to pose a major public health challenge. Despite significant progress in diagnosis, treatment, and vaccination, viral hepatitis remains a leading cause of morbidity and mortality worldwide [1]. The World Health Organization (WHO) recommends targeted testing in high-risk populations and in regions with a prevalence exceeding 2% [2,3]. However, these evidence-based strategies have yet to be fully implemented in many endemic areas.

Viral hepatitis is often divided into acute and chronic forms, depending on the time interval and development of the infection [4]. Acute hepatitis is primarily caused by Hepatitis A Virus (HAV) and Hepatitis E Virus (HEV), both of which can lead to acute liver failure in severe casesHAV epidemics have caused major clinical, financial, and social costs in India. Notably, an Indian study has explored the association between food handlers and HAV infection among adolescents and adults [5].

Similarly, HEV is responsible for numerous outbreaks and sporadic cases, reportedly accounting for approximately 50% of acute viral hepatitis infections in adults [6]. In pregnant women, particularly during the second and third trimesters, hormonal and immunological changes can exacerbate HEV infection, leading to severe complications. These may include acute liver failure, miscarriage, intrauterine death, preterm labor, neonatal hepatitis, low birth weight, and even maternal death [7].

Chronic hepatitis, predominantly caused by Hepatitis B Virus (HBV), affects an estimated 296 million people globally, with 221 million residing in low- and middle-income countries. In the absence of timely intervention, HBV infection can progress to cirrhosis, hepatocellular carcinoma (HCC), and death, with mortality projected to peak at 1.14 million by 2035 [8]. In India, a meta-analysis estimates the HBV prevalence at 1.4%, with HBV-HIV co-infection rates ranging between 0.2% and 0.8% [9].

Hepatitis C Virus (HCV) infection remains another major contributor to chronic hepatitis, with an estimated global burden of 58 million cases, including 3.2 million adolescents and children. Approximately 1.5 million new HCV infections occur each year, and the disease was responsible for 290,000 deaths in 2019 [10]. In 2015, there were between 4.7 and 10.9 million cases. HCV viremia in India, with a frequency of about 0.5%. This underscores a significant national disease burden and highlights the need for accurate prevalence estimates to guide policy formulation and public health interventions [11].

Aim & objective: To determine the overall prevalence of acute and chronic viral hepatitis, in clinically suspected patients at our hospital.

Material and Methods: A retrospective observational study was conducted, over a period of three months from January 2025 to March 2025. Laboratory records of all the clinically suspected hepatitis samples, received in the Microbiology laboratory at our hospital, were analyzed. Demographic details, such as age and gender, along with serological test results, were analyzed. Blood samples were collected from both inpatients and outpatients who showed clinical signs of acute or chronic viral hepatitis. 5 ml of blood collected from suspected patients, who manifested with clinical features of acute and chronic viral hepatitis, from both IPD and OPD, was received in VRDL for serological testing. The specimens were centrifuged and stored at minus 20 deg C. The required test was performed using commercially available rapid tests and confirmation is done by using solid phase enzyme linked immunosorbent assay kits (ELISA). For each hepatitis cases, each serum sample was tested for IgM antibodies of HEV, HBsAg of HBV and anti HCV IgG antibodies utilizing the ELISA. The laboratory investigations were conducted as per standard guidelines and protocol.

Results:

Acute Hepatitis

HAV Seroprevalence: In the present study, a total of 102 cases were tested for hepatitis A virus (HAV). Of these, 58 (56.86%) were males and 44 (43.13%) were females. IgM anti-HAV antibodies were detected in 16 patients,

indicating a seropositivity rate of 15.68% (Figure 1).

Age-wise distribution revealed that the majority of positive cases (14 out of 16; 87.5%) were within the 0–20 and 20–40-year age groups, while only 2 cases (12.5%) belonged to the 40–60 and >60-year categories (Figure 3 and Table 1).

HEV Seroprevalence: A total of 96 cases were tested for hepatitis E virus (HEV), of which 56 (58.33%) were males and 40 (41.67%) were females. IgM anti-HEV antibodies were detected in 3 patients, yielding a seropositivity rate of 3.13% (Figure 1).

Age-wise distribution showed one positive case in each of the following age groups: 20–40 years, 40–60 years, and >60 years, with each group contributing 33.33% of the total HEV-positive cases (Figure 3 and Table 1). No cross-reactivity was observed between hepatitis A and E infections.

Monthly analysis (Table 3) revealed that all HEV-positive cases occurred during the month of March.

Chronic Hepatitis (Figure 2)

HBV Seroprevalence: A total of 3,599 individuals were tested for hepatitis B virus (HBV), comprising 1,826 males (50.74%) and 1,773 females (49.26%). Hepatitis B surface antigen (HBsAg) was detected in 60 individuals, resulting in an overall positivity rate of 1.69% (Figure 2). Age-wise distribution revealed the highest number of positive cases in the 20–40 year age group (24 cases; 40%), followed by those aged >60 years (18 cases; 30%), 40–60 years (14 cases; 23.33%), and the lowest in the 0–20 year group (4 cases; 6.66%) (Figure 3 and Table 2).

HCV Seroprevalence: A total of 5,931 patients were tested for anti-HCV IgG antibodies, including 3,074 males (51.83%) and 2,857 females (48.17%). Among these, 444 individuals (7.49%) tested positive for anti-HCV IgM antibodies, suggesting recent or ongoing infection (Figure 2).

Age-wise analysis revealed the highest seropositivity in the 20–40 year age group, with 177 cases (39.86%), followed by the 40–60 year group with 132 cases (29.73%). Individuals aged >60 years accounted for 84 cases (18.92%), while the lowest number of positive cases was observed in the 0–20 year age group, comprising 51 cases (11.49%) (Figure 3 and Table 2).

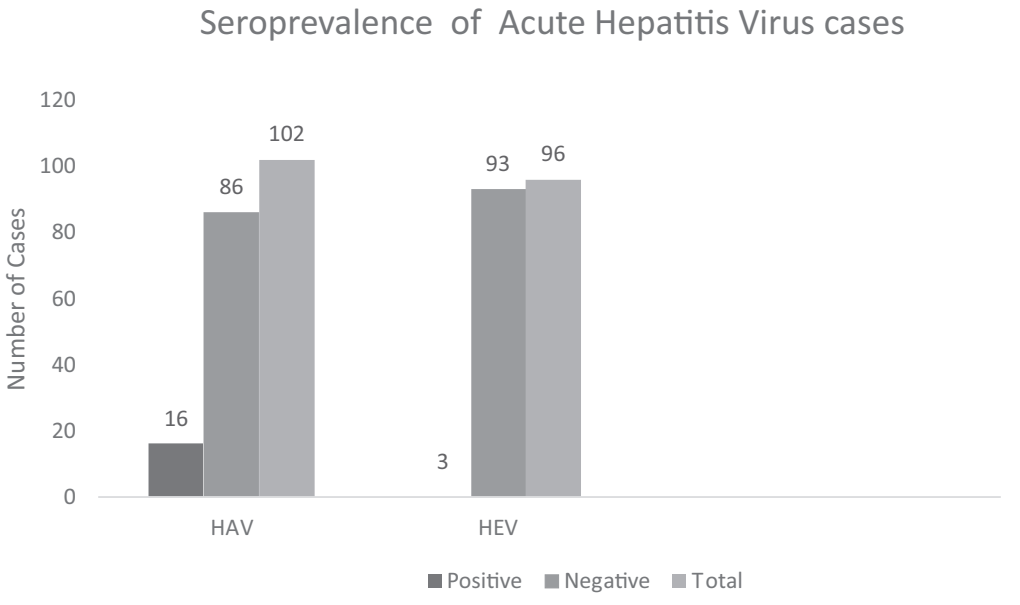


Figure 1: Seroprevalence of Acute Hepatitis Virus cases : HAV and HEV

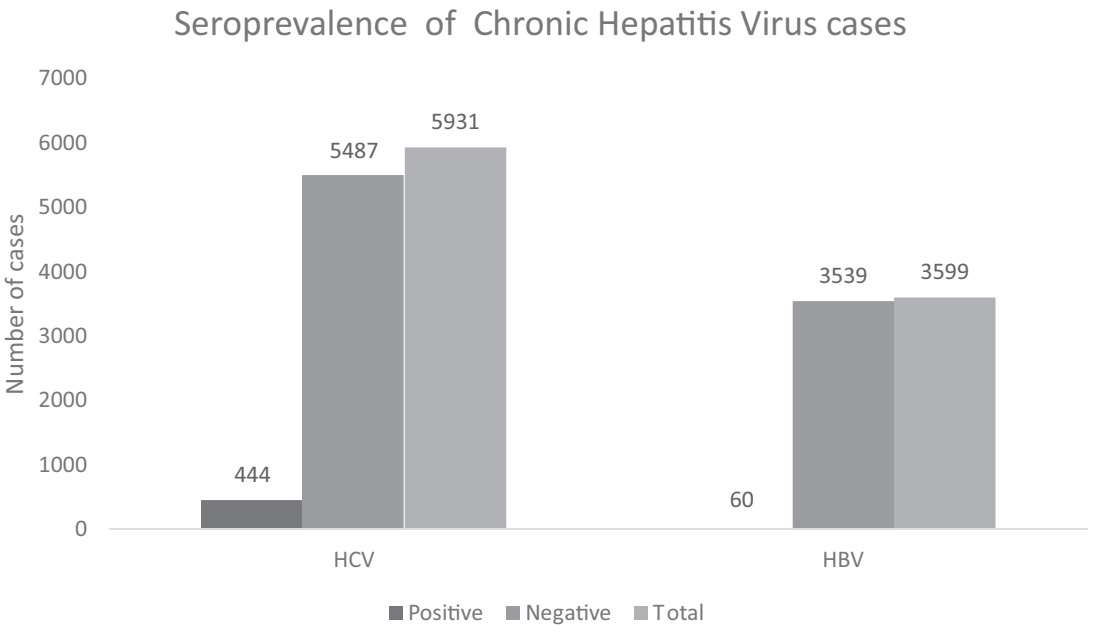


Figure 2: Seroprevalence of Chronic Hepatitis Virus cases: HCV and HBV

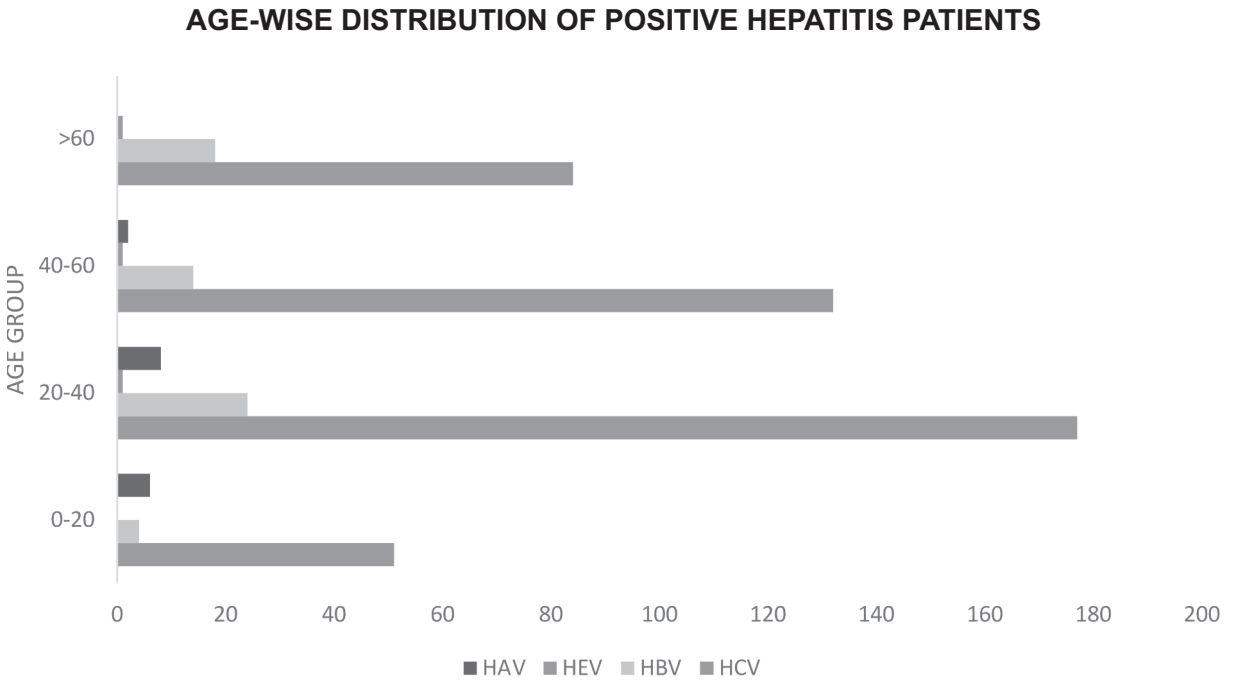


Figure 3:AGE-WISE DISTRIBUTION OF POSITIVE HEPATITIS PATIENTS

Table 1 AGE-WISE DISTRIBUTION OF ACUTE POSITIVE HEPATITIS PATIENTS

Age Group	HAV	HEV
0-20	50%	0 %
20-40	37.5%	33.33%
40-60	12.5%	33.33%
>60	0%	33.33%

Table 2: AGE-WISE DISTRIBUTION OF CHRONIC POSITIVE HEPATITIS PATIENTS

Age Group	HCV	HBV
0-20	11.49%	6.66%
20-40	39.86%	40%
40-60	29.73%	23.33%
>60	18.92%	30%

Table 3:Month wise distribution of Hepatitis Cases

Name of the Hepatitis Virus	January		February		March	
	Total no. patients	Total no. of Positive patients	Total no. patients	Total no. of Positive patients	Total no. patients	Total no. of Positive patients
HCV	2062	195	1645	115	2223	134
HBV	4	1	1440	25	2144	34
HAV	30	13	27	2	45	1
HEV	38	nil	20	Nil	26	3

Residential status of Hepatitis Patients: The residential status of all patients, revealing that the urban population consistently outnumbered the rural population across all cases as shown in table 4. This disparity likely reflects greater accessibility and utilization of testing services in urban areas. Additionally, urban residents exhibited a higher rate of positive diagnoses, which may be attributed to increased population density and greater viral exposure in these settings.

Table 4: Residential Status of Hepatitis Patients

Name of the Hepatitis Virus	Residential Status			
	Rural		Urban	
	Total no. of patients tested	Total number of Positive patients	Total no. of patients tested	Total number of Positive patients
HCV	1755	156	4176	288
HBV	1773	11	2726	49
HAV	Nil	Nil	102	16
HEV	Nil	Nil	96	96

DISCUSSION

The findings of this study present valuable insights into the characteristics of patients undergoing viral testing at a tertiary healthcare center. The predominance of young adults, particularly those in their late twenties, suggests that this age group may either be more susceptible to certain infections or more likely to seek care compared others. This could be attributed to occupational exposure, social mobility, or higher awareness levels in this demographic.

HAV: Our study on the seroprevalence of Hepatitis A Virus (HAV) in 102 patients revealed that 15.68% tested positive for IgM anti-HAV antibodies. The finding of the current study aligns with previous published study conducted by Bansal et al from North India (12). The present study reported the higher prevalence of male over females with a higher prevalence observed in the 0–40 years age group (87.5%). Similar results were observed by study conducted by Palewar et al. (13) and Grover et al. (14).

HEV: In our study, the seroprevalence of hepatitis E virus (HEV) was 1.33% (3/225). This finding is consistent with the low HEV positivity rates reported by Nair et al. (1.2%), Joon et al. (1.8%), and Daniel et al. (9%) [15–17]. However, comparatively higher prevalence rates have been observed in other studies, such as those by Bansal et al. (14.9%), Netra et al. (21.94%), and Kaur et al. (68.4%) [12, 18, 19]. These variations may be attributed to differences in sample size, study populations, socioeconomic status, sanitation conditions, and environmental hygiene.

Gender wise distribution: In the present study, a higher HEV positivity rate was observed among males. This finding is consistent with previous studies by Murhekar et al. (62.55%) [20] and Netra et al. (76.7%) [18], which also reported a male predominance among HEV-positive cases. The higher prevalence in males may be attributed to social and occupational factors that increase their exposure to contaminated water and street food.

Conversely, some studies have reported a higher HEV seropositivity among females, including those by Rawat et al. (69.56%) [21] and Bansal et al. (50.4%) [12].

Regarding age-wise distribution, all HEV-positive cases in our study were among young adult males. This is in line with findings by Bansal et al. [12], who reported a 14.3% HEV positivity rate among young male individuals.

Among Chronic Hepatitis cases, the seroprevalence of hepatitis B virus (HBV) was 1.69% (60/3,599) in present study. This finding is comparable to that of Kaur et al. [23], who reported an HBsAg positivity rate of 1.7% among blood donors in Chandigarh. Similarly, Giri et al. [24] observed a 1.6% prevalence among asymptomatic pregnant women. Mittal et al. [22] reported a slightly higher prevalence of 2.8% in the Uttarakhand region of North India, while Monika Rajani's tertiary-care study documented a 3.5% positivity rate among probable hepatitis cases. In contrast, significantly higher seroprevalence was reported among tribal populations in Himachal Pradesh, with rates reaching 10.6%, highlighting the regional heterogeneity and varying levels of endemicity across different populations [25].

In terms of gender distribution, males slightly outnumbered females in our study, accounting for 50.74% and 49.26% of cases, respectively. The male-to-female ratio (50.7:49.3) and the peak HBV seroprevalence observed in the 20–40 year age group (40%) are consistent with findings reported by Shadaker et al. and Khan et al. [26, 27].Conversely, several studies have documented a female predominance in HBV infection, including those by Sharma et al., Sandhu et al., and Manzoor et al. [28–30].

The age-wise distribution in our study, with the highest seropositivity observed among individuals aged 20–40 years, also aligns with findings from studies conducted in Punjab and Delhi, which report peak HBV prevalence in young adults [31].

The seroprevalence of hepatitis C virus (HCV) in our study was 7.5% (444/5,931), which is relatively high compared to several other studies. This finding is comparable to the 5.18% prevalence reported by Bagga et al. [32]. Kar et al. observed an even higher prevalence of 12% for IgM anti-HCV among acute viral hepatitis patients in Delhi [33].

In contrast, significantly lower rates were reported by Meena et al. (0.57%) [34] and Chaurasia et al. (0.54%) [35]. Mittal et al. documented a 1.8% seroprevalence in a hospital-based community screening [36], while Tandon et al. found an anti-HCV positivity of 1.57% among blood donors in New Delhi [37].

The elevated seroprevalence observed in our study likely reflects the higher proportion of symptomatic individuals in the study population, along with regional risk factors such as unsafe injection practices.

In our study, males comprised 51.83% of HCV-infected individuals, slightly exceeding the proportion of females at 48.17%. This male predominance aligns with findings reported by Kar et al. and Barman et al., who also documented higher rates of HCV infection among men [33, 38]. In contrast, Solomon et al. reported greater HCV seropositivity among women in Northeast India [39].

Urban residents made up the majority of the study population. This could be due to several factors: better healthcare infrastructure in cities, higher awareness, easier access to diagnostic facilities, and increased transmission potential due to dense population clusters. However, the relative underrepresentation of rural patients may mask the actual burden of disease in those communities, especially considering known barriers like transportation, cost, and social stigma in rural health-seeking behaviors.

This study did not investigate specific risk factors for hepatitis transmission, such as contaminated drinking water, blood transfusion history, familial transmission, body tattooing, or exposure among high-risk groups. Key populations including intravenous drug users, blood donors, sex workers, people living with HIV, healthcare workers, migrants from high-prevalence areas, children born to HBV-positive mothers, and close contacts were not assessed. Understanding these factors is essential for a comprehensive epidemiological picture and to inform targeted prevention and management strategies.

CONCLUSION

Despite ongoing awareness campaigns, preventive measures, advanced diagnostic tools, effective treatments, and available vaccines, the burden of hepatitis persists. Moving forward, there is a critical need to develop innovative and strengthened strategies that focus on the rapid identification and timely management of infected individuals. Additionally, bridging the gap between healthcare availability and accessibility—especially among vulnerable populations—is essential to achieve the ultimate goal of hepatitis elimination.

References:

1. Zhang S, Cui F. Global progress, challenges and strategies in eliminating public threat of viral hepatitis. Infectious Diseases of Poverty. 2025 Dec;14(1):1-4.
2. Hellard, M. E., Chou, R., & Easterbrook, P. (2017). WHO guidelines on testing for hepatitis B and C—meeting targets for testing. BMC Infectious Diseases, 17(Suppl 1), 703.
3. World Health Organization. (2022). Global progress report on HIV, viral hepatitis and sexually transmitted infections, 2021.
4. Odenwald MA, Paul S. Viral hepatitis: Past, present, and future. World journal of gastroenterology. 2022 Apr 14;28(14):1405.
5. Agrawal A, Singh S, Kolhapure S, Hoet B, Arankalle V, Mitra M. Increasing burden of hepatitis A in adolescents and adults and the need for long-term protection: a review from the Indian subcontinent. Infectious diseases and therapy. 2019 Dec;8:483-97.
6. Aslan AT, Balaban HY. Hepatitis E virus: Epidemiology, diagnosis, clinical manifestations, and treatment. World journal of gastroenterology. 2020 Oct 7;26(37):5543.
7. Mishra, S., Jha, R. K., Thakur, R., & Tiwari, S. (2016). Study of maternal and prenatal outcome in pregnant women with acute hepatitis E viral infection. International Journal of Reproduction, Contraception, Obstetrics and Gynecology, 5(7), 2300–2303.

8. Schweitzer, A., Horn, J., Mikolajczyk, R. T., Krause, G., & Ott, J. J. (2015). Estimations of worldwide prevalence of chronic hepatitis B virus infection: A systematic review of data published between 1965 and 2013. The Lancet, 386(10003), 1546–1555.
9. Sawant S, Agrawal S, Shastri J. Seroprevalence of hepatitis B and hepatitis C virus infection among HIV infected patients in Mumbai. Indian J Sex Transm Dis 2010;31:126.
10. Stroffolini T, Stroffolini G. Prevalence and modes of transmission of hepatitis C virus infection: a historical worldwide review. Viruses. 2024 Jul 11;16(7):1115.
11. Bakshi S, Chattopadhyay P, Ahammed M, Das R, Majumdar M, Dutta S, Nath S, Ghosh A, Bhattacharjee U, Baskey U, Sadhukhan PC. Efficacy of Different Combinations of Direct-Acting Antivirals Against Different Hepatitis C Virus-Infected Population Groups: An Experience in Tertiary Care Hospitals in West Bengal, India. Viruses. 2025 Feb 16;17(2):269.
12. Bansal Y, Singla N, Garg K, Sharma G, Gill M, Chander J. Seroprevalence of hepatitis A and hepatitis E in patients at a teaching hospital of northern India over a period of 8 years. J Family Med Prim Care. 2022 Feb;11(2):567-572.
13. Palewar MS, Joshi S, Choudhary G, Das R, Sadafale A, Karyakarte R. Prevalence of Hepatitis A virus (HAV) and Hepatitis E virus (HEV) in patients presenting with acute viral hepatitis: A 3-year retrospective study at a tertiary care Hospital in Western India. Journal of Family Medicine and Primary Care. 2022 Jun 1;11(6):2437-41.
14. Grover M, Gupta E, Samal J, Prasad M, Prabhakar T, Chhabra R, Agarwal R, Raghuvanshi BB, Sharma MK, Alam S. Rising trend of symptomatic infections due to Hepatitis A virus infection in adolescent and adult age group: An observational study from a tertiary care liver institute in India. Indian Journal of Medical Microbiology. 2024 Jul 1;50:100653.
15. Nair P, John R, Chithira KG. Seroprevalence of hepatitis E in healthy adults attending a tertiary care hospital in India. J Acad Clin Microbiol. 2019;21(2):80–84.
16. Joon A, Rao P, Shenoy SM, Baliga S. Prevalence of hepatitis A virus (HAV) and hepatitis E virus (HEV) in patients presenting with acute viral hepatitis. Indian J Med Microbiol. 2015;33 (Suppl):S102–S105.
17. Daniel HD, Warier A, Abraham P, Sridharan G. Seroprevalence of hepatitis E virus infection among pregnant women in South India. Int J Sci Res. 2023;10(7):1–4.
18. Netra S, Bithu R, Maheshwari RK. Epidemiological study of hepatitis A virus and hepatitis E virus infection in patients presenting with acute viral hepatitis. Int J Curr Microbiol App Sci 2018;7:899 904.
19. Kaur, M., Sidhu, S.K., Singh, K., Devi, P., Kaur, M., & Singh, N.J. (2017). Hepatitis E virus: A leading cause of waterborne viral hepatitis in Northwest Districts of Punjab, India. Journal of Laboratory Physicians, 9(2), 121–124.
20. Murhekar MV, Ashok M, Kanagasabai K, Joshua V, Ravi M, Sabarinathan R, et al. Epidemiology of hepatitis A and hepatitis E based on laboratory surveillance data India, 2014 2017. Am J Trop Med Hyg2018;99:1058 61.
21. Rawat SK & Jain A. Seroprevalence of hepatitis A and E virus IgM in children suffering from acute hepatitis. J Med Sci Clin Res. 2015;3(3):4616–4620.
22. Mittal G, Gupta P, Gupta R, Ahuja V, Mittal M, Dhar M. Seroprevalence and risk factors of hepatitis B and hepatitis C virus infections in uttarakhand, India. J Clin Exp Hepatol. 2013 Dec;3(4):296-300.
23. Kaur G, Arora S, Arora R, Singh V. Seroprevalence of hepatitis B virus among blood donors in Chandigarh, Punjab. Indian J Med Microbiol. 2010;28(3):297–8.
24. Giri S, Sahoo S, Angadi S, Afzalpurkar S, Sundaram S, Bhrugumalla S. Seroprevalence of Hepatitis B Virus Among Pregnant Women in India: A Systematic Review and Meta-Analysis. J Clin Exp Hepatol. (2022) 12(6):1408-1419.
25. Rajani M. Magnitude and pattern of HBV infection in

clinically suspected infectious hepatitis at a tertiary care hospital. Indian J Public Health. 2014; 58: 45–49.

26. Shadaker S, Sood A, Averhoff F, et al. Hepatitis B prevalence and risk factors in Punjab, India: A population-based serosurvey. J Clin Exp Hepatol. 2022;12(5):1310–1319.
27. Khan F, Akbar H, Idrees M, et al. The prevalence of HBV infection in the cohort of IDPs of war against terrorism in Malakand Division of Northern Pakistan. BMC Infect Dis. 2011;11:176.
28. Sharma B, Katiyar H, Barall D, et al. Genotyping of hepatitis B virus isolates from Lahaul and Spiti district in Himachal Pradesh, India. Indian J Gastroenterol. 2018;37(3):261–265.
29. Sandhu S, Bansal R, Kaur M, et al. Seroprevalence of hepatitis B virus infection among healthcare workers in a tertiary care hospital in North India. Indian J Med Microbiol. 2011;29(3):267–270.
30. Manzoor S, Akhtar S, Khan S, et al. Frequency and risk factors of hepatitis B and C in Afghan patients presenting to tertiary care hospital in Peshawar. J Pak Med Assoc. 2015;65(1):5–8.
31. Ray L, Sarna A, Sebastian MP, et al. HIV, Hepatitis B and C among people who inject drugs: high prevalence of HIV and Hepatitis C RNA positive infections observed in Delhi, India. BMC Public Health. 2015;15:726.
32. Bagga PK, Singh SP. Seroprevalence of hepatitis C antibodies in healthy blood donors—a prospective study. Indian J PatholMicrobiol. 2007; 50(2): 429–32.
33. Kar SK, Sabat J, Ho LM, Arora R, Dwibedi B. High Prevalence of Hepatitis C Virus Infection in Primitive Tribes of Eastern India and Associated Sociobehavioral Risks for Transmission: A Retrospective Analysis. Health Equity. 2019 Nov 1;3(1):567–72.
34. Meena M, Jindal T, Hazarika A. Prevalence of hepatitis B virus and hepatitis C virus among blood donors at a tertiary care hospital in India: a five-year study. Transfusion. 2011;51(1):198–202.
35. Chaurasia R, Sharma N, Das S, Titiyal JS, Tandon R. Comparison of seropositivity of human immunodeficiency virus, hepatitis B virus, hepatitis C virus, and syphilis among Hospital Cornea Retrieval Programme-Donors versus voluntary cornea donors at a large eye bank in Eastern India. Indian J Ophthalmol. 2017;65(12):1270–1274.
36. Mittal G, Gupta P, Gupta R, Ahuja V, Mittal M, Dhar M. Seroprevalence and risk factors of hepatitis B and hepatitis C virus infections in Uttarakhand, India. J Clin Exp Hepatol. 2013;3(4):296–300.
37. Tandon BN, Acharya SK, Tandon A. The prevalence of hepatitis C virus antibodies among the voluntary blood donors of New Delhi, India. Eur J Epidemiol. 2003;18(7):695–8
38. Barman B, Roy A, Nune A, Lyngdoh WV, Jamil M, Tiewsoh I. Epidemiology, clinical, and laboratory profile of patients with hepatitis C: A prospective, observational study from north-eastern India. Indian J Public Health. 2022;66(3):288–293.
39. Solomon SS, Mehta SH, Srikrishnan AK, et al. Epidemiology of HIV and hepatitis C infection among women who inject drugs in Northeast India: a respondent-driven sampling study. Addiction. 2017;112(3):737–745.
40. Bhadoria AS, Dhankhar A, Khwairakpam G, Grover GS, Pathak VK, Pandey P, Gupta R, GUPTA R. Viral hepatitis as a public health concern: a narrative review about the current scenario and the way forward. Cureus. 2022 Feb 4;14(2).
41. Kumar, D., Peter, R. M., Joseph, A., Kosalram, K., & Kaur, H. (2023). Prevalence of viral hepatitis infection in India: A systematic review and meta-analysis. Journal of Education and Health Promotion, 12, 103.

Case Report

BONE METASTASIS FROM ENDOMETRIOID OVARIAN CARCINOMA: A CASE STUDY AND LITERATURE REVIEW

Monika Garg, Rajni Bassi
Immunohaematology and Blood Transfusion

Corresponding Author : Dr. Rajni Bassi
E-mail : rajniajata@yahoo.com

Introduction

Ovarian epithelial malignancy rarely metastasize to the skeletal system. They usually spread by direct extension, transcoelemic spread and by lymphatic channel. Hematogenous spread is rare and late occurrence. Metastasis to bone from these tumours is rare. (0.1- 0.12%) and is reported in a few series. Only a few cases with bone metastasis have been reported at the initial presentation in patients with ovarian cancer.(1) Only 83 cases of primary disease with bone metastasis have been reported in the literature, the majority of which were autopsy findings. Bone metastasis discovered antemortem are the exception. We have registered 46 ovarian cancer cases in the department of pathology over the last 5 years and have not encountered single case of bone metastasis so far. The case is presented because of unique presentation, with no evidence of intraperitoneal disease or other metastasis

Case report

A 75 year old post menopausal lady presented in surgery department with chief complaints of swelling and pain over left thigh since 2 months. Swelling appeared 5 months back, it was drained at that time, but recurred 1 month back . According to patient , she was operated for fibroid 2 year back. A clinical diagnosis of antibioma was made. During surgery , a ill defined growth was seen extending deeply upto the bone. Excision biopsy of swelling was performed with few bony pieces.

Because of deep bony extension of the mass, pelvic MRI was performed which showed a large destructive soft tissue density mass lesion involving and arising from left pubic bones, left acetabulum with calcific foci and large central necrosis. So, a differential diagnosis of chondrosarcoma and metastasis in the pubic bone was made. There was no other evidence of metastasis and a search for second primary proved negative.

Grossly we received skin covered biopsy, partly solid and partly cystic in multiple pieces measuring 12x11.5x 2.5 cm. external surface of skin was ulcerated and cut surface showed necrotic areas. Two bony pieces were received separately.

Microscopic sections examined showed marked inflammatory response consisting of acute and chronic inflammatory cells, fibrosis, multi nucleatd giant cells, foamy macrophages, necrosis, myxoid degeration, haemorrhage and marked congestion.

Section from bony pieces showed deposits of tumour cells amongst normal bony trabeculae. Tumour cells were highly pleomorphic , with high N:C ratio, abnormal mitosis, and forming acini, papillae and few tubules.

So, a diagnosis of bony metastasis from unknown primary was made and clinicians were informed to search for primary in ovary, breast, GIT etc. serum CA 125 levels were increased to 230 U/ml.

The patients was asked to produce all the documents regarding previous surgery. Total abdominal hysterectomy with bilateral salpingoophrectomy was done 2 year back. And histopathological report showed leiomyoma of uterus and serous papillary cystadenocarcinoma of left ovary (Figure 1). Uterus , cervix , both the fallopian tubes and ometum was free of

tumour deposits. The patient was staged as having stage ia ovarian cancer(as per FIGO 1986 staging system). The patient was advised adjuvant chemotherapy , which she did not take.

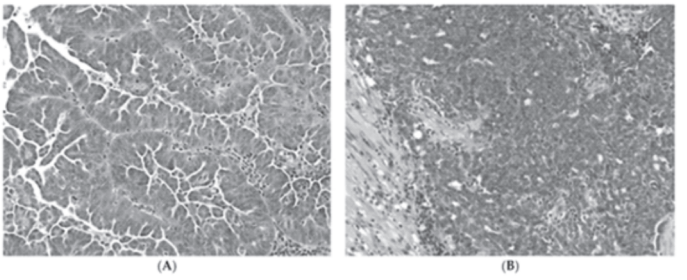


Figure 1: H & E sections revealing variable architectural features including papillary, micropapillary and solid growth patterns. The tumor cells typically exhibit marked nuclear pleomorphism with prominent nucleoli, high mitotic activity (typically > 12 mitoses per 10 high-power fields), including atypical mitoses.

Discussion

Ovarian cancer is the fourth most common type of breast cancer and fifth leading cause of cancer death in women in the united states. Estimated new cancer cases and death from ovarian cancer in united states in 2012 are 22,280 and 15,500 respectively. In India , during the period 2004- 2005, proportion of ovarian cancer varied from 1.7% to 8.7% of all female cancers in various urban and rural population based registries operating under the network of the National Cancer Registry Programme (NCRP) of Indian Council of Medical Research. Metastasis from ovarian cancer are usually implantation or lymphatic to pelvic and para aortic lymph nodes, with distal metastasis being rare and occurring late. Bone metastasis are extremely rare and other clinical series report an incidence of 0.1- 0.12%. (2)Autopsy study reveals bone metastasis in 6-14% cases.(2) Bone involvement occur more frequently in dysgerminoma , as opposed to epithelial tumours of the ovary. We also performed a retrospective chart review of last five years , and only one current case was found to have an initial presentation with bone metastasis among 46 ovarian cancer patients.

The median time to development of bone metastasis after the diagnosis of ovarian cancer is 74 months (range, 68 – 80 months). (3) In our study it is around 24 months therefore bone metastasis in this case may reflect its aggressive nature.

Bone metastasis in patients with ovarian carcinoma are usually associated with symptoms of bone pain and lesions tend to be focal and osteolytic, rarely osteoblastic. The most common sites of metastasis are vertebral bodies followed by ribs, clavicle, skull, femur.

Bone lesions do not usually occur in the absence of advanced disease, , ie. Stage iii or iv . Many cases have been reported in an autopsy , but there is scarcity of clinical cases. Brufman and Mettler etal confirmed this findings in clinical series, with all patients with bone metastasis presenting with stage iii or iv disease.

Hong etal analyzed 336 patients of distant metastasis from ovarian cancer. Of these, four patients had bone metastasis, two of which belonged to thoracic vertebra, one to the clavicle and one had bone marrow involvement. None of the patient in this series had bone metastasis as first site of presentation. In a study of 113 cases of ovarian carcinoma by Karim etal , no patients had bone lesions as the only manifestation of metastatic disease.(3)

Baize etal also reported a case of ovarian cancer which presented with lumber vertebral metastasis soon after treatment , as part of distant spread. (4)

The mean survival interval is 7.5 months (range, 6- 39 months) in patients with bone metastasis of ovarian cancer. The incidence of micro metastasis in the bone marrow could be higher if bone marrow biopsy were conducted on all the patients with ovarian cancer.

Conclusion

We present a rare case of ovarian cancer with an initial presentation of bone meatstasis. Bone metastasis of ovarian tumour is by hematogenous spread. Its identification at clinical presentation predicts treatment failure with a maximum survival of 7.5 month a s reported in the literature.

References

1. J. Sehouli, J. Olschewski, V. Schotters, C. Fotopoulou & K. Pietzner. Prognostic role of early versus late onset of bone metastasis in patients with carcinoma of the ovary, peritoneum and fallopian tube.Annals of Oncology, Volume 24, Issue 12, 3024 – 8
2. Zhang C, Guo X, Peltzer K, Ma W, Qi L, Zhang Y, Han X, Baklaushev VP, Yao Y, Wang G, Chekhonin VP, Wang

X, Ma Y. The prevalence, associated factors for bone metastases development and prognosis in newly diagnosed ovarian cancer: a large population based real-world study. J Cancer. 2019 Jun 2;10(14):3133-9.

3. Hong L, Cristiano L, Peters E, Ioffe Y. Detection of bone metastases in uterine cancer: How common are they and should PET/CT be the standard for diagnosis? Gynecol Oncol Rep. 2021 Jan 8;35:100698.
4. N. Baize, A. Mahamat, E. Benizri, M.C. Saint-Paul, N. Mounier.Bone metastasis from endometrioid ovarian carcinoma: a case study and literature review.Eur. J. Gynaec. Oncol.- 326-8.

Case Report

ACUTE MYELOID LEUKEMIA WITH BASOPHILIA WITH t(6;9) (p23;q34) – A RARE SUBTYPE OF AML.

Vandana Singla[1], Dr Rajni Bassi [2], Monika Garg[3]
Lecturer, Deptt of Pathology[1], Assistant Professor, Deptt of Blood Transfusion [2], Immunohaematology and Blood Transfusion[3]

Corresponding Author : Dr Rajni Bassi
Assistant Professor, Deptt of Blood Transfusion
E-mail : rajniajata@yahoo.com

Introduction:-

Acute Myeloid Leukemia (AML) with the translocation t(6;9) (p23;q34), involving breaks at band 23 of the short arm of chromosome 6 and band 34 of the long arm of chromosome 9, is a rare subtype, representing 0.5% to 4% of AML cases.1 This translocation leads to the creation of a chimeric fusion gene (DEK-NUP214) on the derivative chromosome 6, known as der(6). The translocation was first discovered in AML by Rowley and Potter in 19762, marking a key development in understanding AML genetics. Most patients with AML and the t(6;9) (p23;q34) translocation are classified under the French-American-British (FAB) system as having either AML-M2 or AML-M4. WHO classification of Acute Leukemias 2017 categorized this under AML with recurrent genetic abnormalities. Many of these patients exhibit signs of underlying or preceding myelodysplasia, contributing to the poor prognosis. Complete remission is achieved in only about 50% of cases with conventional chemotherapy with most patients surviving just one year after diagnosis. Bone marrow transplantation may be the only treatment option capable of achieving a potential cure in these cases.

Case Report:-

A 50-year-old male presented with a gradual onset of fever and a cough with expectoration lasting for one month, followed by episodes of hemoptysis and associated generalized weakness. He also reported history of blood transfusions and recurrent infections in the past. The persistent symptoms, along with the patient's medical history, raised concerns for an underlying chronic infection or hematological disorders.

Laboratory investigations - Hemoglobin levels : 8.7 g/dl

Peripheral blood smear (PBF) - Dimorphic red blood cell picture.

TLC- 58,900/cmm

DLC- 75% Blasts, 22% Basophils, 2% Lymphocytes, and 1% Polymorphs.

Platelet count -30,000/cmm.

Given these findings, particularly the high percentage of blasts and low platelets, the patient was advised to undergo a bone marrow biopsy for further evaluation and definitive diagnosis.

Another lab findings include- Increased serum alkaline phosphatase (S. Alk) levels of 152 IU/L, while SGOT , SGPT and RFT levels were within normal limits. There was no evidence of lymphadenopathy, splenomegaly, or hepatomegaly on examination.

Bone marrow findings:-

Cellularity- Moderately cellular

Reaction- Mild megaloblastic

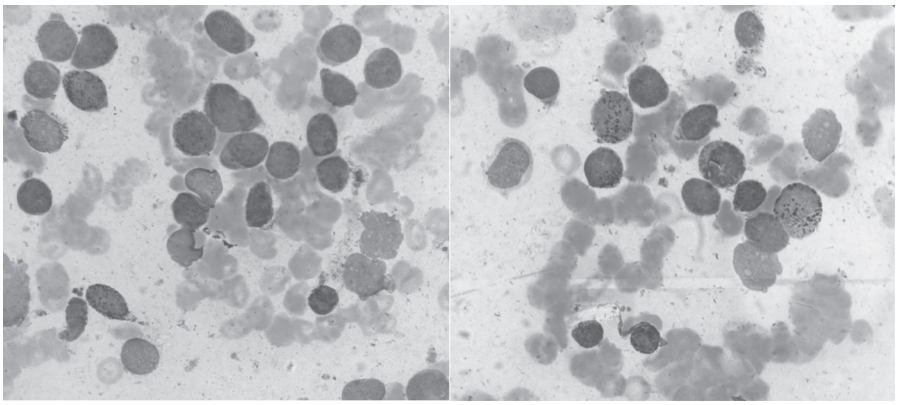
NE:E ratio- 98:2

Erythroid series: Markedly diminished with mild megaloblastic maturation and features of dyserythropoiesis seen.

Myeloid series: Cellularity of marrow is mainly due to medium sized Blasts(70%) showing basophilic cytoplasm with 1-4 conspicuous nucleoli. Scattered cells are seen covered with basophilic granules. Basophilic precursors (17%) are increased in number. Rest of the

myeloid series is markedly diminished.
Rare Megakaryocyte seen.
Impression:- Acute Myeloid Leukemia (AML) with Basophilia.
Advised: 1. FLOW CYTOMETRY
2. CYTOGENETICS for t(6;9) (p23;q34)

A cytogenetic study performed at a higher institute confirmed that the patient tested positive for the t(6;9) translocation.



Discussion:-

The bone marrow in cases of t(6;9) acute myeloid leukemia (AML) is typically hypercellular for the patient's age and shows increased number of blasts (≥20%), which may contain Auer rods or granules. Additionally, bone marrow basophilia and dysplasia, either unilineage or multilineage, can be observed in the residual hematopoietic cells. According to Alsabeh et al.3, there is also an increased incidence of ringed sideroblasts, suggesting features of myelodysplasia in these patients. The largest retrospective study4 to date, involving five cooperative groups (Southwest Oncology Group, Cancer and Leukemia Group B, Eastern Cooperative Oncology Group, Children's Oncology Group, and Children's Cancer Group), reported a prevalence of 44% for marrow basophilia and 67% for evidence of myelodysplasia in t(6;9) AML cases. Immunophenotypically, the blast cells in t(6;9) AML are positive for CD9, CD13, CD33, and HLA-DR; they are usually positive for CD45 and CD38 and may also express CD15, CD34, and terminal deoxynucleotidyl transferase, contributing to its diagnostic profile.5 Alsabeh et al. noted that blasts, initially presenting as CD34-ve, often relapsed as CD34+ve. The t(6;9) translocation is most commonly associated with acute myeloid leukemia (AML) of the FAB-M2 subtype and is sometimes considered a distinct disease entity due to its unique clinical and morphologic features include marrow basophilia and evidence of myelodysplasia, as well as its poor prognosis.

Conclusion:-

Patients diagnosed with t(6;9) acute myeloid leukemia (AML) generally have a very poor prognosis, as current chemotherapy options have not shown significant improvement in overall survival rates. However, early and accurate diagnosis is essential, as these patients may benefit from early allogeneic stem cell transplantation, which can improve outcomes. There is a growing need to explore novel therapies, such as anti-CD33–based treatments and FLT3 inhibitors, to potentially enhance treatment efficacy for t(6;9) AML patients. Furthermore, molecular monitoring of minimal residual disease (MRD) is a valuable tool for assessing risk stratification and guiding disease management. The World Health Organization's classification of hematopoietic tumors highlights the importance of understanding the prognostic implications of cytogenetic abnormalities in hematologic malignancies. Given its distinct morphologic, cytogenetic, and clinical characteristics, AML with t(6;9) (p23;q34) should be recognized as a separate entity within the classification of AML with recurrent cytogenetic abnormalities. This recognition emphasizes the need for tailored treatment approaches

and continued research to address the unique challenges associated with this rare but aggressive subtype of AML.

References:-

1. Schwartz S, Jiji R, Kerman S, Meekins J, Cohen MM. Translocation (6;9)(p23;q34) in acute nonlymphocytic leukemia. Cancer Genet Cytogenet. 1983;10:133–138.

2. Rowley JD, Potter D. Chromosomal banding patterns in acute nonlymphocytic leukemia. Blood. 1976;47:705–721.

3. Alsabeh R, Brynes RK, Slovak ML, et al. Acute myeloid leukemia with t(6;9)(p23;q34): association with myelodysplasia, basophilia, and initial CD34 negative immunophenotype. Am J Clin Pathol. 1997;107:430–437.

4. Slovak ML, Gundacker H, Bloomfield CD, et al. A retrospective study of 69 patients with t(6;9)(p23;q34) AML emphasizes the need for a prospective, multicenter initiative for rare 'poor prognosis' myeloid malignancies. Leukemia.2006;20:1295–1297.

5. Oyarzo MP, Lin P, Glassman A. et al. Acute myelogenous leukemia with t(6;9)(p23;q34) is associated with dysplasia and a high frequency of FLT3 gene mutations. Am J Clin Pathol. 2004;122:348–358.

Case Report

RARE ADRENAL INCIDENTALOMA: GANGLIONEUROMA

H.S. Rekhi, Malkiat Singh, Sudesh Partap, Baljeet Kaur, Samrat Singh, Shubham Chhabra

Corresponding Author : Dr Rajat Talesra
E-mail : talesrarajat@gmail.com

Abstract

Adrenal ganglioneuromas (AGNs) are rare, benign, well-differentiated tumors of neural crest origin. They are usually hormonally non-functional and asymptomatic, frequently discovered incidentally during imaging for unrelated concerns. We present the case of a 19-year-old female who exhibited vague gastrointestinal and autonomic symptoms. Imaging revealed a right adrenal mass suggestive of malignancy. However, hormonal evaluation was unremarkable. Surgical excision was performed, and histopathology confirmed the diagnosis of adrenal ganglioneuroma. This case underscores the diagnostic challenge of such rare adrenal incidentalomas and highlights the importance of multimodal imaging and histopathology in guiding management.

Introduction

Adrenal incidentalomas are adrenal masses incidentally discovered during imaging for non-adrenal indications. With the widespread use of ultrasonography and cross-sectional imaging, their detection rate has significantly increased[1]. Most incidentalomas are benign adrenocortical adenomas, but rare tumors such as ganglioneuromas must also be considered.

Adrenal ganglioneuromas (AGNs) are rare, benign neurogenic tumors derived from neural crest cells. They represent the most mature form in the spectrum of neuroblastic tumors, which includes neuroblastomas and ganglioneuroblastomas[2]. AGNs account for about 20% of all ganglioneuromas and often occur in adolescents and young adults[3]. These tumors are usually hormonally inactive and asymptomatic, often discovered incidentally or when large enough to cause mass effect.

Due to their rarity and non-specific presentation, AGNs pose a diagnostic challenge. Imaging can mimic malignant adrenal tumors, and definitive diagnosis is often made postoperatively via histopathology[4]. This case report illustrates such a scenario.

Case Report

A 19-year-old female presented to the Surgery OPD with complaints of episodic diarrhea and vomiting for the past three months. She also reported dull aching pain in the right epigastrium and right flank for four weeks. Additionally, she experienced episodes of tachycardia, palpitations, chronic sweating, perioral pigmentation, and intermittent headaches.

On general examination, perioral pigmentation was noted. Abdominal and systemic examinations were within normal limits.

Ultrasonography of the abdomen revealed a hypoechoic mass measuring 45 × 38 mm with calcifications in the right adrenal area, abutting the upper pole of the right kidney. Based on this, a functional workup was initiated, which revealed:

- Serum ACTH: 24.3 pg/mL (normal <46 pg/mL)
- Serum cortisol: 13.28 µg/dL (normal 6.72–22.6 µg/dL)
- 24-hour urinary VMA: 8.41 mg/24 hrs (normal

0.4–15.44 mg/24 hrs)

All values were within normal limits, suggesting a non-functional adrenal lesion.

A contrast-enhanced CT (CECT) of the abdomen showed a well-defined, heterogeneously enhancing soft tissue lesion measuring 4.9 cm (AP) × 3.4 cm (T) × 4.2 cm (CC) in the right adrenal region, with an average attenuation of 35–40 HU. The lesion showed calcific foci, abutted the liver (segment VI) with loss of fat planes, indented the upper pole of the right kidney, and contacted the IVC and right crus of the diaphragm. These radiologic features raised suspicion of pheochromocytoma or adrenocortical carcinoma.

Given the inconsistency between the non-functional biochemical profile and radiological suspicion of malignancy, contrast-enhanced MRI (CEMRI) was done. It suggested a differential of adrenal ganglioneuroma or atypical adenoma.

Due to the tumor's size, imaging features, and associated symptoms, an open right adrenalectomy was performed via a posterolateral approach. The mass was excised completely and intact, with no evidence of local invasion.

Postoperatively, the patient had an uneventful recovery. Clear fluids were started the evening after surgery, followed by semisolids on the first postoperative day. She was discharged on the fourth day in satisfactory condition, and sutures were removed on postoperative day twelve.

Histopathological examination (H&E stain) revealed a well-encapsulated tumor composed of Schwannian spindle cells arranged in fascicles and parallel bundles, with scattered large polygonal ganglion cells possessing eosinophilic cytoplasm and prominent nucleoli. The stroma showed mixed inflammatory infiltrates and dystrophic calcification. Peripherally compressed normal adrenal tissue was identified. These findings confirmed the diagnosis of adrenal ganglioneuroma.

As the tumor was benign, no further imaging surveillance was pursued.

Discussion

Adrenal ganglioneuromas are rare, benign tumors originating from sympathetic ganglia and are considered the most differentiated form of neuroblastic tumors[2]. They are more commonly found in the posterior mediastinum and retroperitoneum, with adrenal involvement being uncommon[5].

AGNs usually occur in patients over the age of 10 and are more common in females[3]. They are typically non-functional, although rare cases of hormonal secretion (catecholamines, VIP, or cortisol) have been reported[6]. In our case, although the patient presented with symptoms resembling catecholamine excess, including palpitations, headaches, and sweating, the functional workup was normal.

Imaging plays a crucial role in the detection and preliminary evaluation of AGNs. On CT, they often appear as well-circumscribed, hypodense lesions with mild to moderate enhancement and may contain punctate or coarse calcifications[7]. MRI provides better tissue characterization; AGNs generally show low signal intensity on T1 and high signal intensity on T2-weighted images due to their myxoid content[8].

However, imaging findings are often non-specific, and AGNs may mimic malignant lesions such as adrenocortical carcinoma or pheochromocytoma, as was suspected in our case. Hence, histopathological examination remains the gold standard for diagnosis. The presence of mature ganglion cells, spindle-shaped Schwann cells, and absence of mitotic figures or necrosis confirms the diagnosis[6].

Surgical excision is both diagnostic and therapeutic. Open adrenalectomy is preferred for large tumors or when malignancy cannot be ruled out, while laparoscopic adrenalectomy may be reserved for smaller, well-demarcated lesions[9]. Complete resection typically results in an excellent prognosis, with rare recurrence or malignant transformation.

Conclusion

Adrenal ganglioneuroma is a rare, benign tumor that should be considered in the differential diagnosis of adrenal incidentalomas, especially in young patients with atypical symptoms. Although often asymptomatic, these tumors may present with non-specific gastrointestinal or autonomic features. Radiologic evaluation can raise suspicion but may not definitively distinguish AGNs from malignant tumors. Surgical excision remains the cornerstone of diagnosis and

management, offering excellent outcomes when complete resection is achieved. Awareness and early recognition of this rare entity are essential to avoid unnecessary anxiety and overtreatment.

References

1. Bovio S, Cataldi A, Reimondo G, et al. Prevalence of adrenal incidentaloma in a contemporary computerized tomography series. J Endocrinol Invest. 2006;29(4):298–302.
2. Geoerger B, Hero B, Harms D, et al. Metabolic activity and clinical features of primary ganglioneuromas. Cancer. 2001;91 (10): 1905–1913.
3. Linos D, Stylopoulos N, Vlahos N, et al. Adrenal ganglioneuromas: incidental findings and clinical significance. World J Surg. 1997;21(8):850–854.
4. Yildiz L, Akça T, Vardar E, Yilmaz N. Adrenal ganglioneuroma: a rare tumor. Endocr J. 2003; 50(5):613–616.
5. Ichikawa T, Ohtomo K, Araki T, et al. Ganglioneuroma: computed tomography and magnetic resonance features. Br J Radiol. 1996;69(827):114–121.
6. Hayes FA, Green AA, Rao BN. Clinical manifestations of ganglioneuroma. Cancer. 1989; 63(6): 1211–1214.
7. Lonergan GJ, Schwab CM, Suarez ES, Carlson CL. Neuroblastoma, ganglioneuroblastoma, and ganglioneuroma: radiologic-pathologic correlation. Radiographics. 2002;22(4):911–934.
8. Kinoshita Y, Kurata A, Kinoshita F, et al. MR imaging of adrenal ganglioneuroma: report of two cases. Radiat Med. 1999;17(6):441–444.
9. Gagner M, Lacroix A, Bolté E. Laparoscopic adrenalectomy in Cushing's syndrome and pheochromocytoma. N Engl J Med. 1992;327 (14):1033.

Case Report

MANAGEMENT OF UNDIAGNOSED CASE OF SNAKEBITE PATIENT WITH RESPIRATORY FAILURE IN ICU: A CASE REPORT

Veena Chatrath, Ranjana Khetarpal, Anju Bala, Aarzoo Kamboj, Mallika Gupta
Govt Medical College, Amritsar

Corresponding Author: Dr.Veena Chatrath
Govt Medical College, Amritsar

INTRODUCTION

Venomous snakebite is an acute life -threatening emergency. Snakebite is most common among agricultural workers and children especially in rural areas all over the world.[1] The snake venom can be neurotoxic, hematotoxic or myotoxic. Rarely, snakebite can be occult where history is not clear with no visible bite marks which delays the diagnosis and complicates management. In such cases, clinical suspicion is of utmost importance for unexplained neuroparalysis. Sometimes, neuromuscular paralysis is so severe that it mimics brain death. In north India, the two important neurotoxic snakes leading to muscle paralysis are Cobra and Common krait. Due to respiratory muscle paralysis these patients require ventilator support in addition to anti-snake venom. We present a case of suspected snake bite presenting with loss of consciousness and h/o vomiting and remained in comatose state for over 4 days before gradual recovery.

CASE REPORT

A 20 years old male was brought to medicine emergency of Guru Nanak Dev Hospital, Amritsar on 19/8/2024 with sudden loss of consciousness.

GCS - E1V1M2 i.e.4/15 on arrival

Pupils - fully dilated and unresponsive to light.

O/E, BP- 136/90 mmHg, **PR-** 124/min, **Chest-** clear, B/L A/E equal, **CVS-** S1S2 normally heard.

HOPI- Patient had H/O 2 episodes of vomiting 2 hours back.

Past History- No significant past history.

ECG- normal

MANAGEMENT-

The patient was immediately intubated and was mechanically ventilated using control mode of mechanical ventilation. Supportive treatment was initiated. Enteral feeding was initiated via Ryle's tube after confirming bowel sounds. CT Head and MRI brain showed no significant findings. Urine for toxicology was negative. The patient remained in comatose state for 4 days and showed no change in neurological status. Keeping in mind the season of snake bite and after ruling out other neurological conditions on CT and MRI, we started managing the patient empirically with neostigmine and glycopyrrolate. 20- minute whole blood clotting test (20WBCT) was normal. In the absence of evidence and H/O snakebite along with 4 days of previous H/O unconsciousness, no ASV were administered. Inj. Myopyrrolate infusion was started on day 4 at 0.5 mg/hr. On 5th day, patient was noted being able to move the fingers. On 7th day patient developed cough reflex to ET suctioning while pupils were still unresponsive to light. On day 8, spontaneous eye opening was observed and patient was able to move arms and hands. Inj. Neostigmine-glycopyrrolate was stopped. Mechanical ventilation was continued for another 4 days using SIMV-VC mode (TV-6 ml/kg body wt.) due to persistent respiratory muscle weakness. On day 12th, patient was successfully weaned off from mechanical ventilation. Chest physiotherapy and spirometry was initiated and patient was shifted to ward after 15 days of admission.

INVESTIGATIONS-

Day in ICU	1	2	3	4	5	6	7	8	9	10	11	12
Hb (g/dl)	11.2	11.0	10.9	11.1	10.8	11.2	11.0	10.9	11.2	11.4	11.3	11.5
TLC (*10^3per mm^3)	9.08	9.90	10.45	11.67	13.06	18.68	16.28	11.32	11.93	8.09	8.06	7.06
Plt (*10^5 per mm^3)	3.4	2.68	2.28	1.34	1.86	2.36	2.28	3.02	3.36	3.66	2.98	2.66
PTI	92%				94.4%							93.6%
RBS	116	98	108	128	132	119	106	129	118	100	107	115
S.creatinine (mg/dl)	0.68	0.77	0.74	0.34	0.46	0.55	0.69	0.76	0.8	1.0	0.9	1.1
B. urea(mg/dl)	26	32	30	29	24	26	27	20	29	22	28	32
S. Na+(mmol/l)	133	131	134	131	133	138	136	133	139	138	133	135
S.K+(mmol/l)	3.3	3.7	3.6	3.6	3.4	3.9	3.7	4.7	3.9	3.6	3.8	4.1
ABG												
pH	7.42	7.36	7.42	7.33	7.41	7.429	7.45	7.39	7.38	7.36	7.32	7.33
pCO2 (mmHg)	36.6	47.1	34.5	39.5	33.1	38.2	33.8	37.8	37.3	38.6	30.8	32.6
pO2 (mmHg)	172	192	152	204	189	196	180	190	206	179	188	183

DISCUSSION

In India, there are 216 species, out of which 52 are

poisonous.[2] The most commonly encountered poisonous snakes in our country include Cobra, Common Krait, Russell's viper and Saw scaled viper. Cobra and Krait bite is neuroparalytic whereas Viper snakebite is vasculotoxic. The annual death rate due to snakebite in India is estimated to be 4.1 per 1,00,000 population. High mortality is due to lack of proper health services in rural areas and delay in reaching a well-equipped health care facility where anti-snake venom can be administered.[3,4] The superstitions prevailing in our country regarding snakebite management also delay emergency treatment. India has the highest number of deaths due to snake bite in the world with 35,000-50,000 people dying every year.[5,6]The chances of patient survival largely depend on the access to proper first aid and availability of supportive treatment and anti-snake venom for snakebite. It has been reported in previous studies that snakebites presenting with early morning paralysis are usually misdiagnosed as guillian-barre syndrome due to similar features like respiratory failure and flaccid paralysis.[7,8] Occult snakebite leads to diagnostic challenges thus, delaying the management. Neurotoxic snake bite blocks neuromuscular transmission resulting in muscle paralysis. Common krait bite is associated with profound neuromuscular paralysis with few local signs/symptoms. Management protocol includes adequate ventilation, anti-snake venom, appropriate antibiotics and general supportive care.in neurotoxic snake bite, Inj. Neostigmine-glycopyrrolate is of great help.

CONCLUSION

A significant number of patients die before they reach the hospital due to the fact that most of the victims are unaware that they have been bitten due to lack of local symptoms/pain. Timely recognition and early intervention with mechanical ventilation and respiratory support, early administration of ASV and Neostigmine-glycopyrrolate is life saving.

REFERENCES

1. Gutierrez JM, Calvete JJ, Habib AG, Harrison RA, Williams DJ, Warrell DA. Correction: Snakebite envenoming. Nat Rev Dis Primer. 2017 Oct 5;3(1):17079.
2. Bhattacharya P. Chakraborty A; Neurotoxic snake bite with respiratory failure. Indian J Crit Care Med., 2007; 11:161-164.
3. Gupta YK, Peshin SS. Snake bite in India: Current scenario of an old problem. J Clin Toxicol. 2014;4:1000182.
4. Mithra S. Snake bite in India and its management. J Indian Medical Assoc. 1987;85:129-131.
5. Chippaux JP (1998) Snake-bites: appraisal of the global situation . Bull World Health Organ 76: 515-524.
6. Kashuriratne A, Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, et al. (2008) The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. PLoS Med 5:e218.
7. Paul ME. Occult Snake Bite: A Neurological Dilemma. Int J Sci Res. 2022;11(1):1-4.
8. Lizarzaburu-Ortiz C, Yumi G, Carvajal A, Pachacama AB, Berrazueta A, Rojas E. A Rare and Urgent Consequence after a Snake Bite. Cureus. 2022;14(2):e21910.

Case Report

A HIDDEN FOOT MELANOMA'S PATH TO THE INGUINAL NODES: DIAGNOSTIC PITFALLS AND THERAPEUTIC CONSIDERATIONS

Ashwani Kumar[1],Tejinder Paul Singh, Parth Dhamija[2], Navneeth S[3], Dinesh Kumar Pasi, Jaswinder Singh[4],
Professor & Head[1] S.R.[2], J.R.[3], Assoc. Professor[4],
Deptt of General & Minimal Access Surgery, GMC & Rajindra Hospital, Patiala

Corresponding : Dr. Tejinder Paul Singh, S.R.
Deptt of General & Minimal Access Surgery, GMC & Rajindra Hospital, Patiala
E-mail : drtejinder@gmail.com

Abstract

Malignant melanoma of the lower limb and foot frequently metastasizes to the inguinal lymph nodes, carrying a poor prognosis akin to other tumors with distant spread. Due to patient unawareness, foot melanomas are often misdiagnosed or undetected, delaying diagnosis. In rare instances, the popliteal fossa serves as the initial site of nodal metastasis, with popliteal metastasis rates in lower extremity melanomas ranging from 0.3% to 7%. Concurrent popliteal and inguinal lymph node metastases are exceptionally uncommon. We report the case of a 67-year-old woman presenting with a right inguinal mass, later diagnosed with metastatic malignant melanoma following surgical excision. Further history revealed a prior excision of a lesion on the right sole of the foot without histopathological analysis, suggesting an undiagnosed primary melanoma. This case underscores the diagnostic challenges of foot melanoma and the rare potential for atypical metastatic patterns.

Key Words: Malignant melanoma, Inguinal lymph node metastasis, Lower extremity melanoma, Misdiagnosis

Introduction

Malignant melanoma is a highly aggressive skin cancer, with a median overall survival of less than one year for metastatic cases[1]. Melanoma below the knee has a unique epidemiological profile, with studies indicating a higher prevalence among women and individuals in their fifties and sixties[2]. Lesions on the sole are often misdiagnosed as other skin conditions, with misdiagnosis rates ranging from 25% to 66%[4,5]. More obvious lesion involving areas like face typically prompt faster action from patients and clinicians[4]. Delayed diagnosis due to misidentification is critical, as distant lymph node metastasis significantly worsens survival outcomes[6]. However, further research is needed to explore whether plantar melanoma may follow atypical drainage patterns, potentially contributing to delayed detection of metastasis.

This report describes the case of an elderly woman who presented with a lump in the right inguinal region. Following evaluation, she underwent an excisional biopsy, which revealed melanomatous deposits. Further investigation uncovered a prior history of a skin lesion on the right sole of her foot, previously excised at a local hospital without histopathological analysis. This correlation shows that the older lesion was malignant melanoma. This case highlights the potential for delayed diagnosis of foot melanoma due to its inconspicuous location.

Case Summary

A 67-year-old postmenopausal woman with a history of poorly controlled type 2 diabetes mellitus presented to the surgical outpatient department with a three-month history of a right inguinal mass. The mass had an insidious onset and was progressively increasing in size. It was painless, non-tender, and showed no change in size with coughing or straining. The patient denied any history of trauma, fever, bleeding or discharge per rectum or vaginum, regression in the size of the lump, or multiple sexual partners.

Additionally, the patient reported a history of a raised, black-brown lesion on the sole of her right foot with history of intermittent bleeding from it. This lesion was reportedly excised at a local medical facility one year ago, but no records regarding the procedure or

histopathological examination were available. On physical examination, a firm to hard, non-pulsatile mass measuring 5x5 cm was identified in the right inguinal region. The mass had limited mobility from underlying structures, was not fixed to the overlying skin, and did not demonstrate a cough impulse. (Figure A)

Figure A

Investigations

FNAC of the groin lesion: atypical cells with high N:C ratio around vesicular nuclei and prominent nucleoli with abundant cytoplasm. Few cells with eccentric nuclei and few with cytoplasmic projections were also seen, findings were those of malignant aspirate possibly squamous in origin.

Ultrasound Right Inguinal Region: a large conglomerated lymph node with central echogenic material measuring 2.7cm in SAD in right inguinal region likely necrotic lymph node (figure B).

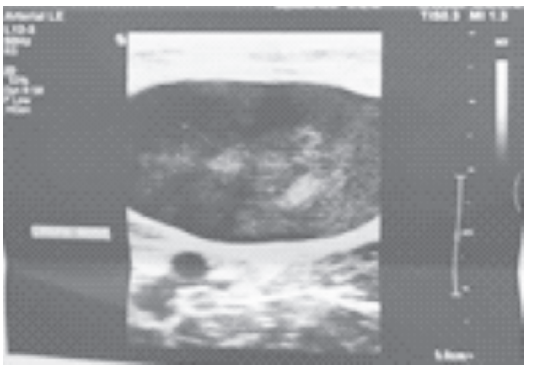


Figure B

PET-CT was done to find the primary lesion and to rule out any other lesions. The findings were suggestive of hyper metabolic lymph node mass in the right inguinal region measuring 5.2 x 4.6cm (SUV max 7.04) favoring mitotic pathology. No abnormal hyper metabolic lesion elsewhere in the body (figure C).

Figure C

MRI Pelvis and Right Inguinal Region: Heterogeneously hyperintense lesion is seen in right inguinal region, showing restriction on DWI. On post contrast scan showed vivid enhancement, likely lymph nodal mass. Anteriorly it is causing contour bulge, posteriorly it is indenting pectineus muscles, however intervening fat planes are maintained. Altered T2 signal intensity is seen in the skin overlying this lesion suggestive of edema (figure D).

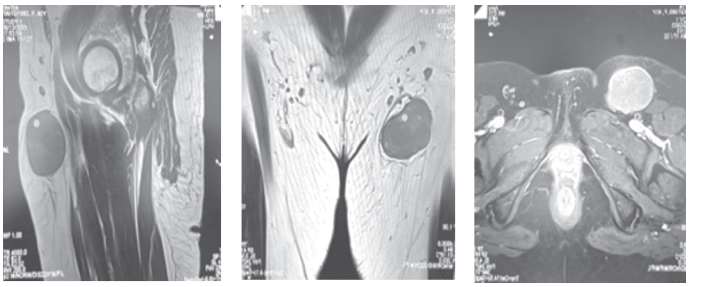


Figure D: (Green dots showing the lesion in Sagittal, Coronal and Axial sections)

Procedure Done

The patient underwent a wide local excision of right groin mass and the specimen was sent for histopathological examination (Figure E1, E2, E3).

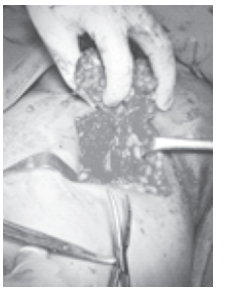


Figure E1
Excision of right inguinal mass

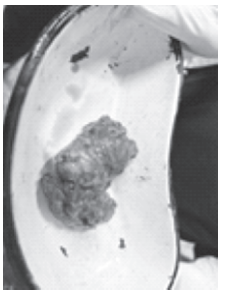


Figure E2
Excised specimen of right inguinal mass

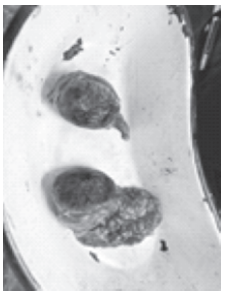


Figure E3
Excised specimen of right inguinal mass (gross examination)

Histopathology

Inguinal Lymph node with H&E stain shows replacement of lymphoid deposits with metastatic deposits of malignant melanoma. High power view shows malignant cells with intracytoplasmic melanin deposits (Figure F1, F2).

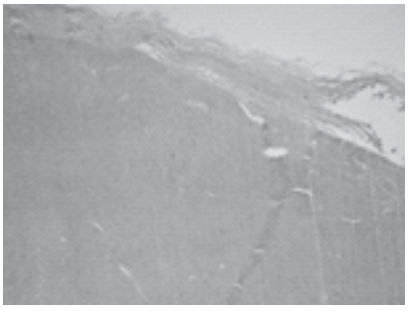


Figure F1
HPE of right inguinal mass
(H&E stain in 200x)

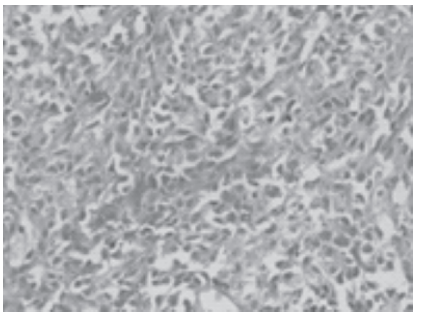


Figure F2
HPE of right inguinal mass
(H&E stain in 400x)

Discussion

Costa SR et.al (2008) suggested plantar melanoma has a particularly poorer prognosis compared to other sites of the body and clinical differentiation between early metastatic melanoma and melanocytic nevus is sometimes very difficult due to similar presentation. These factors lead to delay diagnosis which eventually results in development of thicker lesions and metastasis.[6]

Popliteal lymph nodes are the first station for the lymphatic drainage of squamous carcinomas and sarcomas of lower leg.[8-10] Metastases from below-knee melanomas commonly go through popliteal nodes and then arrive to inguinal lymph node stations. In our case, the FNAC initially suggested squamous origin and the patient underwent wide local excision of the inguinal mass. Further histopathological examination revealed metastatic deposits of malignant melanoma. There was no evidence of any active melanoma at presentation, the palpable inguinal mass corresponded to be the primary disease.

Only 2.5% of patients with lower extremity melanoma had isolated inguinal node metastasis, while 11.8% had combined popliteal and inguinal node metastasis2. Patients with lower leg melanomas have a survival advantage as high as 25% following prophylactic lymph node dissection.[8] As the histopathology demonstrated melanotic deposits, we later did a prophylactic Popliteal lymph node dissection with complete inguinal lymph node dissection because of the conferred survival advantage as well as the possibility of a false-negative PET scan result.

This case highlights the importance of early detection and diagnosis of melanoma, especially in acral areas, such as the sole of the foot, where the disease is often neglected or misdiagnosed. Early diagnosis of melanoma can improve the prognosis and survival of the patients. Therefore, it is essential to educate the public and the healthcare providers about the risk factors, signs, and symptoms of melanoma, and to promote the regular skin examination and biopsy of any suspicious lesions.

Bibliography

1. Jemal A, Siegel R, Xu J, Ward E. Cancer statistics, 2010. CA Cancer J Clin. 2010;60(5):277–300.
2. Piero C, Barberini F, De Giorgi V, Anania G, Santoro A, Bruzzone P, et al. The lymphatic drain of below-knee malignant melanoma: Is the popliteal Fossa a ghost station? Indian J Surg. 2021;83(6):1364–72.
3. Saida T. Malignant melanoma on the sole: How to detect the early lesions efficiently. Pigment Cell Res. 2000;13(s8):135–9.
4. Samdariya S, Kumar D, Vishwanathan C, Ahmed I. An unusual case of masquerading malignant melanoma. Clin Cancer Investig J. 2015.
5. Baumert J, Plewig G, Volkenandt M, Schmid-Wendtner M-H. Factors associated with a high tumour thickness in patients with melanoma. Br J Dermatol. 2007;156(5):938–44.
6. Costa SR, Horta SHC, Henriques AC. Popliteal lymphadenectomy for treating metastatic melanoma: case report. Sao Paulo Med J. 2008;126(4):232–5.

7. Wright S, Armeson K, Hill EG, Streck C, Leddy L, Cole D, et al. The role of sentinel lymph node biopsy in select sarcoma patients: a meta-analysis. Am J Surg. 2012;204(4):428–33.
8. Lhote R, Lambert J, Lejeune J, Gottlieb J, Badaoui A, Battistella M, et al. Sentinel lymph node biopsy in cutaneous squamous cell carcinoma series of 37 cases and systematic review of the literature. Acta Derm Venereol. 2018;98(7):671–6.
9. Morcos BB, Hashem S, Al-Ahmad F. Popliteal lymph node dissection for metastatic squamous cell carcinoma: a case report of an uncommon procedure for an uncommon presentation. World J Surg Oncol. 2011;9(1):130.
10. Silberman AW. Malignant melanoma. Practical considerations concerning prophylactic regional lymph node dissection. Ann Surg. 1987;206(2):206–9.

Life Members

Journal Club Government Medical College Patiala

Registered under Societies Act XXI OF 1860 – No. DIC/PTA/SOCIETY/586 of 2016-17

Official Publication:

GMC Patiala Journal of Research and Medical Education

Life Membership Fee:

Single Member: Rs. 5000/- & Couple: Rs. 8000/-

Cash / Cheque in favour of Journal Club Government Medical College Patiala

SBI RH Patiala Account No. 65277883145 – IFS Code: SBIN0050263

	ANATOMY		
1.	Dr. Harsimarjit Kaur	-	LM/H-3/2017
2.	Dr. Rajan Singla	-	LM/R-4/2016
3.	Dr. Manju Bala	-	LM/M-9/2021
	BIOCHEMISTRY		
4.	Dr. Maninder Kaur	-	LM/M-1/2016
	PHYSIOLOGY		
5.	Dr. Avnish Kumar	-	LM/A-3/2017
6.	Dr. Anupinder Thind	-	LM/A-7/2020
7.	Dr. Gagneen Kaur Sandhu	-	LM/G-4/2020
	PHARMACOLOGY		
8.	Dr. Anjleen Kaur	-	LM/A-1/2016
9.	Dr. Vijay Sehgal	-	LM/V-4/2016
10.	Dr. Neetu Sharma	-	LM/N-4/2021
11.	Dr. Ranjana	-	LM/R-6/2021
	PATHOLOGY		
12.	Dr. Mohanvir Kaur	-	LM/M-2/2016
13.	Dr. Navneet Kaur	-	LM/N-1/2017
14.	Dr. Vijay Bodal	-	LM/V-3/2016 (Couple S-2)
15.	Dr. Vandna Singla	-	LM/V-5/2017
	MICROBIOLOGY		
16.	Dr. Ashok Kumar	-	LM/A-6/2017
17.	Dr. Rupinder Kaur Bakshi	-	LM/R-2 /2016
	FORENSIC MEDICINE & TOXICOLOGY		
18.	Dr. D S Bhullar	-	LM/D-1/2016
19.	Dr. K K Aggarwal	-	LM/K-2/2016
20.	Dr. Preetinder Singh Chahal	-	LM/P-7/2020
21.	Dr. Satinder Pal Singh	-	LM/S-8/2020
	COMMUNITY MEDICINE		
22.	Dr. Puneet Gambhir	-	LM/P-2/2016 (Couple S-1)
	MEDICINE		
23.	Dr. Ardaman Singh	-	LM/A-5/2017
24.	Dr. B L Bhardwaj	-	LM/B-1/2016 (Couple K-1)
25.	DR R P S Sibia	-	LM/R-3/2016

26.	Dr. Sanjay Goyal	-	LM /S-4/2017
		PEDIATRICS	
27.	Dr. Baljinder Kaur	-	LM/B-4/2017
28.	Dr. Gursharan Singh	-	LM/G-3/2017
29.	Dr. Parveen Mittal	-	LM/P-3/2017
30.	Dr. Deeksha Singla	-	LM/D-7/2021
		CHEST & TB	
31.	Dr. Vishal Chopra	-	LM/ V-1/2016
32.	Dr. Sudesh Kumari	-	LM/S-9/2020
		SKIN	
33.	Dr. Dimple Chopra	-	LM/D-2/2016 (Couple V-1)
34.	Dr. Seema Goyal	-	LM /S-3/2017
		PSYCHIATRY	
35.	Dr. B S Sidhu	-	LM/B-2/2016
36.	Dr. Neeraj Mittal	-	LM/N-2/2020
		SURGERY	
37.	Dr. Darshanjit Singh Walia	-	LM/D-4/2017
38.	Dr. H S Rekhi	-	LM/H-I /2016
39.	Dr. Kuldeep Singh Bhatia	-	LM/K-4/2017
40.	Dr. Paras Pandove	-	LM/P-4/2017
41.	Dr. Prem Chand Singla	-	LM/P-5/2017
42.	Dr. Anjana Garg	-	LM/A-8/2021
43.	Dr. Sanjeev Gupta	-	LM/S-11/2021
44.	Dr. Jaswinder Singh	-	LM/J-1/2021
45.	Dr. Dinesh Kumar Passi	-	LM/D-5-2021 (Couple M-9)
46.	Dr. Anand Singla	-	LM/A-12/2021
		ORTHOPEDICS	
47.	Dr. Amandeep Singh Bakshi	-	LM/A-4/2017
48.	Dr. Bhupinder Singh Brar	-	LM/B-5/2017
49.	Dr. Garish Sahni	-	LM/G-1/2017
50.	Dr. Hari Om Aggarwal	-	LM/H-2/2016
51.	Dr. H K S Chawla	-	LM/H-4/2019
52.	Dr. Kuldeep Singh Sandhu	-	LM/K-5-2021
53.	Dr. Ravisha Bhardwaj	-	LM/R-8-2022
		EYE	
54.	Dr. Gursatinder Singh	-	LM/G-2/2017
55.	Dr. Manpreet Kaur Walia	-	LM/M-7/2020
56.	Dr. Chiman Lal	-	LM/C-1/2020
		ENT	
57.	Dr. Dimple Sahni	-	LM/D-3/2017 (Couple G-1)
58.	Dr. Manjit Singh	-	LM/M-6/2017
59.	Dr. Sanjeev Bhagat	-	LM/S-5/2017
60.	Dr. Parvinder Singh	-	LM/P-9/2021
		UROLOGY	
61.	Dr. Harjinder Singh	-	LM/H-5/2021
62.	Dr. Harbhupinder Singh	-	LM/H-6/2021 (Couple - LM/G-6/2021)

	GYNAE OBS.		
63.	Dr. Manjit Kaur Mohi	-	LM/M-4/2017
64.	Dr. Manpreet Kaur	-	LM/M-5/2017
65.	Dr. Preet Kanwal Sibia	-	LM/P-1/2016 (Couple R-3)
66.	Dr. Sarabjit Kaur	-	LM/S-2/2016
67.	Dr. Sangeeta Aggarwal	-	LM/S-6/2017 (Couple-H2)
68.	Dr. Parneet Kaur	-	LM/P-6/2020
69.	Dr. Beant Singh	-	LM/B-6/2021
70.	Dr. Satinder Pal Kaur	-	LM/S-10/2021
71.	Dr. Navneet Kaur	-	LM/N-5/2021
72.	Dr. Aarti Narula	-	LM/A-10/2021
73.	Dr. Balwinder Kaur	-	LM/B-7/2021
74.	Dr. Anju	-	LM/A-11/2021
	PLASTIC SURGERY		
75.	Dr. Kuldeep Garg	-	LM/K-3/2017 (Couple P-3)
	TRANSFUSION MEDICINE		
76.	Dr. Kanchan Bhardwaj	-	LM/K-1/2016
77.	Dr. Rajni Bassi	-	LM/R-1/2016
78.	Dr. Shelly Jetly	-	LM/S-7/2017
	RADIO-DIAGNOSIS		
79.	Dr. Amarjit Kaur Sidhu	-	LM/A-2/2016 (Couple B-2): Radiology
80.	Dr. Manoj Mathur	-	LM/M-3/2017
81.	Dr. Saryu Gupta	-	LM/S-1/ 2016
82.	Dr. Rajesh Kumar Badhan	-	LM/R-5/2020 (Couple S-9)
	RADIATION ONCOLOGY		
83.	Dr. Vinod Dangwal	-	LM/V-2/2016
84.	Dr. Anshuma Bansal	-	LM/A-9/2021 – Radiation Oncology
85.	Dr. Neeru Bedi	-	LM/N-3-2021 – Radiation Oncology
86.	Dr. Raja Paramjeet Singh Benipal	-	LM/R-7/2021
	ANESTHESIA		
87.	Dr. Balwinder Kaur Rekhi	-	LM/B-3/2016 (Couple H-1)
88.	Dr. Mandeep Kaur	-	LM/M-8/2021
89.	Dr. Parmod Kumar	-	LM/P-8/2021
90.	Dr. Lalit Kumar	-	LM/L-1/2021
91.	Dr. Gurjit Singh Gandhi	-	LM/G-5/2021
92.	Dr. Gurlivleen Kaur	-	LM/G-6/2021
93.	Dr. Tripat Kaur	-	LM/T-01/2021
94.	Dr. Davinder Chawla	-	LM/D-6/2021
95.	Dr. Tanveer Singh Kundra	-	LM/T-02/2022

Life Membership No. - Wise

1.	LM/A-1/2016	Dr. Anjleen Kaur - Pharmacology
2.	LM/A-2/2016	Dr. Amarjit Kaur Sidhu - Radiology (Couple B-2)
3.	LM/A-3/2017	Dr. Avnish Kumar - Physiology
4.	LM/A-4/2017	Dr. Amandeep Singh Bakshi - Orthopedics
5.	LM/A-5/2017	Dr. Ardaman Singh - Medicine
6.	LM/A-6/2017	Dr. Ashok Kumar - Microbiology
7.	LM/A-7/2020	Dr. Anupinder Thind - Physiology
8.	LM/A-8/2021	Dr. Anjana Garg - Surgery
9.	LM/A-9/2021	Dr. Anshuma Bansal - Radiation Oncology
10.	LM/A-10/2021	Dr. Aarti Narula - Gynae & Obs.
11.	LM/A-11/2021	Dr. Anju - Gynae & Obs.
12.	LM/A-12/2021	Dr. Anand Songla - Surgery
13.	LM/B-1/2016	Dr. B L Bhardwaj - Medicine, Principal GMC Patiala (Couple K-1)
14.	LM/B-2/2016	Dr. BS Sidhu - Psychiatry
15.	LM/B-3/2016	Dr. Balwinder Kaur Rekhi - Anesthesia (Couple H-1)
16.	LM/B-4/2017	Dr. Baljinder Kaur - Pediatrics
17.	LM/B-5/2017	Dr. Bhupinder Singh Brar - Orthopedics
18.	LM/B-6/2021	Dr. Beant Singh - Gynae & Obs.
19.	LM/B-7/2021	Dr. Balwinder Kaur - Gynae & Obs.
20.	LM/C-1/2020	Dr. Chiman Lal - Eye
21.	LM/D-1/2016	Dr. D S Bhullar - Forensic Medicine & Toxicology
22.	LM/D-2/2016	Dr. Dimple Chopra - Skin & VD (Couple V-1)
23.	LM/D-3/2017	Dr. Dimple Sahni - ENT (Couple G-1)
24.	LM/D-4/2017	Dr. Darshanjit Singh Walia - Surgery (Couple M-7)
25.	LM/D-5/2021	Dr. Dinesh Kumar Passi - Surgery (Couple M-9)
26.	LM/D-6/2021	Dr. Davinder Chawla - Anesthesia
27.	LM/D-7/2021	Dr. Deeksha Singla - Paed
28.	LM/G-1/2017	Dr. Garish Sahni - Ortho
29.	LM/G-2/2017	Dr. Gursatinder Singh - Eye
30.	LM/G-3/2017	Dr. Gursharan Singh - Pediatrics
31.	LM/G-4/2020	Dr. Gagneen Kaur Sandhu - Physiology
32.	LM/G-5/2021	Dr. Gurjit Singh Gandhi - Anesthesia
33.	LM/G-6/2021	Dr. Gurlivleen Kaur - Anesthesia
34.	LM/H-I /2016	Dr. H S Rekhi - Surgery
35.	LM/H-2/2016	Dr. Hari Om Aggarwal - Orthopedics
36.	LM/H-3/2017	Dr. Harsimarjit Kaur - Anatomy
37.	LM/H-4/2019	Dr. H K S Chawla - Orthopedics
38.	LM/H-5/2021	Dr. Harjinder Singh - Urology
39.	LM/H-6/2021	Dr. Harbhupinder Singh - Urology (LM/G-6/2021)
40.	LM/J-1/2021	Dr. Jaswinder Singh - Surgery
41.	LM/K-1/2016	Dr. Kanchan Bhardwaj - Transfusion Medicine
42.	LM/K-2/2016	Dr. K K Aggarwal - Forensic Medicine
43.	LM/K-3/2017	Dr. Kuldeep Garg - Plastic Surgery (Couple P-3)
44.	LM/K-4/2017	Dr. Kuldeep Singh Bhatia - Surgery
45.	LM/K-5/2021	Dr. Kuldeep Singh Sandhu - Ortho
46.	LM/L-1/2021	Dr. Lalit Kumar - Anesthesia (Couple- A-8)
47.	LM/M-1/2016	Dr. Maninder Kaur - Biochemistry

48.	LM/M-2/2016	Dr. Mohanvir Kaur - Hematology
49.	LM/M-3/2017	Dr. Manoj Mathur - Radiology
50.	LM/M-4/2017	Dr. Manjit Kaur Mohi - Gynae
51.	LM/M-5/2017	Dr. Manpreet Kaur - Gynae
52.	LM/M-6/2017	Dr. Manjit Singh - ENT
53.	LM/M-7/2020	Dr. Manpreet Kaur Walia - Eye
54.	LM/M-8/2021	Dr. Mandeep Kaur - Anaesthesia
55.	LM/M-9/2021	Dr. Manju Bala - Anatomy
56.	LM/N-1/2017	Dr. Navneet Kaur - Pathology
57.	LM/N-2/2020	Dr. Neeraj Mittal - Psychiatry
58.	LM/N-3/2021	Dr. Neeru Bedi - Radiation Oncology (Couple - LM/P-9/2021)
59.	LM/N-4/2021	Dr. Neetu Sharma - Pharmacology
60.	LM/N-5/2021	Dr. Navneet Kaur - Gynae & Obs.
61.	LM/P-1/2016	Dr. Preet Kanwal Sibia - Gynae. & Obs. (Couple R-3)
62.	LM/P-2/2016	Dr. Puneet Gambhir - Community Medicine (Couple S-1)
63.	LM/P-3/2017	Dr. Parveen Mittal - Pediatrics
64.	LM/P-4/2017	Dr. Paras Pandove - Surgery
65.	LM/P-5/2017	Dr. Prem Chand Singla - Surgery
66.	LM/P-6/2020	Dr. Parneet Kaur - Gynecology
67.	LM/P-7/2020	Dr. Preetinder Singh Chahal - Forensic Medicine
68.	LM/P-8/2021	Dr. Parmod Kumar - Anesthesia
69.	LM/P-9/2021	Dr. Parvinder Singh - ENT
70.	LM/R-1/2016	Dr. Rajni Bassi - Transfusion Medicine
71.	LM/R-2 /2016	Dr. Rupinder Kaur Bakshi - Microbiology
72.	LM/R-3/2016	Dr. R P S Sibia - Medicine
73.	LM/R-4/2016	Dr. Rajan Singla - Anatomy
74.	LM/R-5/2020	Dr. Rajesh Kumar Badhan - Radiology (Couple S-9)
75.	LM/R-6/2021	Dr. Ranjana - Pharmacology
76.	LM/R-7/2021	Dr. Raja Paramjeet Singh Benipal - Radiation Oncology
77.	LM/R-8/2022	Dr. Ravisha Bhardwaj - Orthopedics
78.	LM/S-1/ 2016	Dr. Saryu Gupta - Radio diagnosis
79.	LM/S-2/2016	Dr. Sarabjit Kaur - Gynecology
80.	LM /S-3/2017	Dr. Seema Goyal - Skin
81.	LM /S-4/2017	Dr. Sanjay Goyal - Medicine
82.	LM/S-5/2017	Dr. Sanjeev Bhagat - ENT
83.	LM/S-6/2017	Dr. Sangeeta Aggarwal - Gynae (Couple-H2)
84.	LM/S-7/2017	Dr. Shelly Jetly - Transfusion Medicine
85.	LM/S-8/2020	Dr. Satinder Pal Singh - Forensic Medicine
86.	LM/S-9/2020	Dr. Sudesh Kumari - Chest & TB
87.	LM/S-10/2021	Dr. Satinder Pal Kaur - Obs. & Gynae.
88.	LM/S-11/2021	Dr. Sanjeev Gupta - Surgery
89.	LM/T-01/2021	Dr. Tripat Kaur - Anesthesia
90.	LM/T-02/2022	Dr. Tanveer Singh Kundra - Anesthesia
91.	LM/ V-1/2016	Dr. Vishal Chopra - Chest & TB
92.	LM/V-2/2016	Dr. Vinod Dangwal - Radiotherapy
93.	LM/V-3/2016	Dr. Vijay Bodal - Pathology (Couple S-2)
94.	LM/V-4/2016	Dr. Vijay Sehgal - Pharmacology
95.	LM/V-5/2017	Dr. Vandna Singla - Clinical Pathology

Format of Application for Membership

To
The Editor
Journal Club Government Medical College Patiala Punjab India

Dear Sir/Madam

I wish to become a Life Member/Annual Member of the Journal Club GMC Patiala. I am furnishing the required particulars below with a request to enroll me in the Journal Club.

The fee of Rs. 5000/ Rs. 8000/- Rs 1000/- for Life Membership (Single/Couple)/ Annual Membership is enclosed as a Demand Draft/ Cheque with No._____of _____Bank, in the name of Journal Club Government Medical College Patiala along with my two passport size photographs.

I have gone/will go through the rules and regulations of the Journal Club and I agree to abide by the same.

PARTICULARS

1. Full name (in block letters)
2. Father's/Husbands' name
3. Qualification
4. Official Designation & Place of Posting
5. Permanent Address
6. Phone No. & Email

Place Yours Sincerely

Date (Signature)

For Use of Journal Club GMC Patiala

Membership accepted with life/Annual Membership No.....

Secretary Finance

Editor-in-Chief

Founder Editorial Board

(Year 2017-2018)

GMC Patiala Journal of Research and Medical Education

(An Official Publication of Journal Club, Government Medical College, Patiala Punjab India)

Founder Team: Editorial Board

N,B. This is the permanent page for the Founder Team (Editorial Board)