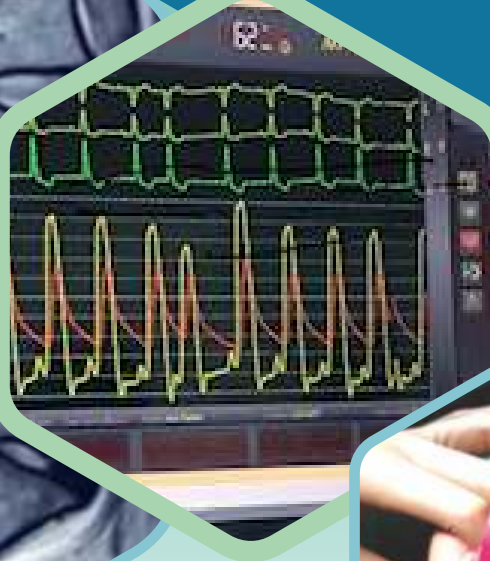


# LILAVATI HOSPITAL MEDICAL TIMES

JANUARY 2022



Lilavati Hospital and Research Centre

*More than Healthcare, Human Care*

NABH Accredited Healthcare Provider

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## From COO's Desk



At the outset I take this opportunity to wish you all a happy and prosperous 2022.

Most of 2020 & 2021 were spent in fear of invisible enemies, may this New Year bring freedom from fear and anxiety. This past year was a year like no other, and will be remembered for the devastating second wave of Covid and now as we are passing through the third wave due to the new variant Omicron we continue to be under the spell of Covid infection. But Lilavati Hospital has risen to the occasion and expanded its Covid bed strength during the second wave much beyond what BMC demanded for. I am grateful to all the staff of LHRC who served the society during the trying times. As a bastion of advanced medical care, we are determined to further contribute to community-based medical care.

The year also brought some good news as well. I am grateful to the Board of Trustees & Mr Lakshminarayanan, IAS (Retd.) – Principal Advisor to the Board of Trustees & LHRC for their generous act of appraising the salary and granting arrears to non-unionised staff and also for successfully completing the charter of demands of unionised category. This will go a long way in developing the confidence of all the employees, and create a harmonious working environment.

This institute has ensured that all our staff are continually updated and appropriately trained in order to provide an excellent holistic clinical service. With these objectives training sessions were conducted for staff which included Effective Complaint Management, interactive sessions on “Stress at Workplace” and others.

I am also happy to announce that Lilavati Hospital was a National level winner “Best Hospital Obstetrics & Gynecology” presented by The Economic Times Healthcare Awards 2021. We’re proud that we continue to rank among the top hospitals in this country.

This year has shown us that when we work together, we can overcome tremendous challenges, achieve great heights, support our community and achieve personal and professional happiness.

I am also aware that our goals would not have been achieved without due contribution from all of you.

I therefore take this opportunity to convey the management’s gratitude and acknowledge the sincere efforts for the contribution by one and all and also believe that all of us shall continue to strive hard in adding more glories and achievements in future as well

**Lt. Gen. (Dr.) V. Ravishankar**

Chief Operating Officer and Consultant Cardiothoracic Surgeon

## Editorial



I thank our CEO Lt. Gen. Dr. Ravishankar and the management of Lilavati Hospital for entrusting me with the responsibility of editing the Lilavati Hospital Medical Times from the year 2022. I sincerely appreciate the exemplary work of our previous editor, Dr Abhay Bhawe, and our Editorial and Marketing teams of Lilavati Hospital. The previous editions are an assurance of the high quality of the clinical and research work that is carried out by the hospital over the past quarter of a century and more.

Lilavati Hospital boasts of an international quality of Consultants doing paramount, worldwide recognised work alongside with their competent teams. I would like to request their active participation in publishing their pioneering work in the future editions of the Lilavati Hospital Medical Times.

Our hospital is a private teaching hospital, offering not just nationally recognised teaching courses(DNB) but also various Fellowships under the guidance of experienced departmental consultants. We would like to encourage the active participation of students and the resident doctors to publish their research work in the future editions. We plan on incentivising the submissions by awarding The Best Case Report and The Best Paper Prizes.

In the subsequent editions, we would also like to publish a new perspective from every speciality branch.

The last two years have been nothing short of challenging due to the Covid pandemic, but it has been handled in an accomplished manner at our Lilavati Hospital. This has been appropriately appreciated by our patients, various Government authorities and the media.

I am confident of a wholehearted cooperation from my editorial team, my colleagues and the marketing and management teams at Lilavati during my tenure, to maximally optimise the academic content of our publication. I look forward to an active feedback and suggestions from our readers which will help us refine the quality of the publications to empower you with more knowledge.

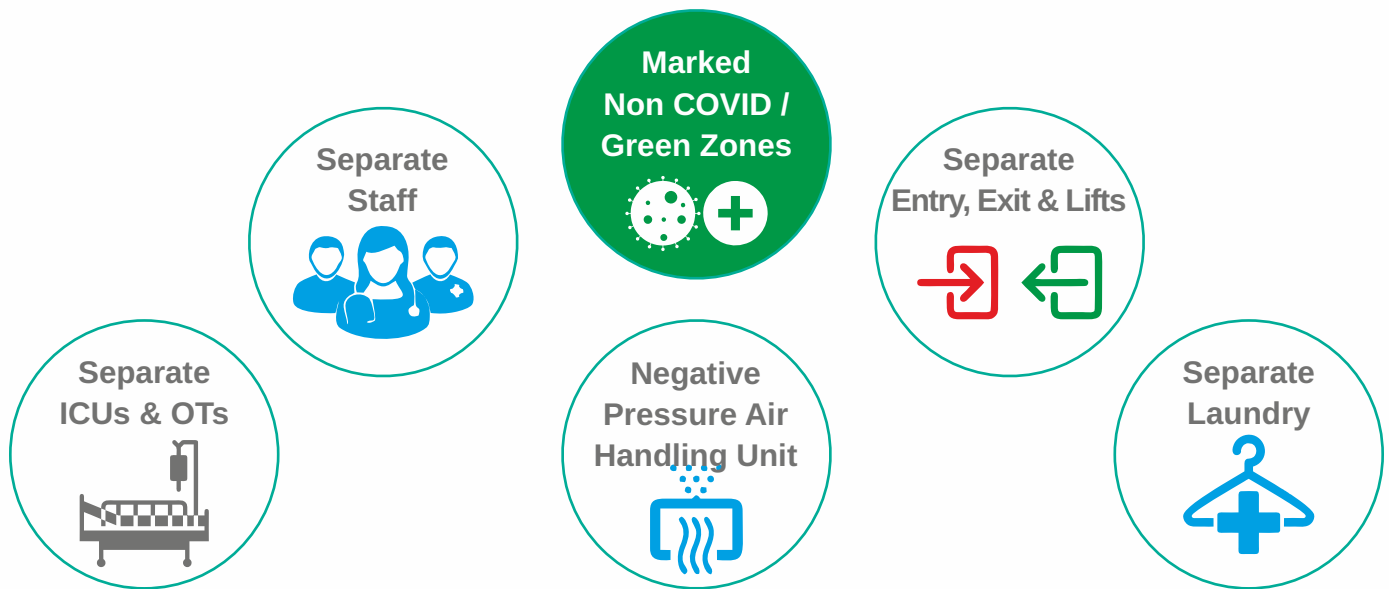
Wishing you all a very happy and healthy new year ahead!

**Dr. Rajeev Redkar**

M.Ch., FRCS (Paed. Surg), MS, DNB, FRCS (Edin), FRCS (Glas), FCPS, IAS  
Chairman, Indian Association of Pediatric Surgeons (Maharashtra Chapter)



# OUR **NEW NORMAL** IS AS SAFE AS EVER



**PANDEMIC IS TEMPORARY**

**OUR DEDICATION TO PATIENT CARE AND SAFETY IS PERMANENT**

## Overview: Lilavati Hospital & Research Centre



Late Shri Kirtilal Mehta



Late Smt. Lilavati K. Mehta

### Lilavati Kirtilal Mehta Medical Trust

Lilavati Hospital and Research Centre is run and managed by Public Charitable Trust - Lilavati Kirtilal Mehta Medical Trust which was formed in 1978. The Trust was started by late Shri Kirtilal Manilal Mehta. The Trust has engaged in innumerable charitable endeavors across India.

#### The Lilavati Kirtilal Mehta Medical Trust is being managed and administered by Board of Trustees:

Smt. Sushila V. Mehta	Shri Nanik Rupani
Shri Kishor K. Mehta	Shri Rashmi K. Mehta
Smt. Charu K. Mehta	Shri Dilip Shanghvi
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Shri Ayushman C. Mehta

Principal Advisor to the Board of Trustees and  
Lilavati Hospital & Research Centre  
**Shri S. Lakshminarayanan, IAS (Rtd.)**

### Lilavati Hospital And Research Centre

Late Shri Vijay Mehta wished to fulfill his parents desire to build a world-class hospital where everyone in need for relief from disease and suffering come in with a certainty to receive the best possible medical care. His passion, attention to details and perseverance resulted in iconic healthcare landmark called **Lilavati Hospital**.

Lilavati Hospital & Research Centre is a premier multispecialty tertiary care hospital located in the heart of Mumbai, close to the domestic and the international airport. It encompasses modern healthcare facilities and state of art technology dedicatedly supported by committed staff.

Lilavati Hospital has focused its operation on providing quality care with a human touch; which truly reflects the essence of its motto, “More than Healthcare, Human Care”. Being a centre of medical excellence where technology meets international norms and standard, the hospital has got what it takes to be a pioneering quality healthcare institute that is also one of the most sought after and patient friendly hospital.

**Mission:** *To provide affordable healthcare of international standard with human care*

**Motto:** *More than Healthcare, Human Care*

## Highlights

- 323 bedded hospital including 77 intensive care beds. Currently number of beds have been temporarily increased for helping fight the COVID pandemic. We have dedicated 144 ward beds and 48 ICU beds to treat COVID positive patients.
- 12 state-of-the-art well equipped operation theatres.
- Full-fledged Liver Transplant, Heart Failure, Hypertension, Bariatric, Foot and Ankle, Dental and Dermo Cosmetology Clinic.
- State of art PET – SPECT CT department.
- Lilavati Hospital is equipped with Coronary GRAFT Patency Flowmeter which is first of its kind in India. This imaging system is used in Cardiac surgery to assess GRAFT flow / perfusion in coronary bypass surgery.
- The hospital has installed state-of-art Philips Azurion 7F20 in its cath lab. This is the first of its kind high end configuration system installed in India. The new system enables excellent imaging for Coronary, Cerebro & Peripheral Vascular Diseases.
- The department of Invasive Cardiology has been upgraded with the addition of a High Definition Optis Mobile OCT (Optical Coherence Tomography) system. It has the latest configuration which gives better 3 – Dimensional perspective of Coronary Artery before and after stent deployment.
- The hospital has added Intraoperative Nerve Monitoring system which enables surgeons to identify, confirm and monitor motor nerve function of the patients which helps to reduce the risk of nerve damage during various operative surgeries.
- The hospital has upgraded its ENT department by adding a top-of-the line surgical operating microscope to carry out various microsurgies under high magnification. The microscope electronics allows the surgeon to electronically control object focusing, magnification, illumination, surgical recording, etc.
- All days round the clock OPD Pathology and Radiology investigations without any Emergency charges.
- More than 300 consultants and manpower of nearly 1,800.
- Hospital attends to more than 10000 In-patient, 40000 Out-patient and performs thousands of surgeries every year.
- Modern Cathlabs having specialized SICU & ICCU with highly trained cardiac care medical staff.

## Lilavati Kirtilal Mehta Medical Trust Research Centre

The Lilavati Kirtilal Mehta Medical Trust Research Centre is a Scientific and Industrial Research Organization approved by Ministry of Science and Technology (Govt. of India). The Research Centre under guidelines of Dept. of Science & Technology works in close collaboration in evaluating and developing technologies for better healthcare to the sick people. The research centre has undertaken multidisciplinary researches in the fields of Cardiology, Radiology, Cerebrovascular Diseases (Stroke), Ophthalmology, Chest Medicine, Nuclear Medicine, Pathology, Oncology, Orthopedics etc., to cite a few. One of the important aim of the research centre is to establish community based epidemiological researches in cerebrovascular disease in stroke. As a policy, Drug and Device Trials are not undertaken at the Research Centre.

# CASE REPORT I: ANAESTHESIOLOGY

## A Clinical Audit of Quality Indicators In Anaesthesia Practice Over Last 5 Years In Lilavati Hospital And Research Centre

**Dr Vaibhavi Baxi, DA, FCPS, DNB, Consultant Anaesthetist**

**Dr Nisha Gandhi, DA, MD, DNB, Consultant Anaesthetist**

**Dr Dixsha T P, DNB Resident**

**Dr Abdul Vahid, DNB Resident**

**Dr Annette Sebastian, DNB Resident**

### INTRODUCTION:

A large number of patients undergo anaesthesia for different kind of surgeries. With the availability of more and more modern and safe anaesthetics and healthcare being focussed on patients complete well being with a holistic approach; it is imperative to effectively monitor the quality of the services provided. Mortality is no longer seen as a good quality indicator of anaesthesia services. Diligent monitoring of the quality of anaesthesia services is required to maintain and improve standards of patient care and safety.

Monitoring involves collecting data upon important quality indicators, processing the data to provide effective feedback and use it to support quality improvement. So this study was designed to assess a few anaesthesia quality indicators and use them to audit the practice for quality of service.

### MATERIALS AND METHOD:

This audit was conducted at Lilavati Hospital and Research Centre, a tertiary care, 300 bedded hospital located in Bandra, Mumbai, India. We did a retrospective study, where we assessed all the patients undergoing different surgeries in our surgical theatre complex of 12 operation theatres, over a period of 5 years. Clearance for this study was duly obtained from institutional research review committee and ethics committee. Data was collected from Jan 2016 to Dec 2020. The quality indicators chosen to be analyzed from the records were as follows:

1. Modification of anaesthesia plan
2. Unplanned ventilation
3. Adverse anaesthesia event
4. Anaesthesia related mortality

### DATA COLLECTION:

As per the department of anaesthesia protocol any unexpected or untoward event during anaesthesia is recorded in the database of quality indicators under the above mentioned categories. We collected the data from this database for last five years. (Yr2015- Yr2020).

The data consisting of number of cases where the following events occurred over each year were collected and tabulated:

**Modification of anaesthesia plan:** cases where the original plan of anaesthesia was modified or changed intra-operatively. For e.g. original plan of anaesthesia being subarachnoid block but plan changed over to general anaesthesia due to either patchy effect of block or extended duration of surgery.

**Unplanned Ventilation:** elective cases where the patient developed some complication or adverse event intra or post-operatively ( e.g. seizures or poor breathing attempts) and had to be re-intubated and mechanically ventilated post-operatively in ICU.

**Adverse anaesthesia events:** cases where there were some adverse events like drug reaction, bronchospasm, tooth dislodgement during intubation, pneumothorax etc.

**Anaesthesia related mortality:** Cases with mortality during anaesthesia were to be noted; but fortunately we had no cases of anaesthesia related mortality over last five years.

### STATISTICAL ANALYSIS:

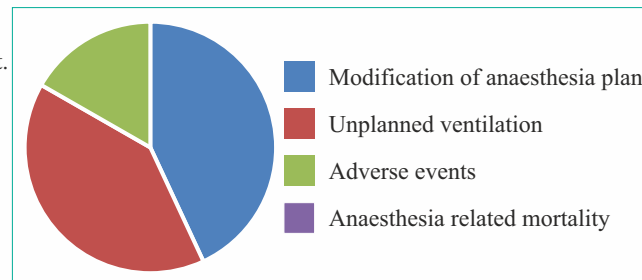
This study involves a review of patient data. Microsoft word and excel have been used to generate tables, graph. Statistical difference between each quality indicators were listed using mean and median.



## RESULT:

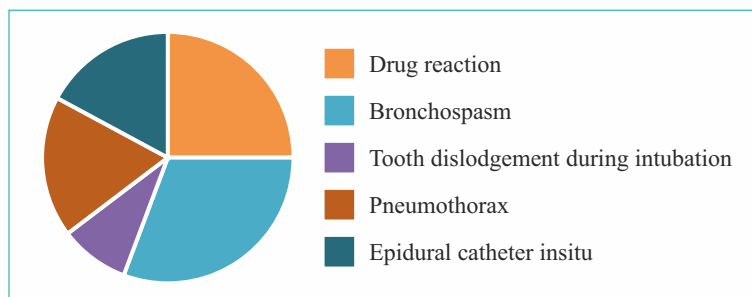
A total of 33901 patients were audited and included in the final data sheet.

Percentage of modification of anaesthesia plan	26/33901 0.076%
Percentage of unplanned ventilation	24/33901 0.070%
Percentage of adverse events	12/33901 0.035%
Percentage of anaesthesia related mortality	0/33901 0.00%



## Percentage of Adverse events:

Drug reaction	0.0088%
Bronchospasm	0.01179%
Tooth dislodgement during intubation	0.0029%
Pneumothorax	0.0058%
Epidural tip insitu	0.0058%



## DISCUSSION

Clinical audit can be defined as a quality improvement process that seeks to improve patient care and outcomes through systematic review of care against explicit criteria and implementation of change. Research on the development of effective quality indicators for clinical practice suggests that they should be transparent, reliable, evidence based, measurable and improvable. Anaesthesia indicators have been renamed as peri-operative indicators by the experts. Nowadays clinical indicators are increasingly being developed and promoted by professional organisations and governmental agencies as a measure of quality of service and performance and these initiatives have boosted the development of indicator programmes.<sup>1</sup>

Out of total 33901 patients, there was modification of anaesthesia plan for 26 patients, which accounted for 0.0766%. The most common reasons being patchy effect central neuraxial blockade, extended duration of surgery and unexpected surgical interference (due to repeated vomiting episodes).

Out of total 33901 patients there was unplanned ventilation for 24 patients which accounted for 0.0707% of cases. The most common reasons being prolonged duration of surgery, poor respiratory efforts, inspiratory stridor, surgical complications (for example – TURP (trans urethral resection of prostate) syndrome, Increased blood loss).

Out of total 33901 patients, there were adverse anaesthesia events in 12 patients which accounted for 0.0353% of the cases. The common reasons being drug reaction (0.0088%), bronchospasms (0.011%), tooth dislodgement during surgery (0.0029%), pneumothorax (0.0058%), epidural catheter tip in situ (0.0058%) respectively.

In Maya Nadkarni et al<sup>2</sup>, more number of quality indicators are included, panning from preoperative assessment till post operative care. According to Walzack RM<sup>3</sup>, the adequacy of anaesthesia care documentation can also be measured during surgery and in postoperative recovery room. In our audit, we have focussed on 4 quality indicators involving intra-operative to post-operative care.

Most indicators measured several dimension of anaesthesia care and safety was the dimension that was most commonly addressed.

Number of cases included is more in our study compared to Maya Nadkarni et al. In Maya Nadkarni et al, there was modification of anaesthesia plan 13.72% (569/4147), unplanned ventilation 9.11% (375/4147), adverse anaesthesia events 25.9% (1077/4147). No anaesthesia related mortalities were there in either of the studies.

In H K Mahajan et al<sup>4</sup> they have compared quality indicators in anaesthesia same as ours but of year 2013 (total no. of patients= 3717) and 2014 (total no. of patients=3931). In their study overall incidence of modification of anaesthesia plan is less than 1% and is comparable to any other peer institution nationally and internationally. Incidence of unplanned ventilation during 2013 than 2014 was approximately 0.9% (average of 2 out of 220 cases per month). Incidence of anaesthesia related events was 0.43% in 2013 and 0.63% in 2014. There is no anaesthesia related mortality in their study.

In Garry D et al<sup>5</sup>, they have included 3817 patients over a year in their study. There is only 0.13% anaesthesia adverse events, 0.41% unplanned ventilation. There was no UK national standards to compare the study, hence they have reported their data to be in as acceptable range compared to other national studies.

In our study, modification of anaesthesia plan 0.076% (26/33901), unplanned ventilation 0.0707% (24/33901), adverse anaesthesia events 0.03% (12/33901). We had no anaesthesia related mortality.

Compared to other studies, numbers are significantly less and the reason could be involvement of multiple specialities in management of

patients both pre and post operatively, intensive care backup and availability of modern gadgets which helps us provide better services. Also being a private tertiary care centre, there is a dedicated senior anaesthetist along with experienced qualified team consisting anaesthesia technician and assistant anaesthesia doctors for each patient. However the other reason for low numbers of each indicator could also be due to poor data reporting.

This audit helped us realize that there is scope for improvement in our services towards the patients. We also look forward to increasing awareness about quality indicators among our team members and motivate them to improve the reporting of data in future. We also would like to increase the number of quality indicators in our next audit to cover further aspects of patient care.

#### **CONCLUSION:**

The concepts of quality assurance and quality control are rapidly gaining popularity in anaesthesia practice as the society is heading towards clinical advancement globally. Quality of anaesthesia services monitored by quality indicators are a major determinant of overall peri-operative outcome and patient safety. Audit of our services motivates us to incorporate suitable remedial measures to improve the quality of anaesthesia care towards our patients.

#### **REFERENCES:**

1. Haller G, Stoelwinder J, Myles PS, McNeil J. Quality and safety indicators in anaesthesia :a systematic review. *Anaesthesiology* 2009 May;110(5):1158-1175.
2. Nadakarni M, Desi P, Sahajananda H. A Clinical Audit of Quality Indicators in Anaesthesia Practice. *J Med Sci* 2015;1(3):47-51.
3. Walczak RM. JCAH perspective: Quality assurance in anaesthesia services. *AANA J* 1982 Oct;50(5):462-464.
4. Mahajan H K, Dhanerwa R, Chauhan P R, Gupta A. A Comparative Study of Key Quality Performance Indicators in Anaesthesia and Surgery. *JMSCR* 2017 Feb;05(02):17269-17277.
5. Garry D, Milne L, Ekpa J, Goose A, Lahkar A, Hariharan V. Quality indicators in anaesthesia – An audit of local practice: *Online J Clin Audits* 2013;5(4).

## CASE REPORT II: ANAESTHESIOLOGY

### Moving towards Regional Anaesthesia for Spine Surgery - Need of the Hour

**Dr. Samidha Waradkar Thakur, DA, DNB Anaesthesiology, PGDMLS,**  
**Consultant Anaesthesiology**  
**Dr Aaliya Mehmood, DA, DNB Anaesthesiology**

The year 2020-2021 saw a global upsurge of covid-19 cases. India being the epicentre of the pandemic faced this crisis head-on.

In the words of John F Kennedy: when written in Chinese the word Crisis (危机) is composed of two characters, 危 represents - danger and the other 机 represents - Opportunity. A similar opportunity was provided in the last one and half year, where maximum lower thoracic to micro-lumbosacral spine surgeries were performed under spinal anaesthesia, with lumbar discectomy/ decompression being one of the most performed procedures.

In today's era of minimally invasive to non-invasive fast track surgeries it has become the need of the hour to provide fast track and well-balanced anaesthesia techniques to accelerate recovery and facilitate faster postoperative discharges. Furthermore, with the ongoing Covid -19 pandemic, providing regional anaesthesia wherever feasible has led to decreased airway handling and the subsequent aerosol generation related complications.

These days with the refinement in the surgical technique, lumbar discectomy and decompression have become minimally invasive. They have a mean surgical duration of approximately 1 hour; with single level fusion requiring 2 hours and double level fusions requiring maximum of 3 hours. This has made spinal anaesthesia an attractive choice for these patients especially in the Covid scenario.

Lumbar spine surgery can be successfully performed under various anaesthesia techniques. Although, neuraxial block using spinal anaesthesia (subarachnoid block) for spine surgery has been established as an accepted technique for many years; General anaesthesia is being widely practised due to a variety of factors, including greater patient acceptance, enabling of long duration surgeries, and capacity for secure airway establishment in the prone position [1,2,3].

Surgical procedures on the lumbar spine for which Spinal Anaesthesia can be administered include

- Discectomy
- Foraminotomy
- Synovial cyst removal
- Decompression
- several types of fusions (single/double level)
- lower thoracic/lumbosacral biopsy

Patient preferred for spinal anaesthesia

- ASA I-II
- Age 18 years and above
- Compliant and cooperative
- Hemodynamically stable
- Short duration of surgical procedure
- Estimated lesser fluid shifts and blood loss

Patients excluded for spinal anaesthesia (GA Preferred) <sup>[4]</sup>

- Patient refusal
- Patients with history of seizure, intracranial hypertension
- allergic to local anaesthetics
- CNS disorders
- Coagulopathy
- infection at site of needling
- hypovolemic or hemodynamically unstable
- severe spinal stenosis
- a near complete or total myelography block
- myelography demonstration of arachnoiditis
- drug or alcohol abuse
- morbidly obese
- not optimised multiple comorbidities
- Patients having any changes in surgical technique
- anticipated massive bleeding during operation which needed blood transfusion
- procedure expected to extend longer than the neuraxial block
- fixed cardiac output states (ischemic heart disease patients with low EF, severe AS/MS)
- indeterminate neurological diseases like GBS, multiple sclerosis (relative contraindication)

Eligible patients should receive written informed consent after detailed explanation of the surgical procedure and anaesthesia technique. All patients are kept NBM (Nil by Mouth) for 6-8 hours prior to the surgical procedure and premedicated with Cap Pan D 2 hours prior in the wards.

Once in OR after attachment of ASA monitors and establishment of a venous access, patients are placed into a sitting position and under all aseptic precautions, Spinal anaesthesia is performed using a 25-gauge Whitacre spinal needle at 1-2 interspace above or below the surgical level, after local infiltration with 2-3 ml of 2% Lidocaine. The Subarachnoid block can be done with 3.0 – 3.6 ml 0.5% (heavy/hyperbaric) Bupivacaine in an 8.5% Dextrose solution combined with 25 µg Fentanyl/15-30 µg Clonidine after preloading/co-loading patients with balanced salt (isotonic) solution over 10-15 minutes. After vigilant administration of drug into the intrathecal space, the patients are placed in supine position. Five to ten minutes after establishment of spinal level of block (which usually occurs between T-6 and T-10), the patients can then gradually be placed into prone position.

The heart rate, systolic, diastolic, mean arterial blood pressure and oxygen saturation are monitored every 5-10 minutes using ECG, non-invasive blood pressure monitoring and pulse oximetry. Oxygen at 2-6L/min via nasal cannula can be administered throughout intraoperative journey.

Intraoperatively, if the patients experience bradycardia (heart rate less than 40 per minutes) or hypotension (systolic blood pressure less than 80 mmHg OR MAP < 50mm Hg), 0.6 mg Atropine/0.2mg Glycopyrolate or 5 mg Ephedrine can be administered. If needed patients can be sedated with 1-2mg Midazolam or IV dexmed 0.2-0.7 ug/kg/hr infusion. At the end of surgery, the infusion should be discontinued, the patients are turned from the prone to supine position and transferred to the PACU/Recovery room. When patients have no pain, nausea, vomiting, and at least regression of spinal block below T10, they can be discharged from the PACU.



Fig. 1 and 2: A 87 years, female patient posted for L2 vertebroplasty with L3-L4 percutaneous screw fixation lying prone comfortably under spinal anaesthesia.



Fig. 3 and 4: AP and Lateral views post L2 vertebroplasty with L3-L4 percutaneous screw fixation.



SA, which is widely used in general orthopaedic and vascular surgery, has several benefits noted in the literature, including rapid onset, less intraoperative blood loss, thrombotic events, pulmonary complications, and postoperative cognitive dysfunction [5-7]. It also allows the patient to breathe spontaneously and reposition themselves to avoid compression injuries during the course of the procedure [8,9]. Various studies comparing GA and SA for lumbar surgery have shown reduced surgical time, postoperative pain, time in the post-anaesthesia care unit (PACU), incidence of urinary retention, postoperative nausea, and more favourable cost-effectiveness.

As per Scott et al [10], pulmonary complications were more common in patients who underwent GA compared with regional anaesthesia. In support, two retrospective studies conducted showed that SA resulted in better outcome compared with GA in patients who underwent surgeries on lumbar spine [11,12].

Similarly, more than 25 cases conducted by us at LHRC had better outcome with SA when compared to GA. None of the 25 patients needed conversion to GA or had an episode of high/complete sympathetic blockade. No incidences of Bagai (vasovagal) syncope, PONV, postop meningism, headache or CSF flow, intraoperative dural csf leak or postop fistula were noted. There were 2 incidences of failed spinal which were easily managed with a lower dose repeat spinal. Overall better postop analgesia and higher patient and surgeon satisfaction compared to GA was observed.

Despite encouraging results in favour of SA, SA does have few related risks as mentioned below, and there is (at least to date) no clear evidence to delineate the difference in morbidity and mortality between the two approaches [20].

Surgery on the lower thoraco-lumbar spine can be safely performed under general or regional anaesthesia. Patient's satisfaction and the ability to carry out prolonged operations in the prone position without airway compromise are advantages of using general anaesthesia (GA) [13,14]. On the other hand, along with the ease of administration, spinal anaesthesia has rapid onset and reversal of effects. It also helps maintain stable haemodynamics throughout the surgical duration without need to increase blood transfusion. It also avoids the polypharmacy and undue drug related complications associated with GA. Lastly, as it decreases recovery room stay with reduced postoperative pain, nausea, vomiting, and requirement for additional analgesics, Spinal anaesthesia again proves as an excellent choice.

In conclusion, it can be said that regional anaesthesia has proved as a powerful weapon for anaesthesiologists to tackle any adverse situation and provided an excellent opportunity to bring their best Endgame to face any crises recently faced in the form of covid-19 pandemic.



Fig. 5: MRI LS spine of a 37-year male, with large sequestration of PVID at L5-S1. Planned for Tubular Micro-endoscopic Discectomy under spinal anaesthesia.



Fig 6: Intraoperative Micro-endoscopic picture through the tubular portal for discectomy showing the nerve roots well decompressed

#### Complications of spinal anaesthesia [4]

##### Minor

- Nausea and vomiting
- Mild hypotension
- Shivering
- Itch
- Transient mild hearing impairment
- Urinary retention

##### Moderate

- Failed spinal
- Postdural puncture headache

##### Major

- Direct needle trauma
- Infection (abscess, meningitis)
- Hematoma
- Peripheral nerve injury
- Total spinal anaesthesia
- Cardiovascular collapse

#### Advantages of SA vs GA <sup>[15-19]</sup>

- controlled hypotensive anaesthesia
- improved operative conditions with bloodless surgical field
- decreased intraoperative blood loss with decreased postop blood transfusion
- prevention of polypharmacy and drug related complications
- decrease in perioperative cardiac ischemic incidents, postoperative hypoxic episodes, arterial and venous thrombosis
- better postoperative pain control
- as the patients can position themselves while they are awake leads to prevention of brachial plexus and other nerve injuries, pressure necrosis of face, glossitis and post-operation vision loss (POVL)

#### REFERENCES:

1. Demirel CB, Kalayci M, Ozkocak I, Altunkaya H, Ozer Y, Acikgoz B. A prospective randomized study comparing perioperative outcome variables after epidural or general anesthesia for lumbar disc surgery. *J Neurosurg Anesthesiol.* 2003;15:185–192.
2. De Rojas JO, Syre P, Welch WC. Regional anesthesia versus general anesthesia for surgery on the lumbar spine: a review of the modern literature. *Clin Neurol Neurosurg.* 2014;119:39–43.
3. Pflug AE, Halter JB. Effect of spinal anesthesia on adrenergic tone and the neuroendocrine responses to surgical stress in humans. *Anesthesiology.* 1981;55:120–126
4. <https://www.nysora.com/techniques/neuraxial-and-perineuraxial-techniques/spinal-anesthesia/>
5. McLain RF, Bell GR, Kalfas I, Tetzlaff JE, Yoon HJ. Complications associated with lumbar laminectomy: a comparison of spinal versus general anesthesia. *Spine (Phila Pa 1976).* 2004;29:2542–2547.
6. McLain RF, Tetzlaff JE, Bell GR, Uwe-Lewandrowski K, Yoon HJ, Rana M. Microdiscectomy: spinal anesthesia offers optimal results in general patient population. *J Surg Orthop Adv.* 2007;16:5–11.
7. Rodgers A, Walker N, Schug S, et al. Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: results from overview of randomised trials. *BMJ.* 2000;321:1493.
8. Attari MA, Mirhosseini SA, Honarmand A, Safavi MR. Spinal anesthesia versus general anesthesia for elective lumbar spine surgery: a randomized clinical trial. *J Res Med Sci.* 2011;16:524–529.
9. Brown MJ. Anesthesia for elective spine surgery in adults. 2015. Available from: <https://www.uptodate.com/contents/anesthesia-for-elective-spine-surgery-in-adults>. Accessed July 26, 2017.
10. Scott NB, Kehlet H. Regional anaesthesia and surgical morbidity. *Br J Surg.* 1988;75(4):299–304.
11. Ditzler JW, Dumke PR, Harrington JJ, Fox JD. Should spinal anesthesia be used in surgery for herniated intervertebral disk. *Anesth Analg.* 1959;38(2):118–24.
12. Hassi N, Badaoui R, Cagny-Bellet A, Sifeddine S, Ossart M. Spinal anesthesia for disk herniation and lumbar laminectomy. Apropos of 77 cases. *Cah Anesthesiol.* 1995;43(1):21–5. [In French]
13. Cucchiara RF, Michenfelder JD. *Clinical Neuroanesthesia.* London: Churchill Livingstone; 1990. Vertebral column and spinal cord surgery; pp. 325–50.
14. Abrishamkar S, Aminmansour B, Arti H. The effectiveness of computed tomography scans versus magnetic resonance imaging for decision making in patients with low back pain and radicular leg pain. *Journal of Research in Medical Sciences.* 2006;11(6):351–4.
15. Modig J, Karlstrom G. Intra- and post-operative blood loss and haemodynamics in total hip replacement when performed under lumbar epidural versus general anaesthesia. *Eur J Anaesthesiol.* 1987;4(5):345–55.
16. Rodgers A, Walker N, Schug S, McKee A, Kehlet H, van Zundert A, et al. Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: results from overview of randomised trials. *BMJ.* 2000;321(7275):1493.
17. Urwin SC, Parker MJ, Griffiths R. General versus regional anaesthesia for hip fracture surgery: a meta-analysis of randomized trials. *Br J Anaesth.* 2000;84(4):450–5.
18. Indelli PF, Grant SA, Nielsen K, Vail TP. Regional anesthesia in hip surgery. *Clin Orthop Relat Res.* 2005;441:250–5.
19. Sakura S. Epidural anesthesia and spinal anesthesia in the elderly. *Masui.* 2007;56(2):130–8. [In Japanese]
20. Kao FC, Tsai TT, Chen LH, et al. Symptomatic epidural hematoma after lumbar decompression surgery. *Eur Spine J.* 2015;24:348–357.



## CASE REPORT III: ANAESTHESIOLOGY

### **Pre-emptive use of high flow nasal cannula (HFNC) in post-COVID patient under monitored anaesthesia care with dexmedetomidine Infusion monitored by bispectral index (BIS) for successful transfemoral percutaneous transcatheter aortic valve implantation – a case report**

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#### **Abstract :**

The transcatheter aortic valve replacement(TAVI) is a well-established treatment option for severe symptomatic AS patients who are at predictive high risk for surgery.

MAC with Dextomedidine infusion was used to minimise drugs, avoid intubation and difficult weaning from the ventilator. The sedation strategy was Moderate/ conscious sedation. The bispectral index was used to monitor and maintain the depth of anaesthesia at 45-70. The patient was having post covid status, room air saturation of 92-94%, paO<sub>2</sub> of 60 mm of Hg, the breath-holding capacity of 4-6 seconds inability to do a 6-minute walk test due to cerebral decline, pre-emptively high flow nasal cannula set up at @ 15 litres/ min flow rate, 37°C, fio<sub>2</sub> of 60% was used to maintained oxygenation. The patient was hemodynamically stable without any incidence of apnea, hypercapnia or hypoventilation.

Well planned, pre-emptive use of HFNC, the sedation depth being monitored by BIS under MAC with dexmedetomidine infusion was key to successful management of post-COVID patient for TAVI during the pandemic.

**Keywords:** Anesthesia, Aortic Stenosis, Transcatheter Aortic Valve Implantation, Dextomedetomidine, Monitored Anaesthetic Care, COVID-19, High flow nasal cannula, Bispectral index

#### **Introduction:**

The transcatheter aortic valve replacement(TAVI) is a well-established procedure for the management of high-risk severe symptomatic AS patients(class I).[1]and for intermediate-risk(class 2a).[2]

The coronavirus pandemic substantially changed the management of severe AS with a shift into the less invasive option of TAVI. This resulted in short in-hospital stay without compromising the short term outcome. (50% Surgical Aortic Valve Replacement rate reduced to 34%).[3]

GA administration in such patients considered to have cardiac and pulmonary morbidity and poor renal function may require higher hemodynamic support and they may be difficult to wean off the ventilatory support post-procedure. Mindful of this complication, MAC with Dexmedetomidine infusion technique was chosen in our post covid status patient.[4,5]

Moderate sedation is a goal during MAC with sedation.[5,6]Moderate sedation / conscious sedation as defined by ASA is a purposeful response to verbal and tactile stimulation with adequate spontaneous ventilation maintained without airway intervention and usually with preserved cardiac function. Successful sedation is the adequate depth of anaesthesia as per patient and cardiac anesthesiologist requirement along with respiratory and hemodynamic stability.[5,7] Bispectral index was used to monitor the adequate depth of sedation..[8]

High flow nasal cannula provides a constant fixed concentration of hot and humidified oxygen with nasopharyngeal PEEP. HFNC 15 litres 60 % and 37 °C was chosen pre-emptively to avoid any desaturation during the procedure.[9,10]

This case report highlight pre-emptive use of HFNC in post covid patient using dexmedetomidine infusion monitored by BIS for successful TAVI procedure.

#### **Case Report**

A 77-year old male presenting with complaints of dyspnea on exertion NYHA grade 2, cough since 2 months and cognitive decline since 3-4 months( aggressive behaviour in morning and depression in evening) was admitted to our hospital. He was a known case of diabetes mellitus, hypertension and chronic kidney disease.

On admission COVID test was positive, HRCT showed CORAD -5 and multiple peripheral Ground Glass Opacities. The patient was treated as per COVID protocol.

TTE showed severe degenerative AS, concentric left ventricular hypertrophy, Ao Valve area of 0.48 cm<sup>2</sup>, peak and mean gradient across Ao valve of 111/67 mm of Hg and maximum trans aortic valvular velocity of 5.3 m/sec.

After 2 weeks the patient was discharged, quarantined for 14 days. The patient was then posted for TAVI.

On examination, the patient was conscious but confused and aggressive. He was afebrile with a pulse of 82/min and a Blood pressure of 107/60 mm of Hg. SpO<sub>2</sub> was 92% on room air, RR- 22-24/ min. Cardiovascular examination showed Pan systolic murmur in the aortic area. Respiratory system examination showed a normal chest with respiratory breath holding time of 4-6sec. Central nervous system examination - GC score was 15 and the power and reflexes were normal.

The blood investigations were as follows: The Complete blood count showed a Hb-13.90mg/dl, Wbc - 9300 , platelets-2,68,000. Serum creatinine- 1.69, CRP -6.64, blood group A positive, PT 13.10/11.70 INR -1.12, PTT:31.3/control 31.8 LFT, serum electrolyte, thyroid profile and blood sugar normal. NT pro BNP : 1734 pg/ml.

Coronary angiography CT showed 40% narrowing in proximal LAD.

ABG: pH 7.47, pCO<sub>2</sub> 37.5, pO<sub>2</sub> 60.5, spo<sub>2</sub> 94% Lactate 12.4 mg/ dl, glucose: 356 mg/ dl

### Management:

On airway evaluation the mallampatti score was Class I and neck movements were normal. Two bottles of packed cells were kept ready. cardiac operation theatre was kept ready. The patient was kept nil orally for 8 hrs. Budecort nebulisation was given the previous night and on the day of TAVI. Early in the morning pantoprazole 40 mg, eleprenolone 25 mg, invabradine 5 mg, aspirin 100 mg and clopidogrel 75 mg were given with sips of water. Antidiabetic were omitted as blood sugar was 146 mg/ dl. at 7.00 am. Inj hydrocortisone 100 mg was given intravenously. Before shifting to the cardiac cath laboratory, premedication Inj pentazocine 30 mg and Phenergan 25 mg were given intramuscularly.

On arrival at the cardiac cath laboratory, the patient was calm. Standard monitorings were set up including (5- lead ECG, pulse oximetry, NIBP). The peripheral line was placed and the Ringer lactate started. Central line three lumen catheter in right IJV and arterial line with 20 G catheter in right radial artery were placed under local anaesthesia before sedation started. Baseline parameters included, pulse -62/min, blood pressure -130 / 78 mm of Hg, SpO<sub>2</sub> - 94% on room air. A high flow nasal cannula (HFNC) was set up with 15 litres per minute flow rate started, (temp 37 °C and FiO<sub>2</sub>: 60%), patient was breathing spontaneously and oxygen saturation of 100% was achieved. The end-tidal concentration of the CO<sub>2</sub> cannula tip was plastered closed to the nostril to analyse ETCO<sub>2</sub>. Temporal leads for BIS applied and monitored. 12 External defibrillator pads attached over the chest and connected to a machine for emergency use.

Noradrenaline infusion of 8mg in 50 ml of Normal saline was prepared and connected to the central line and started at 2 ml per hour preemptively. The emergency drugs prepared and kept ready were Atropine, adrenaline 1:100 dilution syringes, xylocard, avil and effcorlin. As plan B, general anaesthesia drugs were kept ready.

In the beginning, sedation was given with Inj midazolam 0.5 mg and Inj fentanyl 50 micrograms intravenously. Then Inj dexmedetomidine (100 micrograms/ 50 Cc NS) started at 0.5 micrograms/ kg/hr. (12 ml) started through the central line. BIS was maintained between 45-70 and ETCO<sub>2</sub> was maintained at 30-40 mm of Hg., SpO<sub>2</sub> maintained at 100%.

Before femoral puncture Injection fentanyl, 25 micrograms was given intravenously to avoid leg movements. 20 cc of 2% local anaesthesia was infiltrated in the groin before the cannulation. BIS depth of sedation of 45 was achieved.

Left femoral vein cannulated with 6 F sheath and pacemaker lead placed in Right ventricular apex. The pacemaker was set the demand mode. Right femoral artery (RFA) cannulation was done with a 6 F sheath. Progressive dilatation of RFA done and an 18 F sheath was placed. Anticoagulation achieved with heparin 1 mg/kg, ACT achieved was 331 sec.

Fentanyl 25 microgm bolus was given prior to balloon dilatation. Under fluoroscopic guidance, the balloon aortic dilatation of the valve done. The EVOLUTE pro 26 mm valve was deployed in stages making sure of correct positioning with rapid ventricular



Fig no 1 : Pre induction HFNC and BIS along with other monitoring setup.



Fig no 2: a) pre procedure pressure gradient across aortic valve, b) RVP during valve deployment



pacing(RVP) at 180/min. SBP dropped to 45-55 mm of Hg. By serial fluoroscopy and aortography valve position was confirmed. Once the valve deployed, RVP stopped. Normal-pressure restored by giving noradrenaline in a small dose and 100 ml Bolus of fluid.

On Trans Thoracic Echocardiography(TTE) valve position was confirmed. There was no gradient across the valve, trivial AR, and LVEF was 40%.

After satisfactory valve position confirmation, Dexmedetomidine infusion was discontinued. Optimal homeostasis achieved. Suture ligation pain was controlled by giving Injection Perfalgan 1 gm iv. There was no incidence of apnea, hypercapnia, hypoventilation and hypoxemia noted during the procedure. The patient was hemodynamically stable all through with minimal support of norepinephrine which was started 15 mins after the start of sedation and reduced to minimal before shifting to ICU.

There were no adverse events such as tachyarrhythmias, hypotension, CPR or an emergency need for extracorporeal circulation(ECC). The procedure was uneventful. No rescue medicines like etomidate or propofol were used. The patient tolerated the procedure very well.

The total duration of the procedure was 120 mins.

Post-procedure the patient was fully awake responding to verbal commands with a Richmond –Agitation Sedation score index(RASS) of 2 ( opens eyes on the verbal command not sustained more than 10 sec) and the BIS of 92. Patient was shifted to ICU for further hemodynamic monitoring.ECG showed normal sinus rhythm without atrioventricular blockages. The patient was in the ICU for 3 days and discharged from the hospital on the 5th day.

### Discussion:

Different anaesthesia agents propofol, fentanyl, midazolam and remifentanyl, sufentanil, ketamine and a combination of one or more agents along with or without muscle relaxants were used to provide the still and stable conditions for TAVI.[3,5,6]

With increasing experience, technical expertise and improved smaller size femoral sheath availability, TAVI is being done under dexmedetomidine or propofol+ fentanyl combination. Both groups were compared concerning the periprocedural gas exchange and hemodynamic support. Dex was associated with a lower incidence of apnea/hypercapnia, the requirement for hemodynamic support and a lower rate of unplanned intubation. Thus DEX was a more promising alternative to PO for TAVI.[7] N Patrick et al, Lorenda et al confirmed these findings and additionally, the DEX group has less delirium, reduced ICU / hospital stay..[6]L.Bergmann et al studied retrospectively 100 patients who for TAVI under sedation,(remifentanyl and midazolam) versus general anaesthesia. In these patients, ICU/ hospital stay and mortality were comparable. Post-procedure ionotropic support was more in the GA group. [5]

Dexmedetomidine is a highly selective alpha 2 adrenergic receptor agonist associated with sedative, analgesic, anxiolytic ( reduced delirium and agitation), perioperative sympatholytic, cardiovascular and respiratory stability. Dexmedetomidine has a distribution half-life of 6 minutes and an elimination half-life of 2 hrs. Context-sensitive half-life varies from 4 minutes to 250 minutes depending on the time of infusion. Due to the short elimination half-life, dexmedetomidine is used for cooperative sedation. Dexmedetomidine enhances cognitive function, decreased cerebral oxygen requirement, decreased cerebral blood flow, reduces excitotoxicity, improves perfusion to ischemic penumbra. Dexmedetomidine does not affect respiratory rate and gas exchange.[10,11]

This patient had cognitive decline with right parietal-temporal infarct and post-covid lung status. So dexmedetomidine was the agent of choice. Instead of a loading dose, we gave our patient an infusion at .50 mcg kg/hr to avoid biphasic response (Initial increases in blood pressure followed by fall).

Post covid patient should be symptom-free and RT PCR should be negative. For mild to moderate covid 4 weeks interval for the elective procedure is recommended. Post covid anaesthesia concerns include residual pulmonary dysfunction, arterial and venous emboli(11.5% in non-ICU and 29.4% in ICU) and anticoagulation therapy-related concerns( administration of heparin or tissue plasminogen activator for clot lysis), stress cardiomyopathy, adrenal insufficiency associated with steroid administration for more than 3 weeks, neurologic events like stroke, haemorrhage, critical illness myopathy and post-traumatic stress disorder.[3,9,12,13]

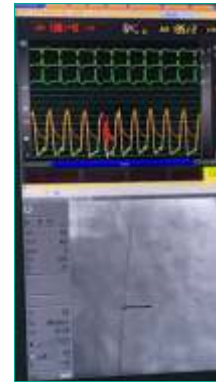


Fig no. 3  
Post procedure  
gradient across  
aortic valve.



Fig no 4: post procedure  
BIS @ 94 and patient  
opening eyes on command  
before shifting to ICU

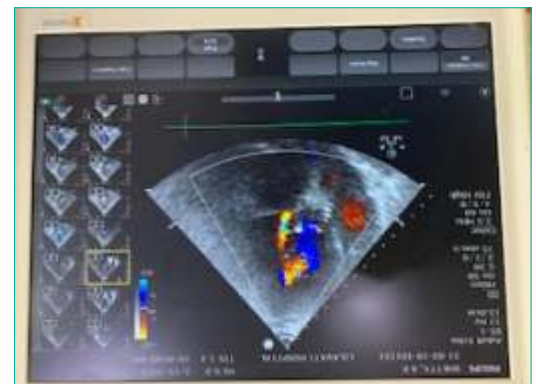


Fig no 3 : a) post valve deployment TTE  
shows two PVL jet present  
b) post balloon dilation of valve -  
mild AR No PVL.

This patient had 4 weeks interval from the negative RT-PCR report. This patient had COVID affected lung with room air sat of 92-94% and PaO<sub>2</sub> of 60.5 and saO<sub>2</sub> of 94 %, and breath-holding capacity of 4 seconds and because of cognitive decline the patient was unable to do pulmonary function tests. Hence we decided to use HFNC.

HFNC is a versatile and user-friendly device, allows constant nasopharyngeal PEEP, which in turn increases interalveolar volume and thereby increase functional reserve capacity. It provides hot and humidified air hence workload of breathing is reduced. High flow prevents air mixing. Thus the constant fixed concentration of oxygen is delivered to the patients.[10]

HFNC 15 litres 60 % and 37 °C was chosen preemptively to avoid any desaturation condition intra-procedural and we were able to maintain saturation at 100 % without any hiccups. During RVP saturation had dropped which coincided with a drop in blood pressure as expected, but soon recovered. No episodes of apnea or hypoventilation occurred and the patient remained hemodynamically stable.

Since the procedure was done in MAC with sedation, the patient was assessed by serial fluoroscopy and aortography with post-procedure TTE.

## CONCLUSION:

We successfully managed TAVI under dexmedetomidine infusion, using HFNC in a patient with post covid status, depth of sedation monitored with bispectral index, under fluoroscopic guidance and serial aortography. TTE was done immediately after valve placement to confirm the position and presence or absence of a paravalvular leak. Rescue medicine fentanyl top-up was needed before the femoral puncture and valve implantation to control pain induced limb movements.

Adequate airway assessment, an experienced cardiac anesthesiologist and a team of cardiologists with the required expertise and a good backup plan is key to successful management of TAVI under MAC with dexmedetomidine.

## REFERENCES:

1. Hadi Mahmaljy, Adam Tawney, Michael Young et al: Transcatheter Aortic Valve Implantation. stat Pearls(Internet), Treasure Island(FL) statpearls publishing, 2021 JAN: PMID28613729.
2. Robert G.Nampi, MD Liliya Pospishil, MD Peter J. Neuburger, MD et al: TAVR Versus SAVR for the Treatment of Aortic Stenosis: Do We Have a Clear Winner? Editorial / JCVA, 34, (2020) 21002102
3. Mohammed.A. Balghith, a,b Ahmed.A. Arifi, a,b,\* Dalia.M. Ahmed et al: The Impact of COVID-19 Pandemic on the Hospital Management of TAVI Patients: TAVI Team Thoughts and Recommendation. J Saudi Heart Assoc. 2020; 32(5): 11–15. Published online 2020 May 18. DOI: 10.37616/2212-5043.10303.
4. Hee-sun Park, Kyung-Mi Kim, Kyoung-Woon Joung et al: Monitored anaesthesia care with dexmedetomidine in transfemoral percutaneous trans-catheter aortic valve implantation: two cases report. Korean J Anesthesiol. 2014 Apr; 66(4): 317–321. DOI: 10.4097/kjae.2014.66.4.317.
5. Bergmann L, Kahlert P, Eggebrecht H, Frey U, Peters J, Kottenberg E. Transfemoral aortic valve implantation under sedation and monitored anaesthetic care feasibility study. Anaesthesia. 2011;66:977–982. [PubMed] [Google Scholar]
6. Patrick Mayr1, G Wiesner1, P van derStarre2, J Michell1, G Goppell1, M Erlebach1, M Kasell1, C Hengstenberg1, O Husser1, H Schunkert1, P Tassani1: Dexmedetomidine versus propofol/opioid for sedation in TAVI: a propensity-matched analysis of effects on perioperative gas exchange and haemodynamic support, JCVA: 31 (2017) S34–S69.
7. Loredana Cristiano, Francesco Coppolino 2, Valerio Donatiello: Use of Dexmedetomidine in transfemoral Transcatheter Aortic Valve Implantation (tf-TAVI) Procedures. Advances in therapy: 37, pages 2337–2343 (2020) Original Research, Published: 15 April 2020.
8. Wei HE1, Rong-Rong HUANG1, Qing-yu SHI 1, Xian-bao LIU2, Jian-an WANG2, Min YAN†‡1 et al Bispectral index-guided sedation in transfemoral transcatheter aortic valve implantation: a retrospective control study. J Zhejiang Univ-Sci B (Biomed & Biotechnol) 2017 18(4):353-359 35310.
9. Fabio Guarracino1, Stanton K Shernan 2, Mohamed El Tahan 3, Pietro Bertini 4, Marc E Stone 5, Bessie: EACTA/SCA Recommendations for the Cardiac Anesthesia Management of Patients With Suspected or confirmed COVID-19 Infection: An Expert Consensus From the European Association of Cardiothoracic Anesthesiology and Society of Cardiovascular Anesthesiologists With Endorsement From the Chinese Society of Cardiothoracic and Vascular Anesthesiology. PMID: 33766471 PMCID: PMC7889009 DOI: 10.1053/j.jvca.2021.02.03911.
10. Yigal Helviz 1 and Sharon Einav 1,2: A Systematic Review of the High-flow Nasal Cannula for adult patients. Crit Care. 2018; 22: 71. Published online 2018 Mar 20. DOI: 10.1186/s13054-018-1990-4.
11. Candiotti KA, Bergese SD, Bokesch PM, Feldman MA, Wisemandle W, Bekker AY. Monitored anaesthesia care with dexmedetomidine: a prospective, randomized, double-blind, multicenter trial. Anesth Analg. 2010; 110:47–56. [PubMed] [Google Scholar]
12. Jamel Ortoleva MD, Anesthetic Considerations for Recovered COVID-19 Patients, Journal of Cardiothoracic and Vascular Anesthesia (2020), DOI: <https://doi.org/10.1053/j.jvca.2020.10.032>
13. Bartłomiej Perek, Anna Olasinska-Wisniewska, Marcin Mistowski et al: How the COVID-19 pandemic changed the treatment of severe aortic stenosis: a single cardiac centre experience. J Thorac Dis 2021; 13(2):906-917 | <http://dx.doi.org/10.21037/jtd-20-3025>
14. Syed Maqbool, Vijay Kumar, Vishal Rastogi, and Ashok Seth: Transcatheter aortic valve implantation under conscious sedation – the first Indian experience Indian Heart J. 2014 Mar; 66(2):208–210. DOI: 10.1016/j.ihj.2014.02.004

## CASE REPORT IV: PAEDIATRIC SURGERY

### A Case of Synchronous Malignancy with Novel Missense Mutation in a Child: Is This Li-Fraumeni Syndrome or a Novel Case Masquerading as Li-Fraumeni Syndrome??

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#### ABSTRACT:

We report the case of an 11-month-old child who presented with a change in voice, increased weight, and hirsutism, who was also found to have elevated levels of serum cortisol and testosterone, showing three synchronous malignancies in the liver, left adrenal gland, and posterior mediastinum. Clinical exome sequencing report revealed germline TP53 (P177A) and MLH3 (V741P) mutations with NMYC positive neuroblastoma. At the outset, this may look like a Li-Fraumeni syndrome (LFS) with TP53 germline mutation but lacks other features to be termed as LFS or Li-Fraumeni-like syndrome. The gene mutation variant found in this case (P177A) is a novel missense mutation which has never been reported, and the MLH3 gene mutation variant V741P has not been previously associated with any of the malignancies seen in this child.

**KEYWORDS:** *Adrenocortical malignancy, hepatoblastoma, mismatch repair gene, neuroblastoma, synchronous malignancy*

#### INTRODUCTION

Synchronous malignancies are rare occurrences in children and pose a challenge in terms of chemotherapy and surgery.[1] This challenge is intensified if there is an association with TP53 which predisposes the individual to development of tumors when exposed to chemotherapy or radiotherapy.[2] This is such a case which had high-risk malignancies otherwise treated by high-dose chemotherapy and/or radiotherapy, but because associated with TP53 and MLH3 gene mutations, management strategy had to be changed. The rarity of the gene mutations associated makes it a unique case. Li-Fraumeni syndrome (LFS) is a familial cancer syndrome associated with multiple malignancies and TP53 gene mutation.[3] This case report comes close to being labeled as LFS.

#### CASE REPORT

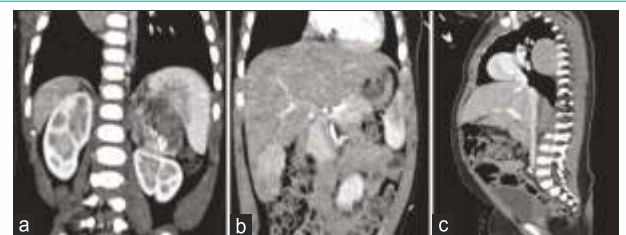
An 11-month-old male child was admitted with the complaints of hoarseness of voice and hirsutism from 8 to 9 months of age. The child had generalized coarse hair overgrowth over the entire body along with weight gain for which an endocrine consultation was done. Hormonal assay and radiological imaging studies were done after the endocrine consult.

Medical investigations revealed high levels of serum cortisol (20.85 µg/dL) and elevated testosterone levels (free testosterone 250 ng/dL and dihydrotestosterone 2500 ng/dL). Urinary vanillylmandelic acid levels were normal, and alpha-fetoprotein level raised to 912 ng/mL (normal <10 ng/mL).

Radiological investigation revealed a mass in the left adrenal gland, which was heterogeneous and well encapsulated with areas of focal calcification seen within the tumor [Figure 1a]. There was an exophytic mass in the segment VI of the liver seen without any extrahepatic, inferior vena cava (IVC), portal vein, or nodal involvement [Figure 1b]. Computed tomography also revealed a well-encapsulated mass in the posterior mediastinum extending between the T4 and T7 vertebrae without any intraspinal extensions [Figure 1c]. The lungs were clear of metastasis, so was the brain.

There were no dysmorphic features present in the child and no history of malignancy in the siblings.

The mother had a history of six previous abortions and hormonal treatment during pregnancy. The abortions were not spontaneous but for



**Figure 1:** (a) Radiological image of adrenocortical carcinoma, (b) radiological image of hepatoblastoma, (c) radiological image of neuroblastoma

wanting a male child. The child's father was formerly treated for renal cell carcinoma, underwent partial nephrectomy 5 years ago, has not received any chemotherapy postprocedure, and is symptom free. There was no other family history of any malignancy.

There were three masses in this child and in order to get a bearing as to which is primary and which is metastasis, we decided to biopsy one of them and the safest was to perform a liver biopsy. He underwent an ultrasound-guided liver biopsy under corticosteroid cover, and it was reported as fetal variant hepatoblastoma (ICD-O-3 code 8970). Further, a right thorascopic biopsy was done to rule out metastasis, but it was reported to be a neuroblastoma stroma poor type (ICD-O-3 code 9490).

After ruling out adrenal medullary involvement (by urinary vanillylmandelic acid and homovanillic acid level) to prevent the intraoperative adrenal storm, he underwent a left adrenalectomy [Figure 2a] and excision of hepatoblastoma under corticosteroid cover [Figure 2b]. The left adrenal mass was reported as adrenocortical malignancy (ICD-O-3 code 8370) without a breach in the capsule and the hepatoblastoma was of fetal variant without any hepatic vein, IVC, or extrahepatic involvement and clear margins of resection. The chest mass was excised 2 weeks later, and its biopsy was reported as neuroblastoma, which was well differentiated and of stroma poor type [Figure 2c].

Hormonal evaluation post tumor excision revealed a reduction in the levels of cortisol (2.0 µg/dL), testosterone (free testosterone 0.1 ng/dL and dihydrotestosterone 27 ng/dL), and alpha-fetoprotein (3.0 ng/mL). Currently, the levels of the elevated hormones have normalized, and he is off steroids which were stopped after tapering doses.

Because the child has three histologically distinct malignancies, gene mutation studies were done which revealed germline TP53 gene mutation (c. C530G, p. Pro177Arg) with allelic ID 468390 and MLH3 gene (c. G2221T, p. Val741Phe) mutations. Interphase fluorescent in situ hybridization (FISH) technique revealed a 15-fold increase of NMYC signals to chromosome enumeration probe signals, confirming NMYC amplification. Following this, multiplex ligation-dependent probe amplification (MLPA) assay was also performed on the neuroblastoma tissue to check for segmental chromosomal anomalies on chromosomes 1, 2, 3, 4, 7, 9, 11, 12, and 17 [Table 1]. The parents were offered to undergo genetic testing to find out any genetic abnormalities, but they refused to undergo such tests.

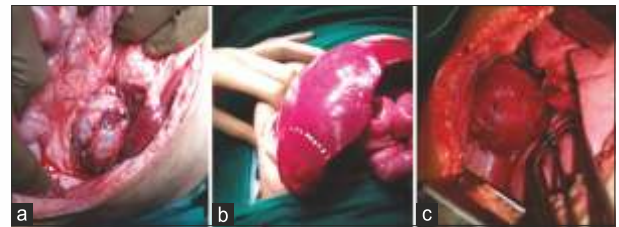
While on cisplatin and doxorubicin regimen chemotherapy (drugs common for both hepatoblastoma and neuroblastoma), he presented with convulsions (probably hypoglycemic) and shock requiring inotropic support and cardiopulmonary resuscitation. He was then diagnosed to have severe chemoport-site sepsis (culture positive), which had to be removed once stable. Post this incident, the child developed hypoxic brain injury with neurological deficits in the form of weakness and regression of milestone. He is now on the road to recovery. At the time of reporting this case, he was not on any chemotherapy and was undergoing limb physiotherapy.

## DISCUSSION

Primary multiple tumors are defined as malignancies with different histopathological origin in one person. Its increased incidence seen nowadays is owing to better diagnostic techniques and improved treatment methods.[4] Multiple malignancies are reported along with TP53 mutation and LFS. Two distinct histological types of malignancies predominate the TP53 mutation reports. The most commonly associated malignancies with germline TP53 mutations and LFS are the adrenocortical malignancies, soft-tissue sarcomas, brain tumors, and breast tumors.[5]

There are multiple criteria which have been laid down for the identification of LFS which are classic and Chompret.[6] For those who do not fit into the classic criteria, Birch and Eeles have laid down criteria for identifying Li-Fraumeni-like syndrome (LFL). There is another syndrome which is described in literature called hereditary neoplastic syndrome. This is also associated with multiple malignancies and characterized by earlier onset of tumors than their described age. However, these are associated with endocrine tumors.[7]

Our patient does not fit into the LFS or LFL with any of the criteria laid down even with the presence of a renal cell carcinoma in his father. This



**Figure 2: (a) Intraoperative picture of adrenocortical carcinoma, (b) intraoperative picture of hepatoblastoma, (c) intraoperative picture of neuroblastoma**

**Table 1: Genetic and molecular findings on multiplex ligation-dependent probe amplification and fluorescent in situ hybridization in cases of neuroblastoma and its significance**

Genetic alteration	Copy status	Significance
ALK	Normal	Helps activating <i>NMYC</i> , high risk tumor
DDX1	Amplification	High risk tumor
<i>NMYC</i>	Amplification	High risk tumor
1p	None	Near-diploid or tetraploid tumors
11q23	Normal	Higher disease state and poor prognosis
17q	Gains	Poor outcome
2p	Amplification	Very aggressive course
3p	Normal	Tumor suppressor gene, higher age at diagnosis
4p	Normal	Tumor suppressor gene
7q	Normal	Tumor suppressor gene



makes our case a unique one with germline TP53 mutation and three histologically different malignancies (one of them being adrenocortical carcinoma) not matching the criteria for LFS and having malignancies other than the ones usually reported with LFS or LFL. This is a novel mutation, but its clinical significance needs to be proven in future.

Considering this case as a novel case of synchronous malignancies, the age of presentation also makes this unique, as most commonly the multiple malignancy syndromes are seen in the second or third decade.[8] This makes our case a unique one as the three distinct histopathological malignancies are reported at the age of 11 months. Three other reports were found where the patients are aged 6, 8, and 10 months.[3,9,10]

There are multiple cases of neuroblastoma which are reported with TP53 gene mutation [Table 2]. There are six reported cases of TP53 mutation with adrenocortical cancer (ACC) and neuroblastoma. This case is seventh such report.

**Table 2: Cases of neuroblastoma combined with other tumors and TP53 mutation**

Case number	Location	Year	Age/sex	Function	Gene variant	Exon	Other associated malignancy	NMYC amplification	Reference
1	The USA	1998	18 months/male	Loss of function	R248W 45XO	7	ACC	No	[1]
2	The USA	2008	10 months/male	Loss of function	R248W	7	ACC	No	[10]
3	Brazil	2015	NA	Loss of function	R337H	10	ACC	Present	[11]
4	Brazil	2015	NA	Loss of function	R337H	10	ACC	Present	[11]
5	The USA	2015	8 months/male	Unknown	I162F	5	ACC	No	[9]
6	China	2017	6 months/male	Loss of function	N268E	8	ACC	No	[3]
7	The UK	2014	2 years/male	Loss of function	R248Q	Whole exome	Benign myofibroblastic proliferation and sarcoma	Present	[5]
8	The USA	2013	3 years/male	Loss of function	P219S	Whole exome	High risk neuroblastoma	Present	[2]
9	The UK	1992	1 year/male	Loss of function	R273H	NA	Osteosarcoma	NA	[12]
10	India	2018	11 months/male	Unknown	R177A	5	Hepatoblastoma and ACC	Present	This study

ACC – Adrenocortical cancer; NA – Not available

The reported mutation in TP53 gene in our present case is a missense mutation c. C530G, p. Pro177Arg (P177A) with allelic ID 468390. This germline mutation has been identified in scientific literature to be present within the DNA-binding domain of TP53 but has not been biochemically characterized, and, therefore, its effect on TP53 protein function is still unknown, although speculated to have a partially gain-of-function mutation.[13] According to the International Agency for Research on Cancer (IARC) TP53 database, there has been only one somatic TP53 P177A mutation identified in colorectal cancer and no germline TP53 P177A has been identified till August 2018. Totally, there are 498 gene variants identified with TP53 mutation in the IARC TP53 database.[14]

The clinical exome sequencing also identified a mutation in the MLH3 gene which was a missense mutation c. G2221T, p.Val741Phe (V741P) which is associated with hereditary nonpolyposis colorectal cancer (HNPCC) type 7. This type of HNPCC is known to cause early-onset colorectal, endometrial, gastric, and breast cancers.[15] According to the Centre for Genomic Study and Genetics, Zhejiang University, there are a total of 54 gene variants which are described for MLH3 mutation and most of them are on exon 2, as in our case. According to the same genetic registry, there have been only two instances of reports with V741P mutation, which were seen in adult familial endometrial carcinoma and colorectal cancer.[16]

The neuroblastoma tissue which was subjected to check for NMYC amplification test by interphase FISH assay revealed a 15-fold increase in the NMYC signals. Further, MLPA assay was performed to identify segmental chromosomal anomalies, which revealed alterations as tabulated in Table 1.

This case is particularly unique due to presentation with three histologically malignant tumors at 11 months of age. It did not fit into the criteria laid down for LFS in the presence of TP53 mutation and ACC with neuroblastoma. The gene mutation variant found is a novel missense mutation which has never been reported in germline mutation, while only one case is reported that of a somatic mutation (with colorectal cancer).[14] The MLH3 gene mutation variant V741P has not been previously associated with any of the malignancies seen in this child.[16] This indirectly may point toward the child developing any other malignancy association with this genetic aberration later in his life.

The MLPA assay and NMYC amplification rendered the neuroblastoma as a high-risk tumor. The chemotherapy regimen for high-risk neuroblastoma with NMYC, DDX1, and 2p amplifications consists of high-dose chemotherapy, irradiation, followed by immunotherapy.[17] The treating doctors were pushed against the wall for deciding the therapy as radiotherapy or high-dose chemotherapy cannot be performed because of TP53 mutation as these patients are known to develop more malignancies when exposed to radiation or other cytotoxic drugs.

A management protocol for these patients with multiple malignancies cannot be formulated as these are one-off presentation and each such presentation must be dealt individually.

### Conclusion – Take-Home Message

Multiple synchronous malignancies are a rare occurrence in children. Cytogenetics (karyotyping, FISH, and comparative genomic hybridization) and molecular cytogenetics (MLPA and restriction fragment length polymorphism) in these cases help identifying the known mutations, whereas molecular cytogenetic techniques (denaturing gradient gel electrophoresis, single-strand conformational polymorphism, and heteroduplex analysis) will identify the unknown mutations. Genetic mapping of parents and siblings is crucial to identify inherited mutations or de novo mutations. Establishing a chemotherapy regimen and tailoring it for these patients requires a multidisciplinary approach. Follow-up of these children with an eye out for a possible malignancy is of utmost importance.

### Consent

A written informed consent has been obtained from the parents of this patient for publication of this case report and accompanying images. A copy will be made available at request.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient's parents have given consent for their child's images and other clinical information to be reported in the journal. The patient's parents understand that their child's name and initial will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

### REFERENCES:

1. Pivnick EK, Furman WL, Velagaleti GV, Jenkins JJ, Chase NA, Ribeiro RC. Simultaneous adrenocortical carcinoma and ganglioneuroblastoma in a child with Turner syndrome and germline p53 mutation. *J Med Genet* 1998;35:328-32.
2. Pugh TJ, Morozova O, Attiyeh EF, Asgharzadeh S, Wei JS, Auclair D, et al. The genetic landscape of high-risk neuroblastoma. *Nat Genet* 2013;45:279-84.
3. Tang YJ, Yu TT, Ma J, Zhou Y, Xu M, Gao YJ. Composite adrenocortical carcinoma and neuroblastoma in an infant with a TP53 germline mutation: A case report and literature review. *J Pediatr Hematol Oncol* 2019;41:399-401.
4. Moertel CG, Dockerty MB, Baggenstoss AH. Multiple primary malignant neoplasms. I. Introduction and presentation of data. *Cancer* 1961;14:221-30.
5. Behjati S, Maschietto M, Williams RD, Side L, Hubank M, West R, et al. A pathogenic mosaic TP53 mutation in two germ layers detected by next generation sequencing. *PLoS One* 2014;9:e96531.
6. Tinat J, Bougeard G, Baert-Desurmont S, Vasseur S, Martin C, Bouvignies E, et al. 2009 version of the Chompret criteria for Li Fraumeni syndrome. *J Clin Oncol* 2009;27:e108-9.
7. Frank TS. Hereditary cancer syndromes. *Arch Pathol Lab Med* 2001;125:85-90.
8. Gonzalez KD, Noltner KA, Buzin CH, Gu D, Wen-Fong CY, Nguyen VQ, et al. Beyond Li-Fraumeni syndrome: Clinical characteristics of families with p53 germline mutations. *J Clin Oncol* 2009;27:1250-6.
9. Courtney R, Ranganathan S. Simultaneous adrenocortical carcinoma and neuroblastoma in an infant with a novel germline p53 mutation. *J Pediatr Hematol Oncol* 2015;37:215-8.
10. Rossbach HC, Baschinsky D, Wynn T, Obzut D, Sutcliffe M, Tebbi C. Composite adrenal anaplastic neuroblastoma and virilizing adrenocortical tumor with germline TP53 R248W mutation. *Pediatr Blood Cancer* 2008;50:681-3.
11. Seidinger AL, Fortes FP, Mastellaro MJ, Cardinalli IA, Zambaldi LG, Aguiar SS, et al. Occurrence of neuroblastoma among TP53 p.R337H Carriers. *PLoS One* 2015;10:e0140356.
12. Porter DE, Holden ST, Steel CM, Cohen BB, Wallace MR, Reid R. A significant proportion of patients with osteosarcoma may belong to Li-Fraumeni cancer families. *J Bone Joint Surg Br* 1992;74:883-6.
13. Kotler E, Shani O, Goldfeld G, Lotan-Pompan M, Tarcic O, Gershoni A, et al. A systematic p53 mutation library links differential functional impact to cancer mutation pattern and evolutionary conservation. *Mol Cell* 2018;71:178-90, e8.
14. IARC TP53 Search. Available from: <http://p53.iarc.fr/TP53GeneVariations.aspx>. [Last accessed on 2019 Jul 18].
15. Liu HX, Zhou XL, Liu T, Werelius B, Lindmark G, Dahl N, et al. The role of hMLH3 in familial colorectal cancer. *Cancer Res* 2003;63:1894-9.
16. All genes-Zhejiang University Center for Genetic and Genomic Medicine (ZJU-CGGM) & Dian Diagnostics. Available from: <http://www.genomed.zju.edu.cn/LOVD3/genes> [Last accessed on 2019 Jul 18].
17. Casey DL, Kushner BH, Cheung NK, Modak S, LaQuaglia MP, et al. Local control with 21-Gy radiation therapy for high-risk neuroblastoma. *Int J Radiat Oncol Biol Phys* 2016;96:393-400.

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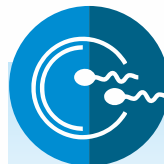


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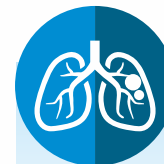
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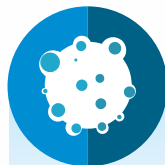
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## CASE REPORT V: PAEDIATRIC SURGERY

### Risk Prediction Scoring System to Predict the Postsurgical Outcomes of Biliary Atresia

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#### ABSTRACT

**Aim:** To find out association between liver function, liver histopathology and outcomes of biliary atresia (BA) following Kasai Portoenterostomy (KPE).

**Materials and Methods:** This is a retrospective study of children who underwent KPE at a single institute by single surgeon. The patient records analyzed and data of complete blood counts, liver function tests, coagulation profile and histopathology reports collected. The outcomes recorded as alive and jaundice free, alive but jaundiced, and deceased. Statistical analysis done using SPSS 23.

**Observations:** Total of 148 children operated during January 2000 to December 2018. Of these, 26 matched inclusion criteria. The parameters assessed were percentage of direct bilirubin, ratios of Aspartate transaminase (AST) to Alanine transaminase (ALT); Gamma glutamyl transferase (GGT) to AST; GGT to ALT and Aspartate transaminase to platelet ratio index (APRI). Among histopathology reports, fibrosis grade and bile ductular size noted. Among 26, 16 alive and ten are deceased. Among 16 alive, all are jaundice free. Of the parameters, ratio of AST to ALT, APRI and grade of fibrosis found statistically significant and further analysis showed if AST to ALT ratio  $< 2.1$ , APRI  $< 1.8$  and grade of fibrosis  $< 4$ , irrespective of age at surgery, had 96.2 % probability of successful KPE. Based on these observations, a scoring system and risk prediction model constructed based on Receiver operating characteristic (ROC) curves which are first in BA management.

**Results and Conclusion:** Although numbers are sufficient for statistical analysis, we further intend to validate the scoring system in a prospective trial. BA children can be subjected to risk prediction model and KPE performed in those who have a score less than seven and offered to those with score between eight and 16 out of 20.

**Key Message:** The scoring system and risk prediction model can guide in the management and post-operative follow up of children with biliary atresia.

**Keywords:** Aspartate platelet ratio index, biliary atresia, kasai portoenterostomy, outcomes, risk prediction model

#### INTRODUCTION

Biliary atresia if left untreated is almost surely fatal by 3 years of age due to liver failure.[1] There are no current guidelines for surgical management, and the available options are performing a kasai portoenterostomy (KPE) or liver transplantation.[2] Liver transplantation can be performed as a primary procedure or a rescue procedure following failed KPE.[3] The success of KPE is variable, and the reported success rates are over and above 50%.[4] There are multiple factors which are considered to play an important role in the outcome of these children, and the most important one was the age at surgery. The age at surgery was considered as a golden window of opportunity in these children, and when operated within this time frame, the likelihood of a successful outcome was higher. The outcome of these children can be represented by a spectrum, where on the one end, there are children who are jaundice free following KPE, and on the other end, there are children who have persistent symptoms.[1]

We have shown that the outcomes of these children who are operated between 30–60 days and 61–90 days were compared with those who were operated between 91 and 120 days.[5] Anticipating that there is more than just age at work which predicts the outcomes of these children, this is an attempt to identify these probable factors at work.

#### Materials and Methods

This is a retrospective observational study. After getting the institutional ethics committee clearance, the records of the operated children of biliary atresia by a single surgeon at the same institute were studied. Data of complete blood counts, liver function tests including coagulation profile, and TORCH titer reports done during the admission before the procedure were collected. After confirmation of diagnosis with an intraoperative cholangiogram, KPE was performed with a 40 cm Roux-en-Y limb. A liver biopsy was done at the same time. The children were followed up initially every month up to 3 months and 3 monthly up to 1 year followed by yearly visits up to at least 10 years of age. All the children who did not fulfill the above criterion were excluded from the study. All the children with syndromic biliary atresia were excluded from the study. Further, the data were analyzed, and percentage of direct bilirubin was calculated, aspartate transaminase (AST)-to-alanine



transaminase (ALT) ratio was calculated, aspartate platelet ratio index (APRI) was calculated, and gamma-glutamyltransferase (GGT)-to-AST and GGT-to-ALT ratios were calculated. The histopathology report was analyzed, and data regarding the grade of fibrosis according to Ishak grading,[6] the bile ductular size, and the presence of cytomegalovirus (CMV) inclusion bodies studied by H and E staining and presence of CMV antigens by immunohistochemistry (IHC) (Biocare Medical DT 10+ BC 90 CMV clone) staining were collected. All the data collected were tabulated and analyzed using SPSS version 23.0 software (IBM, NY, USA).

### Observation and Results

Out of the 149 cases, there were 26 cases which fulfilled the above criteria and included in the study. There were 10 female and 16 male children. The youngest of the infants operated was of 40 days and the oldest infant was 169 days. All the children underwent KPE and liver biopsy at the same time. Among 26 children, 10 are deceased and 16 are alive, and all the alive children are jaundice free with the minimum follow-up period of 1 year. The eldest child alive and jaundice free among these is 9 years old. The calculated percentage of direct bilirubin ranged from 61% to 99%. The AST-to-ALT ratio ranged from 0.62 to 3.64, whereas the APRI ranged from 0.36 to 5.15. The calculated ratios between GGT to AST and GGT to ALT ranged between 0.24–7.32 and 0.33–13.38, respectively. The grade of fibrosis as per Ishak staging ranged from 1 to 6 [Table 1] and bile duct size from 20 to 2000  $\mu$  on the liver biopsy.

All the 26 liver biopsy tissues were negative for CMV antigens through IHC staining. Only one biopsy showed CMV antigen positive in lymph node. Among the parameters studied, the AST-to-ALT ratio,

**Table 1: Ishak stage**

Microscopic appearance	Grade of Fibrosis
No fibrosis	0
Fibrous expansion of some portal areas, with or without fibrous septa	1
Fibrous expansion of most portal areas, with or without short fibrous septa	2
Fibrous expansion of most portal areas with occasional portal to portal bridging	3
Fibrous expansion of portal areas with marked bridging	4
Marked bridging with occasional nodule (incomplete cirrhosis)	5
Cirrhosis, probable or definite	6

**Table 2: Statistical analysis**

Variables	Score	df	Significance	AUROC
Grade of fibrosis	6.754	1	0.009	0.816
AST/ALT ratio	7.490	1	0.006	0.866
APRI	6.949	1	0.008	0.863
Bile duct size	2.340	1	0.126	0.659
Overall statistics	18.604	4	0.001	

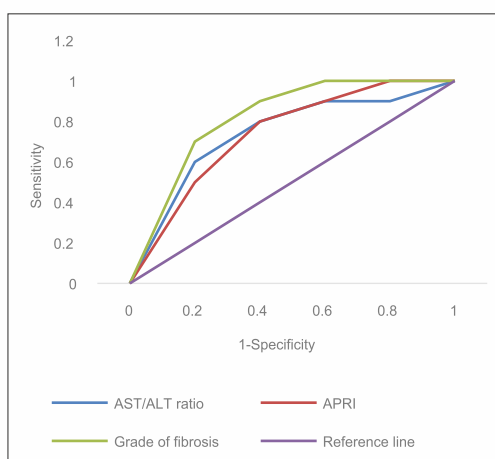
AUROC: Area under receiver operating characteristic curve, AST: Aspartate transaminase, ALT: Alanine transaminase, APRI: Aspartate platelet ratio index

Redkar, et al.: Risk prediction scoring system to predict the postsurgical outcomes of biliary atresia

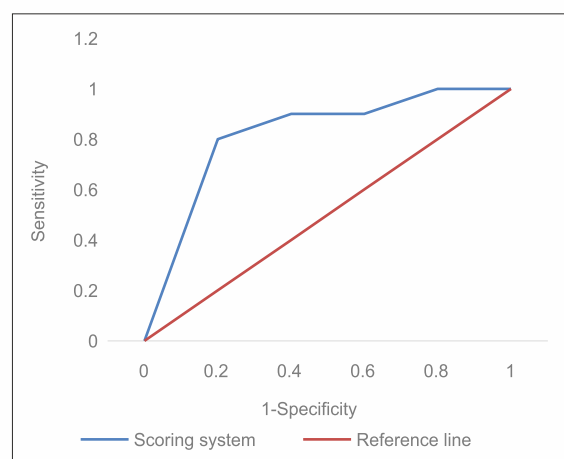
**Table 3: Raj and Redkar's scoring system and risk prediction model for post-surgical outcomes of biliary atresia**

Grade of fibrosis	Score	Ratio of AST/ALT	Score	APRI index	Score	Cumulative score	Risk
Grade 1	1	0-1.0	1	0-1	1	3-7	Mild
Grade 2	2	1.01-2.19	2	1.01-1.8	2	8-16	Moderate
Grade 3	3	2.20-2.39	3	1.81-2.39	3	17-20	Severe
Grade 4	4	>2.4	4	>2.4	4		
Grade 5	10						
Grade 6	12						

AST: Aspartate transaminase, ALT: Alanine transaminase, APRI: Aspartate platelet ratio index



**Figure 1: Receiver operator curve parameters**



**Figure 2: Receiver operator curve scoring system**

APRI index, and grade of fibrosis were found to be statistically significant in predicting the outcome of these children post KPE.

It was found that the ratio of AST to ALT, APRI index, and grade of fibrosis had an impact on outcome of these children post KPE [Table 2]. When these factors were found to be statistically significant, it was then tried to find the corroborative impact of these factors when added together. This led to finding that if the ratio of AST to ALT is  $<1.8$ , APRI  $<2.2$ , and grade of fibrosis is  $<4$ , then the rate of survival increases to 96.2% irrespective of age at surgery following KPE.

These parameters were statistically analyzed further to find the diagnostic power of these tests. For this, the adequacy of number of cases was calculated using the Kaiser-Meyer-Olkin measure of sample adequacy and factor analysis, and the numbers were found to be adequate. Further, receiver operating characteristic curve (ROC) for each of the parameters was plotted [Figure 1] and found that they can be used as tests for diagnosis when clubbed together. When all these three tests were found to have diagnostic strengths, a scoring system was developed [Table 3] incorporating these factors which would help predict a successful outcome following KPE in children with biliary atresia. Such a scoring system and risk prediction model is the first of its kind to the best of our knowledge.

To construct the scoring system, the grade of fibrosis, the ratio of AST to ALT, and APRI were graded according to clinical significance. The histological grade of fibrosis was given a higher value, and both the other parameters were given the same value. Using the scores given, the outcomes of our analysis were then plotted to draw ROC [Figure 2] which helped us to decide the cumulative cutoff for the risk prediction model. They are summarized in Table 2.

By applying this risk prediction score on our cases in the study, a 100% match for mild and severe risk group was seen [Figure 3]. Based on this risk prediction model, which was developed using the ROC, a cumulative score between 3 and 7 had a 100% survival rate, a score between 8 and 16 had a 45% survival rate, and a score of more than 17 had a 100% mortality rate irrespective of the age at operation.

We intend to further validate this scoring system by applying this to our prospective cases and analyze their results.

## DISCUSSION

Biliary atresia is a progressive disorder causing liver failure. The extent of liver failure can be variable depending on the time of onslaught of etiological process which can either be antenatal or postnatal. Hence, the child presents at different ages with different clinical profiles and different degrees of liver damage. The exact cause of biliary atresia is unknown but speculated to occur in three forms:[4] the syndromic form also known as biliary atresia splenic malformation or BASM, the acquired form found sporadically, and the ones associated with CMV. Biliary atresia associated with CMV has bagged a name of its own due to its propensity to have worse prognosis than the rest.[7] With the etiology being unclear and the age at surgery which was considered as a window of opportunity for success being challenged, an attempt was made to find the other probable factors at play which lead to progressive liver damage and failure. It was also found that age is just a number and does not individually influence the outcomes of these children,[5] and this is an attempt to find other probable factors at play.

Looking at the literature, there are several factors which have been studied at the time of surgery to predict the outcomes of these cases post KPE. These can be grouped into the effect of age at surgery,[8] effect of immune mediators at the time of surgery,[9] effect of type of biliary atresia, and effect of portal plate histology[10] including the level of fibrosis and cirrhosis of the liver.[11]

For finding the other probable factors at play, we focused on immune mediators, the histopathology of liver biopsy and liver function tests. To keep a similar playing field, we excluded the cases of syndromic biliary atresia that are known to have poor prognosis.

Various markers have been studied to identify the extent of liver damage in these children. Of these, the AST-to-ALT ratio is one of the most common factors studied and more so in the adult population,[12] and it was attempted to extrapolate the same in these children. Whenever there is a reversal of AST-to-ALT ratio, the liver seems to have undergone a certain degree of damage. As ALT is more specific for liver and AST has more than one source, low ALT indicates a failing liver. This ratio is found to be associated with chronic liver disease. This ratio of more than two is suggestive of cirrhosis and has been used as a surrogate marker in adults.[13] In our study, the ratio of  $<1.8$  had a good outcome post KPE.

Aspartate to platelet ratio index is another such study to predict the prognosis of biliary atresia and this was developed by Wai et al. for patients suffering from hepatitis C in order to prevent performing repeated liver biopsies.[14] In this article, they went on to conclude that this noninvasive test can be utilized for monitoring these patients with chronic hepatitis C, and this could replace performing repeated liver biopsies. Many other studies have been later conducted and found this test to be useful in monitoring the grade of fibrosis in liver.[15] While in children with biliary atresia, this has been used as a surrogate marker to predict their outcomes. In a study by Yang et al. APRI effectiveness in diagnosing significant liver fibrosis, especially cirrhosis, in biliary atresia infants was studied and they concluded that is it effective way to screen the children with biliary atresia.[16] APRI was calculated using the formula:

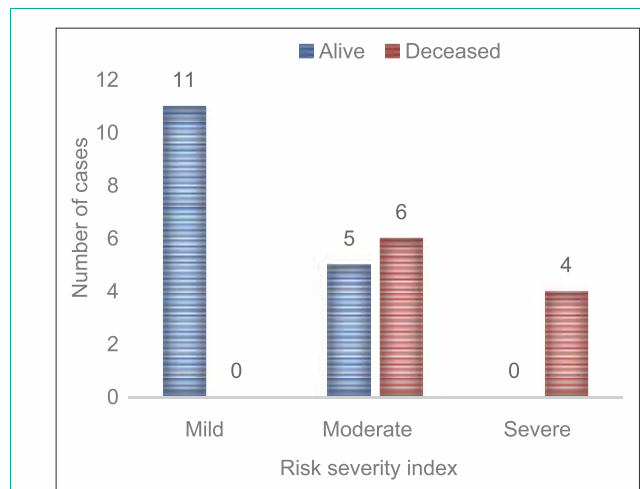


Figure 3: Risk prediction model

$APRI = (AST \text{ Upper limit of } AST / \text{Platelet counts in } 10) \times 100$

In another study by Davenport et al., APRI value of 1.22 had the sensitivity and specificity of 75% and 84% for identifying cirrhosis in biliary atresia children.[17] While in our study, we had a cut off value of 2.19 for predicting significant cirrhosis and poor outcome post KPE.

While in yet another study, APRI value of more than 3 was considered to have failure of jaundice clearance and need for liver transplantation.[18]

Further, in biochemical tests, we studied a percentage of direct bilirubin, and this was calculated using the formula:

$\text{Percentage of direct bilirubin} = (\text{Direct bilirubin} / \text{Total bilirubin}) \times 100$

This is probably more indicative of obstructive liver damage and probably removes the age of these children as a confounding factor. For analysis, they were divided into four groups, starting from 61%–70% to 91%–100%. The outcome when compared to percentage of direct bilirubin was not found to be statistically significant.

The GGT-to-AST and ALT ratios were also studied.[13] These ratios are studied previously in biliary atresia cases, but nothing conclusive has been proved so far. The present study also did not find this factor to be of any significance.

Liver biopsy is the next parameter requiring mention in the study. The biopsies were done at the time of KPE and all were reported by the same institutional histopathologist. The grading of fibrosis was done according to the Ishak staging.[6]

Among histological parameters, the level of  $\alpha$ -SMA is known to affect the degree of fibrosis. Higher the  $\alpha$ -SMA, higher was the grade of fibrosis and cirrhosis. This could help predict the outcomes of native liver survival in the children post KPE.[19] The other parameters studied are cytokeratin-7-positive percentage which is also a marker for fibrosis and cirrhosis. In our study, we had children who had Grade 1 fibrosis and had fibrosis all the way up to Grade 6. It was noted that higher the grade of fibrosis, higher was the failure rate of KPE which was statistically significant. Anything higher than Grade 4 had a significantly higher risk of a failed KPE. It was noted that greater age at surgery was not uniformly associated with higher grades of fibrosis or cirrhosis. This also shows that the time of onslaught of the disease process is not uniform in these children.

The bile ductular size in the portal plate was also assessed in our study. The sizes ranged from 20 to 2000  $\mu$ . It was noted that all children with larger bile ductules did not have uniformly successful KPE, but we did note that children with larger bile ductules faring better than those which smaller bile ductules. Although the bile duct size of  $>250 \mu$  has been reported in the literature to have a better outcome,[20] the size of bile duct was not found to be statistically significant in our study probably as a result of a very large range of values.

The role of CMV has been widely studied in relation to both etiology and prognosis of biliary atresia. In one of the studies, it was found that CMV-positive biliary atresia had a significantly higher degree of inflammation in the liver biopsy, poorer outcome, poorer jaundice clearance, poorer native liver survival, and increased mortality.[7] In the current study, analysis of TORCH titer was done in all the children and subjected their liver biopsies to IHC for CMV antigens. Twelve of the 26 had CMV IgG positive, and of these 12, only 6 had CMV IgM positive. On the respective liver biopsy IHC staining, none of these had CMV inclusion bodies and CMV antigens, while only one showed CMV antigens being positive in lymph node sent along with the biopsy. In a similar study on histopathology in biliary atresia involving CMV infections, there were 81 cases which were evaluated in which none of the cases had detectable CMV infection in liver biopsy[21] even though 27 of them were CMV IgM positive. In other studies, Fischler et al. had found CMV DNA from liver tissue from 9 of the 18 children that they were investigating[22] and the studies done by Domiati-Saad et al. had similar results.[23]

In a similar study by Jevon and Dimmick, they did not report CMV DNA in liver biopsy tissue in any of the 12 cases they were investigating.[24] The exact mechanism by which CMV causes obliteration of the bile duct is not known, but their affection is definitely proved when more intense stains were obtained from children who did poorly following surgery than those who did well after KPE.[25]

The results of this study indicate that whatever the etiology of biliary atresia is, does not affect all the children at the same time. The pathogenesis is a continuous process which manifests in varying degrees at different ages of the child. Hence, the time of affection of the liver is more important than age at surgery. With the three parameters stated above, we can identify those children with biliary atresia whose liver is salvageable with KPE as their disease process is not advanced to irreversible cirrhotic stage.

## CONCLUSION

Since biliary atresia is a progressive disorder and liver damage is ongoing, the exact extent of liver failure cannot be predicted based on the age of the patient. Using this scoring system and risk prediction model, one can predict the extent of liver damage irrespective of age and either perform a KPE, suggest KPE, or suggest liver transplantation. With the results of this study, it is possible to carefully select children who will benefit from KPE and help to monitor these children post KPE. Since the numbers are only significant statistically, we intend to further validate the scoring system in a prospective trial.

## Recommendations

Based on the above findings of the study, the authors would like to recommend the following.

The risk prediction scoring system will be applied to all the children presenting with biliary atresia and based on the scores, and the following can be undertaken:

- If the score is between 3 and 7 – should perform a KPE
- If the score is between 8 and 16 – could recommend a KPE if the child is not fit for a liver transplant
- If the score is between 17 and 20 – should recommend a liver transplantation.

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## Conflicts of interest

There are no conflicts of interest.

## REFERENCES:

1. Davenport M. Biliary atresia: Outcome and management. *Indian J Pediatr* 2006;73:825-8.
2. Wildhaber BE. Biliary atresia: 50 years after the first Kasai. *ISRN Surg* 2012;2012:9-24.
3. Shneider BL, Mazariegos GV. Biliary atresia: A transplant perspective. *Liver Transpl* 2007;13:1482-95.
4. Sinha CK, Davenport M. Biliary atresia. *J Indian Assoc Pediatr Surg* 2008;13:49-56.
5. Redkar R, Karkera PJ, Raj V, Bangar A, Hathiramani V, Krishnan J. Outcome of biliary atresia after Kasai's portoenterostomy: A 15-year experience. *Indian Pediatr* 2017;54:291-4.
6. Goodman ZD. Grading and staging systems for inflammation and fibrosis in chronic liver diseases. *J Hepatol* 2007;47:598-607.
7. Zani A, Quaglia A, Hadzić N, Zuckerman M, Davenport M. Cytomegalovirus-associated biliary atresia: An aetiological and prognostic subgroup. *J Pediatr Surg* 2015;50:1739-45.
8. Schoen BT, Lee H, Sullivan K, Ricketts RR. The Kasai portoenterostomy: When is it too late? *J Pediatr Surg* 2001;36:97-9.
9. Davenport M, Gonde C, Narayanaswamy B, Mieli-Vergani G, Tredger JM. Soluble adhesion molecule profiling in preoperative infants with biliary atresia. *J Pediatr Surg* 2005;40:1464-9.
10. Langenburg SE, Poulik J, Goresky M, Klein AA, Klein MD. Bile duct size does not predict success of portoenterostomy for biliary atresia. *J Pediatr Surg* 2000;35:1006-7.
11. Weerasooriya VS, White FV, Shepherd RW. Hepatic fibrosis and survival in biliary atresia. *J Pediatr* 2004;144:123-5.
12. Govindarajan KK. Biliary atresia: Current trends in outcome and management. *Indian Pediatr* 2017;54:277.
13. Thapa BR, Walia A. Liver function tests and their interpretation. *Indian J Pediatr* 2007;74:663-71.
14. Wai CT, Greenson JK, Fontana RJ, Kalbfleisch JD, Marrero JA, Conjeevaram HS, et al. A simple noninvasive index can predict both significant fibrosis and cirrhosis in patients with chronic hepatitis C. *Hepatology* 2003;38:518-26.
15. Lin ZH, Xin YN, Dong QJ, Wang Q, Jiang XJ, Zhan SH, et al. Performance of the aspartate aminotransferase-to-platelet ratio index for the staging of hepatitis C-related fibrosis: An updated meta-analysis. *Hepatology* 2011;53:726-36.
16. Yang LY, Fu J, Peng XF, Pang SY, Gao KK, Chen ZR, et al. Validation of aspartate aminotransferase to platelet ratio for diagnosis of liver fibrosis and prediction of postoperative prognosis in infants with biliary atresia. *World J Gastroenterol* 2015;21:5893-900.
17. Grieve A, Makin E, Davenport M. Aspartate Aminotransferase-to-Platelet ratio index (APRI) in infants with biliary atresia: Prognostic value at presentation. *J Pediatr Surg* 2013;48:789-95.
18. Leung DH. Hepatic fibrosis scores and serum biomarkers in pediatric hepatology. *Clin Liver Dis (Hoboken)* 2017;9:125-30.
19. Shteyer E, Ramm GA, Xu C, White FV, Shepherd RW. Outcome after portoenterostomy in biliary atresia: Pivotal role of degree of liver fibrosis and intensity of stellate cell activation. *J Pediatr Gastroenterol Nutr* 2006;42:93-9.
20. Shah I, Madgum N. Correlation of APRI index with Metavir index in children with neonatal cholestasis without biliary atresia. *Ann Hepatol* 2018;17:592-5.
21. Zhang S, Wu Y, Liu Z, Tao Q, Huang J, Yang W. Hepatic pathology of biliary atresia: A new comprehensive evaluation method using liver biopsy. *Turk J Gastroenterol* 2016;27:257-63.
22. Fischler B, Ehrnst A, Forsgren M, Orvell C, Nemeth A. The viral association of neonatal cholestasis in Sweden: A possible link between cytomegalovirus infection and extrahepatic biliary atresia. *J Pediatr Gastroenterol Nutr* 1998;27:57-64.
23. Domiati-Saad R, Dawson DB, Margraf LR, Finegold MJ, Weinberg AG, Rogers BB. Cytomegalovirus and human herpesvirus 6, but not human papillomavirus, are present in neonatal giant cell hepatitis and extrahepatic biliary atresia. *Pediatr Dev Pathol* 2000;3:367-73.
24. Jevon GP, Dimmick JE. Biliary atresia and cytomegalovirus infection: A DNA study. *Pediatr Dev Pathol* 1999;2:11-4.
25. Liu C, Chiu JH, Chin T, Wang LS, Tai CH, Li AF, et al. Expression of aminopeptidase N in bile canaliculi: A predictor of clinical outcome in biliary atresia and a potential tool to implicate the mechanism of biliary atresia. *J Surg Res* 2001;100:76-83.





## CASE REPORT VI: NEPHROLOGY

### Disproportionate BUN to S. Creatinine ratio

**Dr Hemant Mehta, MD, DM, DNB, Consultant Nephrologist**

**Dr Pallavi Tanpure, MD, Nephrologist**

**Dr Jayant Barve, MD, Consultant Gastroenterologist**

**Dr Paresh Carry, MS, DNB, Consultant GI Surgery**

**Dr Vasant Navelkar, MD, Consultant Internal Medicine**

**Dr Prakash Jiandani, MD, Consultant Emergency Medicine**

**Dr Sanjeev Mehta, MD, FCCP, FAPSR, Consultant Chest Medicine**

#### Introduction:

Blood urea nitrogen (BUN) measures the waste product of protein metabolism. Reference values for BUN is normally 5-20 mg/dl, men may have slightly higher values than women, whereas elderly may have slightly increased values due to lack of renal concentration. In pregnancy, the values decrease by about 25% due to increased plasma volume and the newborns have values slightly lower than adult ranges. More than 99% of urea is synthesized in the liver. Its primary source is dietary protein. The amount of urea produced varies with substrate delivery to the liver and the adequacy of liver function.

Severe liver failure causes a reduction of urea in the blood. A patient who is severely dehydrated have a high BUN due to the lack of fluid volume to excrete waste products, and over hydration causes a decreased BUN. Because urea is an end product of protein metabolism, a diet high in protein, such as high-protein tube feeding, may also cause the BUN to increase. Extensive bleeding into the gastrointestinal (GI) tract will also cause an elevated BUN because digested blood is a source of urea.

The normal serum creatinine (S. Cr) is a waste product from the normal wear and tear of muscles and varies with the body muscle mass and with the technique used to measure it. For the adult male, the normal range is 0.6 to 1.2 mg/dl by the kinetic or enzymatic method, and 0.8 to 1.5 mg/dl by the older manual Jaffe reaction. For the adult female, with her generally lower muscle mass, the normal range is 0.5 to 0.9 or 1.1 mg/dl by the enzymatic method. Unlike urea, creatinine is largely unaffected by gastrointestinal bleeding or by catabolic factors such as fever and steroids. Ingestion of cooked meat can raise the S. Cr because cooking converts the creatine in meat (or muscles) to creatinine.

The BUN and S. Cr are screening tests of renal function. The ratio of BUN to creatinine (BCR) is usually between 10:1 and 20:1. It greater than 20:1 in pre-renal disease due to the increase in the passive reabsorption of urea that follows the enhanced proximal reabsorption of sodium and water. Thus, a high BCR ratio is suggestive of pre-renal disease as long as some other cause of a high ratio is not present. The BCR can exceed 20:1 when loss of muscle mass in a chronically ill or older patient lowers creatinine production and, therefore, the S. Cr. concentration, independent of the glomerular filtration rate (GFR).

Besides these, there are certain situations where there is increased BCR, as follows: (Can be remembered with mnemonic: Drivers Can use GPS)

- dehydration/pre-renal failure
- corticosteroids
- GI hemorrhage
- protein-rich diet
- severe catabolic state

Similarly, the causes of decreased BCR can be (Pneumonic: I am a SIMPLE SR):

- severe liver dysfunction
- intrinsic renal damage
- malnutrition
- pregnancy
- low protein diet
- SIADH
- rhabdomyolysis (or muscle injury, like myonecrosis, crush injury etc.)

Recently we encountered two clinical situations, one in which there was increased BCR due to elevated BUN, and another, where S. Cr was elevated in the presence of normal BUN. These are described below:

### **Case 1: BUN value >>> S. Cr**

85 years old female was admitted in the Covid ICU with h/o hematemesis. She had been otherwise healthy without any co-morbidities. She had no previous laboratory values available. When seen by us, she was conscious but had irrelevant talking, she was dehydrated but hemodynamically stable. The laboratory values showed Hb of 6.5 gm/dl and BUN of 100 mg/dl, with S. Cr of 1 mg/dl. She was transfused blood and given IV fluids and the BUN values settled down to normal over next 5 days, with improvement in neurological status; she was discharged to home by day 7.

### **Case 2: Normal BUN with >>> S. Cr**

57 years old male was transferred from another hospital with a diagnosis of massive necrotizing myofascitis of abdominal wall after several debridement and drainage. There was a previous history of untreated anal fistula with purulent discharge with an external opening at 7 O'clock position for the past 4 years. He was then confirmed to have a high anal fistula with spreading perianal, supralelevator, pelvic and abdominal wall (extra-peritoneal) collections on CT and MR imaging, with high suspicion for severe sepsis. He then underwent multiple debridement and drainage of the soft tissue infection, both from abdominal and perianal approaches. Simultaneously, a Seton (draining thread) insertion of the perianal fistulous tract was also done. Multiple broad spectrum intravenous antibiotics according to bacteriological studies were given and negative pressure wound therapy (VAC dressings) were employed to control severe infection and expedite healing. In spite of showing good recovery after 3 weeks with respect to sepsis control and healing, he developed high grade fever, tachycardia, leukocytosis, oliguria, cardiorespiratory failure and acute kidney injury (AKI), which was notable for: a) it was noted 3 days after starting Inj Colistin (Polymyxin E, PE) and b) the initial values showed BUN of 15 mg/dl with S. Cr of 3.79 mg/dl; the next day values were BUN/S. Cr of 17/4.9 mg/dl. C) Urine output was good. Nephrology opinion was sought, the AKI progressed and patient required hemodialysis, and multiorgan failure support.

Although initial imaging was inconclusive, eventually MRI revealed an organized collection (8 X 5 cm) in the region of the right obturator internus muscle suggestive of a deep seated abscess. He was then operated through a perianal approach which identified the lesion, it was adequately debrided and drained. He made a slow and steady recovery with complete withdrawal of multiorgan supports and was discharged uneventfully with normal diet and improving renal function.

### **Discussion: Two cases of altered BCR are presented.**

First one, in which BUN was disproportionately higher than S. Cr. The cause was upper GI bleed. BUN represents the terminal products of protein metabolism via ammonia. When upper GI bleeding occurs, the blood is digested to protein metabolized to BUN in the urea cycle within the liver. Higher BUN values are therefore associated with the digestion of blood. A hemorrhage of one liter of blood into the GI tract may elevate the BUN up to 40mg/ml. (1). High BCR can help differentiate the site of bleeding to some extent. Considering the relatively proper specificity and positive predictive value of BCR, in cases that bleeding source cannot be determined using other non-invasive methods, values higher than 35 can predict upper GI bleeding with high probability. However, due to the low sensitivity, values less than 35 are not diagnostic (2).

BUN rise within 24 hours of admission independently predicts the composite outcome, regardless of underlying renal disease status. It is speculated that the increase in BUN reflects inadequate volume resuscitation with pre-renal azotemia or evidence of ongoing bleeding (3).

In the second case, S. Cr was high with normal BUN. This challenging case had life threatening septic complication of anal fistula and further development of AKI. There was a diagnostic dilemma as to the cause of AKI. Was it due to sepsis, antibiotics (especially PE) or some other cause, and whether Inj PE should be stopped?

AKI due to sepsis occurs in almost 40-50% of patients and increases mortality 6-8 folds (4). AKI is multifaceted and several, concurrent mechanisms may be at play, besides hypo perfusion. These include inflammation, profound, heterogeneous distortion of peritubular and glomerular micro vascular flow, and the tubular epithelial cell (TEC) metabolic response to injury. Early sepsis-induced AKI may be the clinical and biochemical manifestation of the survival response strategy tubular cells trigger in this context.

Nephrotoxicity is a known major adverse event of PE (5) due to the d-amino butyric and fatty acid components of the drug, which increase tubular cell permeability, resulting in anionic, cationic, and water influx causing cell swelling and lysis. The result is acute tubular necrosis, which typically occurs after 5-7 days of PE therapy. According to the clinical practice guidelines from Kidney Disease: Improving Global Outcomes (KDIGO), nephrotoxicity, in patients with normal renal function (serum creatinine of 1.3 mg/dl in women and 1.5 mg/dl in men), is apparent when one of the following are fulfilled: (i) increase in serum creatinine by  $\geq 0.3$  mg/dl within 48 h, (ii) increase in serum creatinine to  $\geq 1.5$  times baseline, which is known or presumed to have occurred within the prior 7 days, and/or (iii) urine volume  $< 0.5$  mL/kg/h for 6 hours.

Our patient had normal BUN with elevated S. Cr value and hence sepsis and Inj. PE induced AKI were unlikely causes, as both would cause AKI with elevation in both BUN and S. Cr values. Hence it was advised to continue Inj PE.

The cause of AKI with normal BUN and elevated S. Cr value was attributed to muscle injury / muscle necrosis, the site of the same was not known at that time. This presumed muscle necrosis diagnosis as the cause of AKI was persisted with and the source of the same was sought and ultimately discovered. It was found to be an organized right obturator muscle myonecrosis and hematoma but no evidence of obvious suppuration. Myonecrosis had occurred due to adjacent infection at a very deep pelvic location (obturator internus muscle), the muscle injury was the suspected cause of AKI, this was proven and its treatment resulted in patient recovery. The tissue didn't grow any organisms and showed myonecrosis with intramuscular hematoma on histology.

**Conclusion:**

We have presented two cases of disproportionate BCRs, encountered at Lilavati Hospital in recent past. A careful history, clinical examination and appropriate analysis of relevant events are necessary to arrive at proper conclusion as to determine the cause of altered BCR and appropriate investigations can get the correct diagnosis.

**REFERENCES:**

1. Blood Urea Nitrogen (BUN). <https://www.rnceus.com/renal/renalbun.html>
2. Zia Ziabari SM, Rimaz S, Shafaghi A, Shakiba M, Pourkazemi Z, Karimzadeh E, Amoukhteh M. Blood Urea Nitrogen to Creatinine ratio in Differentiation of Upper and Lower Gastrointestinal Bleedings; a Diagnostic Accuracy Study. Arch Acad Emerg Med. 2019; 2;7(1) e30. PMID: 31432040; PMCID: PMC6637801.
3. Kumar NL, Claggett BL, Cohen AJ, Naylor J, Saltzman JR. Association between an increase in blood urea nitrogen at 24 hours and worse outcomes in acute nonvariceal upper GI bleeding. Gastrointest Endosc. 2017; 86(6):1022-1027.e1. doi: 10.1016/j.gie.2017.03.1533. Epub 2017 Apr 2. PMID: 28377105
4. Gómez H, Kellum JA. Sepsis-induced acute kidney injury. Curr Opin Crit Care. 2016; 22(6):546-553. doi: 10.1097/MCC.0000000000000356. PMID: 27661757; PMCID: PMC5654474.
5. Ozkan G, Ulusoy S, Orem A, Alkanat M, Mungan S, Yulug E, Yucesan FB. How does colistin-induced nephropathy develop and can it be treated? Antimicrob Agents Chemother. 2013; 57(8):3463-9. doi: 10.1128/AAC.00343-13. Epub 2013 Apr 29. PMID: 23629704; PMCID: PMC3719728.



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## List of Publications (International)

S. No.	Author	Title of the Paper / Chapter	Name of Journal
1	Dr. Sanzgiri P Dr. Shingare V Dr. Reddy CKV Dr. Smriti BK Dr. Jagannath P Dr. Suratkal V	Evaluation and Management of Cardiotoxicity Induced By 5-FU in GI-Cancer Patients with Low Cardiac Risk Score: An Observational Study	Journal of Cardiology and Cardiovascular Research Volume 3 Issue 1 Published: 10 October, 2021
2	Dr. P. Krishnappa Dr. A. Tripathi Dr. R. Shah	Surgical outcomes and patient satisfaction with the low cost semi rigid Shah penile prosthesis: a boon to the developing world	Journal of Sexual Medicine, August 2021

## List of Publications (National)

S. No.	Author	Title of the Paper / Chapter	Month of Publication
1	Dr. Salil Mehta Dr. Vasant Nagvekar Dr. G. Gupta	Utility of 2-deoxy-2-(18F) fluoro-Dglucose positron emission tomography scan in the systemic evaluation of patients with post-covid 19 endogenous presumed fungal endophthalmitis.	The Indian Journal of Ophthalmology in Oct 2021



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## Straight from the Heart - Patient Testimonials

*I would like to express my sincere gratitude, appreciation and respect to Dr. Atul Goel Sir, Dr. Abhinav Sir, Dr. Apoorva Sir, Dr. Ashutosh Sir, Dr. Achal Sir and other members of neurosurgery department at Lilavati Hospital and Research Centre, Mumbai. Their dedicated efforts have given me new life. I was completely bedridden in March 2021 and struggling to survive. Now I am able to walk again and my health has been improving ever since my surgery. I owe my life to you all. Thank you for accomplishing what seemed impossible.*

**Bhushan Mehar**

*I like the appointment process, doctor's consultation & excellance in their specialities, helpfull staff & support given by them. Thank you.*

**Priyatam Hazare**

*All the nursing and housekeeping staff on the 7th floor are to be commended for the manner in which covid patients were nursed back to health. It's an honor to have nurses who serve with complete dedication the way each nurse on the floor has done. Dr Sanjeev Mehta is a brilliant pulmonary specialist who took on a rare case and helped me heal in 5 days ! I am grateful to every single member of staff for looking after me like one of their own. Right from getting me sponged, ensuring food and medicines were taken on time - they were all nurturing beyond the call of duty.*

**Joy Vedamuthu**

*I like professionalism, expertise, behaviour of all staff attended to me. Overall servicess & facilities are really good.*

**Chirajneet Kumar**

*Excellent Team of doctors and at par Nursing care offered, I am very happy with the overall facilities.*

**Pradip Aroskar**

*Friendly approach by all the technicians and doctors, they made us feel comfortable.*

**Sangeeta Mohapatra**

*Courteous and good behaviour friendly atmostphere. All are best!*

**Yusuf Husain**





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Roshni Eye Bank

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## Benevolence

The social service wing of the hospital - SEWA serves to the health requirements of needy people. This department seeks to bridge the gap between the needy patients and the fast evolving medical technology. Various social activities such as free OPD, services to senior citizen, sending mobile vans to Adivasi areas to organize free health check-up camps, free camps are undertaken as an on-going process. The Roshni Eye Bank managed by Lilavati hospital is a well-equipped comprehensive centre for cornea removal, processing, storing, supplying and corneal transplantation.

*Under this service Lilavati Hospital & Research Centre offers:*

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2. Health Check up Camps at Nana Nani Parks
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<b>CT Scan Department</b>	<b>8044</b>
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<b>ENT / Audiometry</b>	<b>8232</b>
<b>Health Check-up Department</b>	<b>8354 / 8356</b>
<b>IVF</b>	<b>8226</b>
<b>Medical Social Worker (SEWA)</b>	<b>8361</b>
<b>MRD</b>	<b>8358 / 8359</b>
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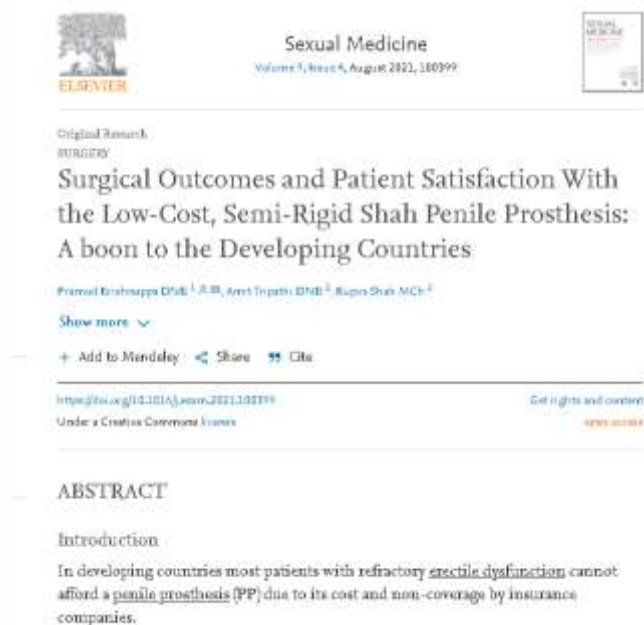


### BEST RESIDENT PAPER PRESENTATION

**Dr Shalini Saurabh** – Clinical Associate - Dept. of Histopathology was Awarded the “Best Resident Paper Presentation Award” at the Bombay Neurosciences Association, Monthly Clinical Virtual Program in July 2021

### PUBLICATION

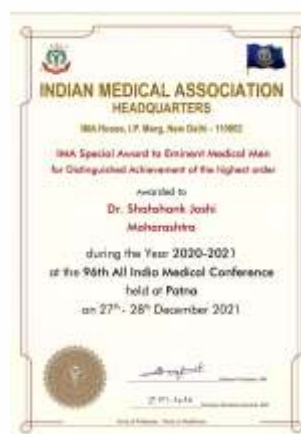
DNB Urology Ex-student **Dr. Amit Tripathi** has published his thesis in the Journal of Sexual Medicine, August 2021



### CERTIFICATE

COVIDSurg and GlobalSurg collaborators achieved the distinction of having the most authors - 15,025 on a single peer-reviewed academic paper. Consultants and Residents of Lilavati Hospital were part of this study.

Dept. of Pediatric Surgery  
Dept. of General Surgery  
Dept. of Orthopedic & Spine Surgery  
Dept. of CVTS



### CERTIFICATE

**Dr Shashank Joshi** was bestowed with IMA Special Award to Eminent Medical Men for Distinguished Achievement of the highest order by India Medical Association at the 96th All India Medical Conference held at Patna.

### Challenges in the Pandemic

Joseph Varon  
Paul Marik  
Jose Iglesias  
Christopher de Souza



Thieme

The book **Challenges in Pandemic** is edited and authored by our ENT Colleague **Dr Chris de Souza**. The book emphasizes the multidisciplinary nature of the pandemic and the need for all specialties to integrate with each other in order to treat and manage the terrible disease effectively and cure patients suffering from it. The book is also authored and edited by eminent deeply respected physicians from all over the world.

Clinical Associates and MUHS Fellows from Dept. of Histopathology presented paper and posters at the Annual Maharashtra Pathology conference (MAPCON 2021):

- a) **Dr Madhavi Musale (Ex-GIHPB fellow)** - paper on Lymph node yield in colorectal carcinoma resections - correlation with clinicopathological factors
- b) **Dr Anshika Rai (GIHPB fellow)** - poster on systematic review of a new & controversial diagnosis - MiNEN
- c) **Dr Shalini Saurabh (Clinical Associate)** - poster on Kikuchi necrotizing lymphadenitis in children.





**Dr. Rajeev Redkar** (Senior Consultant Paediatric Surgery) has been elected as The Chairman of the Maharashtra Chapter of Indian Association of Pediatric Surgeons



**Dr. Nandita Palshetkar** (Obstetrician & Gynaecology, LHRC) honoured with a **HONORIS CAUSA** at the Royal College of Obstetricians & Gynaecologists, London.

**Congratulations** to our doctors for being honoured and felicitated for **outstanding work during the COVID-19 pandemic** by His Excellency Shri Bhagat Singh Koshiyari, Governor of Maharashtra

**Prof. Dr. Vijay Bang** has been elected as President of The Cardiology Society of India.



**Lt. Gen. (Dr.) V. Ravishankar**  
COO and Consultant  
Cardiothoracic Surgeon



**Dr. Tushar Rege**  
Consultant Diabetic Foot Surgery



**Dr. Shashank Joshi**  
Consultant Diabetology &  
Endocrinology



**Dr. Abha Mahashur**  
Consultant Chest Medicine



**Dr. Jalil Parkar**  
Chest Physician



**Dr. Prahlad Prabhudesai**  
Chest Physician



At Indian Association of Pediatric Surgery Conference IAPSCON 2021, held on 19th Oct 2021, our DNB resident **Dr. Rahul Deo Sharma** won the **Best Short Paper Award** on : Gallbladder Duplication with Choledochal cyst- an unusual case of biliary anatomy

## Doctors Associated with Lilavati Hospital

### Andrology

Dr. Shah Rupin S.

### Anaesthesiology

Dr. Baxi Vaibhavi  
Dr. Budhakar Shashank  
Dr. Gandhi Nisha  
Dr. Gaiwal Sucheta  
Dr. Gawankar Prakash  
Dr. Kharwadkar Madhuri  
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Dr. Mahajan Anjula  
Dr. Mascarenhas Oswald  
Dr. Kothari Namrata  
Dr. Patil Prajakta  
Dr. Shah Falguni  
Dr. Waradkar Samidha

### Audiology & Speech Therapy

Mr. Bhan Satyan  
Ms. Gorawara Pooja  
Ms. Parulkar Bakul  
Ms. Satam Sneha (Cochlear)

### Bariatric Surgery

Dr. Palep Jaydeep  
Dr. Shah Shashank

### Blood Bank

Dr. Saraswat Shubhangi

### Cardiovascular & Thoracic Surgery

Dr. Bhamre Bipeenchandra  
Dr. Bhanushali Amol  
Dr. Bhattacharya S.  
Dr. Honnekeri Sandeep T.  
Dr. Iriraya Krishna Prasad  
Dr. Jaiswal O. H.  
Dr. Joshi Suresh  
Dr. Kumar Pavan  
Dr. Mehra Arun P.  
Dr. Nand Kumar  
Dr. Pandey Kaushal  
Dr. Rachmale G. N.  
Dr. Ravishankar V.  
Dr. Vichare Sanjeev

### Cardiology

Dr. Bajaj Harish  
Dr. Ballani Prakash  
Dr. Bang Vijay  
Dr. Dargad Ramesh R.  
Dr. Gokhale Nitin S.  
Dr. Jhala Darshan  
Dr. Kothari Snehal N.  
Dr. Lokhandwala Yash  
Dr. Mehan Vivek  
Dr. Merchant S. A.  
Dr. Menon Ajit R.  
Dr. Mehta Haresh G.  
Dr. Nabar Ashish  
Dr. Pillai M. G.  
Dr. Pinto Robin  
Dr. Punjabi Ashok H.

Dr. Rao Anand  
Dr. Rao Ravindra Singh  
Dr. Samuel K. Mathew  
Dr. Sanzgiri P. S.  
Dr. Shah Chetan  
Dr. Sheth Siddharth  
Dr. Suratkal Vidya  
Dr. Vijan Suresh  
Dr. Vyas Pradeep R.  
Dr. Vora Amit  
Dr. Vajifdar Bhavesh

### Chest Medicine

Dr. Chhajed Prashant  
Dr. Mahashur Abha  
Dr. Mehta Sanjeev K.  
Dr. Prabhudesai P. P.  
Dr. Parkar Jalil D.  
Dr. Rang Suresh V.

### Colorectal Surgery

Dr. Chulani H. L.

### Dentistry / Dental Surgery

Dr. Bhavsar Jaydeep P.  
Dr. Deshpande Dilip  
Dr. Gala Jigar  
Dr. Joshi P. D.  
Dr. Khatavkar Arun  
Dr. Kamdar Rajesh J.  
Dr. Parulkar Darshan  
Dr. Samath Shyamcharan  
Dr. Sanghvi Sameer

### Dermatology

Dr. Goyal Nilesh  
Dr. Mehta Nimesh  
Dr. Oberai Chetan  
Dr. Parasramani S. G.  
Dr. Pillai Jisha

### Diabetic Foot Surgery

Dr. Rege Tushar  
Dr. Vaidya Sanjay

### Diabetology

Dr. Panikar Vijay

### Diabetology & Endocrinology

Dr. Joshi Shashank R.  
Dr. Naik Vaishali

### Dietician

Dr. Pai Veena

### ENT

Dr. Dhingra Preeti  
Dr. D'souza Chris E.  
Dr. Jayashankar Narayan  
Dr. Parasram Kamal S.  
Dr. Pusalkar A.  
Dr. Shetty Adip (Cochlear Implant)

### Endocrine Surgery

Dr. Agrawal Ritesh

### Endo Urology

Dr. Utture Anand

### Gastro Intestinal Surgery

Dr. Bharucha Manoj  
Dr. Kulkarni D. R.  
Dr. Mehta Hitesh

Dr. Palep Jaydeep  
Dr. Shaikh Taher  
Dr. Varty Paresch  
Dr. Wagle Prasad K.  
Dr. Zaveri Jayesh P.

### Foot and Ankle

Dr. Kini Abhishek

### Gastroenterology

Dr. Barve Jayant S.  
Dr. Choksi Mehul  
Dr. Kanakia Raju R.  
Dr. Phadke Aniruddha Y.  
Dr. Parikh Samir S.  
Dr. Shah Saumil K.

### General Surgery

Dr. Khandelwal Nidhi  
Dr. Mehta Narendra  
Dr. Nikam Narendra  
Dr. Parikh Ratna  
Dr. Trivedi Narendra

### Gynaecology

Dr. Agarwal Rekha  
Dr. Chhabra Neelam  
Dr. Coelho Kiran S.  
Dr. Dudhedia Udhavraj  
Dr. Goyal Swarna  
Dr. Medhekar Mansi  
Dr. Nanavati Murari S.  
Dr. Pande Shinjini  
Dr. Pai Hrishikesh  
Dr. Pai Rishma D.  
Dr. Palshetkar Nandita  
Dr. Salunke Vivek  
Dr. Shah Cherry C.

### Haematology

Dr. Agarwal M. B.  
Dr. Bhavne Abhay

### Hair Restoration

Dr. Agrawal Sumit

### Headache & Migraine

Dr. Ravishankar K.

### Healthcheckup Consultant

Dr. Desai Sandeep

### Histopathology

Dr. George Asha Mary  
Dr. Tampi Chandralekha

### Infectious Diseases Consultant

Dr. Nagvekar Vasant C.

### Intensivist / Physician

Dr. Jiandani Prakash  
Dr. Kavita S.  
Dr. Shekade Kiran  
Dr. Shrinivasan R.  
Dr. Vas Conrad Rui

### Interventional Neuroradiology

Dr. Limaye Uday S.

### Interventional Radiology

Dr. Rai Jathin Krishna  
Dr. Sahu Amit  
Dr. Sheth Rahul  
Dr. Warawdekar Girish



### **Joint Replacement Surgery**

Dr. Maniar Rajesh N.

### **Lactation Consultants**

Dr. Joshi Mugdha

Ms. Temkar Swati

### **Liver Transplant**

Dr. Mehta Naimish

Dr. Shaikh Taher

### **Nephrology**

Dr. Mehta Hemant J.

Dr. Shah Arun

Dr. Suratkal L. H.

Dr. Upadhyaya Kirti L.

### **Neurology**

Dr. Chauhan Vinay

Dr. D'souza Cheryl

Dr. Deshpande Rajas

Dr. Sirsat Ashok M.

Dr. Soni Girishkumar

Dr. Vyas Ajay

### **Neuropsychology**

Ms. Panjwani Siddhika

### **Neuro Surgery**

Dr. Ambekar Sudheer

Dr. Andar Uday

Dr. Dange Nitin

Dr. Goel Atul

Dr. Parekh Harshad

Dr. Pawar Sumeet

Dr. Ramani P. S.

### **Nuclear Medicine**

Dr. Krishna B. A.

Dr. Shimpi Mahajan Madhuri

### **Oncology**

Dr. Lokeshwar Nilesh

Dr. Menon Mohanakrishnan

Dr. Parikh Bhavna

Dr. Smruti B. K.

### **Oncosurgery**

Dr. Bushan Kirti

Dr. Chabra Deepak

Dr. Chedda Yogen

Dr. Jagannath P.

Dr. Katna Rakesh

Dr. Mullerpatan Prashant

Dr. Parikh Deepak

Dr. Rao Satish

Dr. Sharma Sanjay

Dr. Shah Rajiv C.

Dr. Shetty Shravan S.

### **Ophthalmology**

Dr. Agrawal Vinay

Dr. D'souza Ryan

Dr. Mehta Salil

Dr. Mehta Himanshu

Dr. Nagvekar Sandeep S.

Dr. Parikh Rajul

Dr. Shah Manish

Dr. Shah Sneha

Dr. Vaidya Ashish R.

### **Orthopaedic Surgery**

Dr. Agrawal Pranav

Dr. Agrawal Vinod

Dr. Amyn Rajani

Dr. Archik Shreedhar

Dr. Bhandari Hemant

Dr. D'silva Domnic F.

Dr. Garude Sanjay

Dr. Gurav Suraj

Dr. Joshi Anant

Dr. Kasodekar Vaibhav

Dr. Kodkani Pranjal

Dr. Kohli Amit

Dr. Mukherjee Sunirmal

Dr. Nadkarni Dilip

Dr. Nazareth Ritesh

Dr. Padgaonkar Milind

Dr. Panchal Lalit

Dr. Pandey Alok Kumar

Dr. Panjwani Jawahar S.

Dr. Shetty Nagraj

Dr. Vatchha Sharookh P.

Dr. Vengsarkar Nirad

Dr. Warriar Sudhir

### **Pathology**

Dr. Chavan Nitin

Dr. Kamble Rahul

Dr. Mehta Kashvi

Dr. Natarajan Shripriya

Dr. Rangwalla Fatema

### **Paediatric Surgery**

Dr. Bangar Anant

Dr. Karmarkar Santosh J.

Dr. Nathani Rajesh

Dr. Redkar Rajeev G.

### **Paediatrics**

Dr. Chittal Ravindra

Dr. Gupta Priyam

Dr. Lokeshwar M. R.

Dr. Sharma Shobha

Dr. Ugra Deepak

### **Paediatric Cardiology**

Dr. Bhalgat Parag

### **Paediatric Critical Care/NICU**

Dr. Arya Manish Kumar

Dr. Sheikh Minhaj Ahmed

### **Paediatric Endocrinology**

Dr. Parikh Ruchi

### **Paediatric Hemato-Oncology**

Dr. Kanakia Swati

### **Paediatric Neurology**

Dr. Kulkarni Shilpa

Dr. Shah Krishnakumar N.

### **Paediatrics Nephrology**

Dr. Ali Uma

### **Paediatric Ophthalmology**

Dr. Doshi Ashish

### **Paediatric Orthopedics**

Dr. Aroojis Alaric

### **Paediatric Pulmonology**

Dr. Khosla Indu

### **Pain Medicine**

Dr. Baheti Dwarkadas

Dr. Jain Jitendra

### **Physicians / Internal Medicine**

Dr. Ballani A. G.

Dr. Bandukwala S. M.

Dr. Gidwani Vinod N.

Dr. Jadwani J. P.

Dr. Medhekar Tushar P.

Dr. Medhekar Amey T.

Dr. Nair C. C.

Dr. Shimpi Shrikant

### **Plastic & Reconstructive Surgery**

Dr. Barve Devayani

Dr. Dixit Varun

Dr. Jain Leena

Dr. Kumta Samir

Dr. Nehete Sushil

Dr. Prakash Siddharth

Dr. Purohit Shrirang

Dr. Wagh Milind

### **Psychiatry**

Dr. Deshmukh D. K.

Dr. Shah Bharat R.

Dr. Vahia Vihang N.

### **Psychology**

Ms. Chulani Varkha

### **Physician / Rheumatology**

Dr. Sangha Milan

### **Physiotherapy**

Ms. Garude Heena

### **Radiology & Imaging**

Dr. Deshmukh Manoj

Dr. Dhedia Khyati

Dr. Doshi Pankaj

Dr. Gupta Kanchan

Dr. Kamath Satish

Dr. Lokhande Kaustubh

Dr. Mehta Mona

Dr. Tyagi Neha

### **Rehab Medicine**

Ms. Shah Labdhi

### **Rheumatology**

Dr. Chitnis Neena

Dr. Gill Niharika

Dr. Sabnis Shailaja

### **Spine Surgery**

Dr. Bhojraj Shekhar

Dr. Chaddha Ram

Dr. Kundnani Vishal

Dr. Mohite Sheetal

Dr. Nagad Premik

Dr. Nene Abhay

Dr. Patel Priyank

Dr. Varma Raghuprasad

### **Urology**

Dr. Bhagat Suresh

Dr. Pahade Sachin

Dr. Pathak Hemant R.

Dr. Raina Shailesh

Dr. Raja Dilip

Dr. Sanghvi Nayan

Dr. Shah Sharad R.

Dr. Vaze Ajit M.

### **Urological Laparoscopy Surgery**

Dr. Ramani Anup

### **Urodynamics Consultant**

Dr. Dastur B. K.

### **Vascular Surgery**

Dr. Patel Pankaj

Dr. Pai Pares





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**THEWEEK**

**HANSA RESEARCH BEST HOSPITALS SURVEY 2021**

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