Billroth Hospitals

ACCIDENT & EMERGENCY MEDICINE/ CRITICAL CARE UNIT

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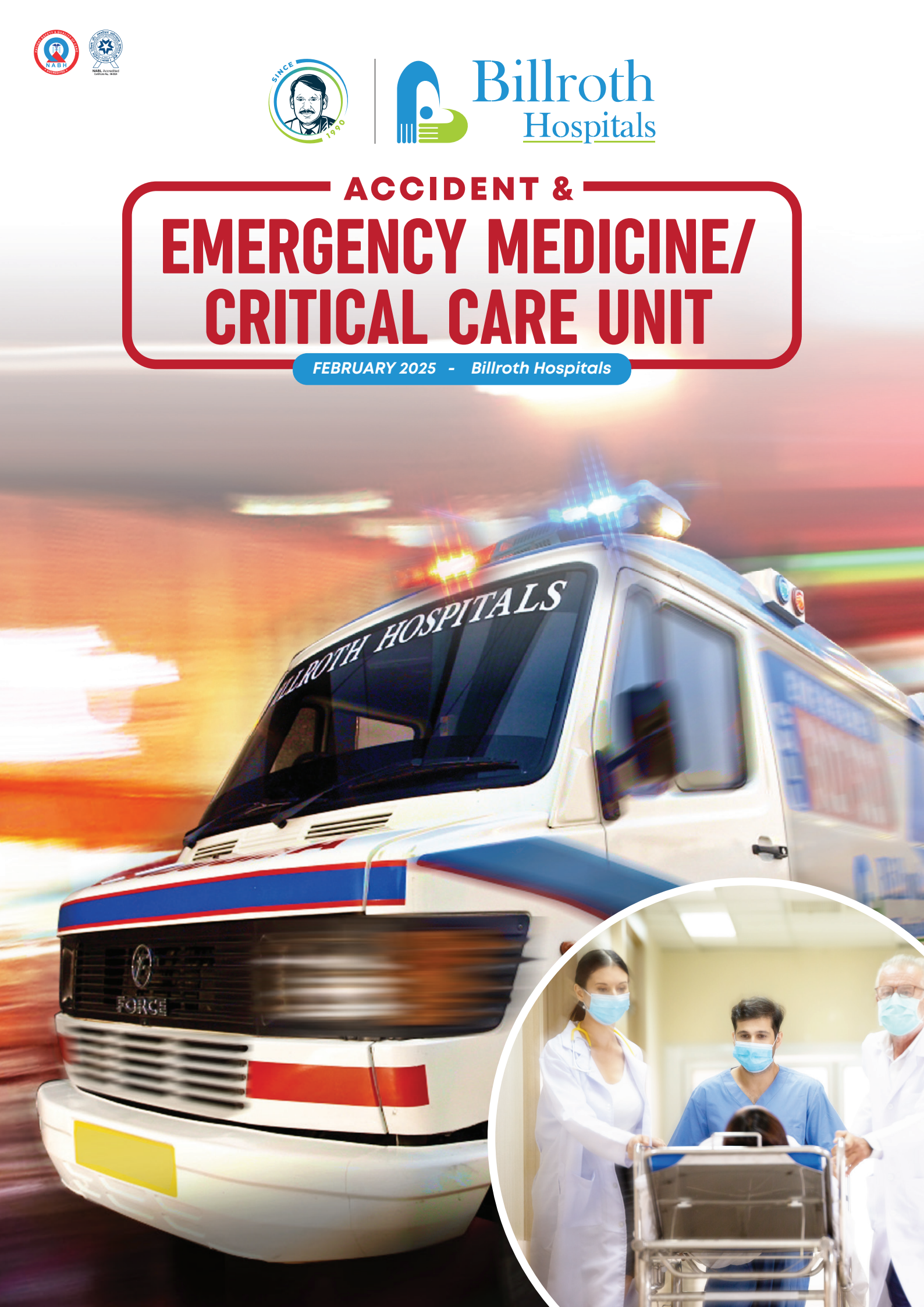

Billroth Hospitals

Dr. V. Jeganathan
Founder, Billroth Hospitals

"It has been said that a gentle word, a warm hand, a willing ear and small acts of kindness, often taken for granted, can change a life. We believe that to be true. Because we have seen first-hand the power of caring with compassion."

~ Dr. V. Jeganathan - Founder

MAN WITH VISION...

The Seed was Sown

An extraordinary physician of our times, **Dr. V. Jeganathan** watched thoughtfully as the first bricks for his dream hospital were laid. His vision for creating a world-class healing environment that would attract the best medical minds was taking shape. He dreamt of creating an institution that would serve as a beacon of hope to patients from across the world, offering them the highest standards of excellence in medical care, delivered with compassion. And so began a journey that started with a 70-bed hospital for Gastroenterology. Now Billroth Hospitals, offers an entire spectrum of Medical Care.

SINCE 1990, THERE WERE NO COMPROMISES AND NO LOOKING BACK AT BILLROTH HOSPITALS.

Where **Care** Comes **First**

CASE REPORT - 1

ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

INTRODUCTION

Chronic Obstructive Pulmonary Disease is characterized by airflow restriction brought on by structural alterations and airway narrowing due to prolonged exposure to harmful particles or gases (1). A poor prognosis decreased physical activity, and a higher frequency of exacerbations are linked to patients' steady reduction in lung function and ongoing respiratory symptoms, such as coughing, sputum production, or dyspnea (2). The development of COPD is extremely varied, influenced by both environmental variables and genetic predisposition. About 50% of smokers will acquire COPD at some point in their lives, making tobacco use the primary risk factor for the disease. Air pollution, nutritional variables, infections, age, genetic predisposition, and occupational exposure to hazardous gasses or biomass smoke are additional risk factors for COPD (3,4).

Acute exacerbation is the rapid worsening of respiratory and airway function in patients with COPD. Along with viral and environmental factors, bacterial infections are also responsible for AECOPD. Comorbidities such as cardiovascular disorders and heart disorders can also exacerbate AECOPD episodes. Bronchodilators, steroids, antibiotics, and extra oxygen support are all part of the treatment. For certain people, mucolytics, physical therapy, and airwayclearing equipment are also beneficial. Type-II respiratory failure, sometimes referred to as hypercapnic respiratory failure, arises when the respiratory system is unable to sufficiently eliminate carbon dioxide from the body (5).

CASE REPORT

A 67-year-old male with a 20-pack-year smoking history presented to the emergency department with worsening dyspnea, productive cough, and increased sputum purulence over three days. He reported reduced exercise tolerance and fatigue but denied fever or hemoptysis. His medical history included moderate COPD diagnosed three years prior, hypertension, and gastroesophageal reflux disease. He had been prescribed inhaled long-acting bronchodilators but admitted to inconsistent use. On examination, the patient showed signs of anxiety and tachypnea. Vital signs showed an oral temperature of 37.2°C, a respiratory rate of 28 breaths per minute, a heart rate of 105 beats per minute, a blood pressure of 145/85 mmHg, and an oxygen saturation of 89% on room air. Bilateral expiratory wheezes and reduced breath sounds in the lower lung areas were detected by auscultation.

The results of the initial tests, which included arterial blood gas analysis, were consistent with type 2 respiratory failure, with a pH of 7.35, partial pressure of carbon dioxide (PaCO2) of 48 mmHg, and partial pressure of oxygen (PaO2) of 62 mmHg. A full blood count showed slight leukocytosis and chest radiography ruled out pneumothorax and pneumonia. Haemophilus influenza was eventually found to be the causal pathogen through sputum culture. Due to acute sickness, spirometry was postponed; nevertheless, previous measures showed a projected FEV1 of 52% and a FEV1/FVC ratio of 55%.

MANAGEMENT

The patient was diagnosed with AECOPD likely precipitated by a bacterial respiratory infection. Management included:

1. **Oxygen Therapy:** Initiated to maintain SpO2 between 88-92%, preventing oxygeninduced hypercapnia.
2. **Bronchodilators:** Nebulized salbutamol (2.5 mg) and ipratropium bromide (500 µg) every four hours.
3. **Systemic Corticosteroids:** Intravenous methylprednisolone 40 mg daily for five days.
4. **Antibiotics:** Oral amoxicillin-clavulanate 875/125 mg twice daily for seven days based on sputum culture sensitivity.
5. **Noninvasive Ventilation (NIV):** Implemented due to persistent hypercapnia and respiratory acidosis.

Counseling on quitting smoking, chest physical therapy, and water were all supportive interventions. The patient was continuously watched for problems like heart attacks or respiratory failure.

OUTCOME AND FOLLOWUP

After 48 hours of NIV, the patient's dyspnea resolved and their blood gas values returned to normal, indicating a considerable improvement in their clinical condition. On day seven, he was released with a modified inhalation regimen that included an inhaled corticosteroid (ICS), a long-acting beta-agonist (LABA), and a tapered dose of oral prednisone. Two-week followup verified steady respiratory function, treatment compliance, and a dedication to quitting smoking.

DISCUSSION

The course of the disease, bacterial or viral infections, exposure to environmental irritants, and changes in ambient temperature can all contribute to the acute aggravation of the underlying chronic inflammation of the airways, which is reflected in AECOPD (6). The symptoms of AECOPD include hypoxemia with or without hypercapnia, irregular gas exchange, and significant airflow obstruction that increases breathing effort (7). The risk of death is increased when hypercapnia, which is frequently linked to severe exacerbations, occurs during respiratory failure. A higher rate of loss in lung function is correlated with the frequency of exacerbations (8).

Its prevalence and mortality rates are rising rapidly; the latest statistics indicate the prevalence of COPD in people aged ≥ 40. Acute exacerbation is the leading cause of hospitalization and mortality among COPD patients. Severe exacerbation is linked to a high risk of early mortality and a median survival of only 3.6 years (9). Other chronic illnesses like osteoporosis, cardiovascular disease, and metabolic syndrome are frequently associated with COPD. The problem is made worse by asymmetric septal hypertrophy, an abnormal increase in the thickness of the heart wall. Physical examinations may reveal increased expiratory wheezes. With AECOPD, the cough may worsen and the sputum volume may increase. During AECOPD, hemoptysis—which may involve blood mixed with purulent sputum—occurs often (10).

AECOPD is a major event in the natural history of COPD, often accelerating disease progression. This case underscores the importance of identifying and addressing exacerbation triggers, optimizing pharmacologic therapy, and implementing nonpharmacologic interventions like smoking cessation and pulmonary rehabilitation (11). The GOLD guidelines emphasize a stratified approach to AECOPD management, tailoring interventions to disease severity andexacerbation etiology (12).

CONCLUSION

This case demonstrates the need for a comprehensive strategy that includes early diagnosis, focused treatment, and preventative measures to manage AECOPD. In order to lower the frequency of exacerbations and enhance long-term results for patients with COPD, collaborative care approaches and patient education continue to be essential.

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CASE REPORT - 2
ACUTE CORONARY SYNDROME

A 42 years/Male, retrosternal chest pain radiating to left arm associated with palpitation, sweating, vomit, giddiness since morning. ECG showed Anterior wall myocardial infarction.Loading dose given. He was thrombolysed with Inj.Tenecteplase at ED . patient underwent coronary angiogram revealed double vessel disease, he underwent PTCA and stent to mid LAD using 2.5x40mm ORSIRO BIOTRONIK, procedure uneventful Patient is hemodynamically stable and being discharged.

CASE REPORT - 3
HYPOXIC ISCHEMIC ENCEPHALOPATHY/S/P CARDIAC ARREST (OUTSIDE)/ SEPTIC SHOCK/INFECTED LEFT FEMUR IMPLANT/DM/CAD

A 67 years/Male, known case of DM/CAD - TVD on medical management/ BA / Old PTB came to ER on 08/01/2025 with intubated from outside hospital.history of patient was planned for wound debridement for surgical site infection - s/p left femur implant removal re-plating on 14/11/2024. Post spinal anaesthesia patient went into cardiac arrest resuscitated, connected to ventilator.Neurophysician opinion obtained in view of low GCS and advised to do MRI brain and EEG. MRI brain was done which showed features suggestive of Hypoxic ischemic encephalopathy. EEG showed records within normal limits.percutaneous tracheostomy was done in view of prolonged ventilator support.CT pelvis was done which showed displaced fracture of proximal femoral shaft noted with bone fragment displaced medially, deformed bone with ill-defined proximal femoral shaft- likely secondary to chronic osteomyelitis.Patient was gradually weaned from ventilator support and shifted to ward. Patient`s clinical condition, nature of the disease and prognosis has been clearly explained in detail to patient attenders. Hence getting discharged with advice medicine.

CASE REPORT - 4
ACUTE NECROTIZING PANCREATITIS/ ANEMIA

80 years/Female, abdominal pain radiating to back, dyspnea, cough with sputum, vomiting. Patient is on RT feed @ Residence -10 day started after giving feed, NECT abdomen done showed Acute necrotising pancreatitis with resolution and collection, residual mild inflammation and collection of air under diaphragm. CT chest screening showed now onset nodular consolidation and ground glass attenuation nodules.On admission patient Hb - 6.3mg/dl . Hence 1 unit PRBC transfused. patient was hemodynamically stable hence discharged.

OVERVIEW

What is acute coronary syndrome?

Acute coronary syndrome (ACS) is a broad term for three types of coronary artery disease that affect millions of people each year. These potentially life-threatening conditions occur when a blockage causes blood flow to your heart to suddenly slow or stop.

People with ACS can experience unstable angina or a heart attack (myocardial infarction). Common signs include chest pain or pressure (angina), shortness of breath (dyspnea) or dizziness.

What types of heart conditions does ACS include?

Acute coronary syndrome involves three types of coronary artery disease that damage or destroy heart tissue. The specific type depends on:

- Where blood flow to your heart is blocked.
- How long the blockage lasts.
- The amount of damage it causes.

Types of ACS are:

- **Unstable angina:** This involves sudden, unexpected chest pain or pressure, even while resting. It's a warning sign of a heart attack and occurs when stable angina worsens.
- **Non-ST-elevation myocardial infarction:** An NSTEMI is a heart attack that providers can detect with blood tests but not with an electrocardiogram (EKG). It means your coronary arteries aren't fully blocked or were blocked for a short amount of time.
- **ST-elevation myocardial infarction:** A STEMI is a much more severe heart attack that providers can detect with blood tests and EKG. It occurs when blood flow to your heart is fully blocked for a long time, affecting a large part of your heart.Who gets acute coronary syndrome?

Acute coronary syndrome can affect anyone. However, certain risk factors raise the likelihood of developing ACS.

Age and lifestyle:

- Age (men who are over 45 years of age or women who completed menopause).
- Having overweight/obesity.
- Cocaine use.
- Lack of physical activity.
- Smoking.
- Unhealthy diet.

Conditions you have (or had) and family history:

- COVID-19.
- Diabetes.
- Family history of chest pain, heart disease or stroke.
- High blood cholesterol.
- High blood pressure (hypertension).
- High blood pressure, preeclampsia or diabetes during pregnancy.

CASE REPORT - 5

POST VIRAL SEQUEALE/PSEUDO THROMBOCYTOPENIA

22 years/Male,fever, fatigue, nausea since 2 weeks,initially evaluated in outside hospital, diagnosed as dengue fever with severe thrombocytopenia, In view of decreased platelet count,4 units of RDP and 1 unit of SDP (B positive) transfused with no adverse effect done in CCU, then shifted to ward., Patient condition improved hemodynamically stable and hence getting discharge

OVERVIEW

Pseudothrombocytopenia (PTCP) or spurious thrombocytopenia is an in-vitro sampling problem which may mislead the diagnosis towards the more critical condition of thrombocytopenia. The phenomenon may occur when the anticoagulant used while testing the blood sample causes clumping of platelets which mimics a low platelet count

Platelet satellitism

Platelet rosetting, or satelliting, around white blood cells can lead to undercounting by automated analyzers.[5]

Clotted samples

Coagulation within the sample leads to undercounting, because the analyzer samples the liquid part of the blood, while some of the platelets remain in the tube, trapped in the clot. Overfilling the sample, or inadequately mixing with anticoagulant, may allow small clots to form. Unlike platelet clumps, clots usually cannot be detected by reviewing the peripheral blood smear, but may be detected by probing with wooden sticks, including checking under the cap.[6]

Failsafes and avoiding false-positives

A pseudothrombocytopenia false-positive result may occur when automated platelet counting devices are used. As a means of double checking the results, the patient's blood sample is often examined under a microscope. If the clumping is visible and the number of platelets appears normal, pseudothrombocytopenia may be concluded. A second sample run with a different anticoagulant such as sodium citrate (blue top tube) to confirm the finding of pseudothrombocytopenia may be requested if there are doubts or concerns.

CASE REPORT - 6

ACUTE PULMONARY EDEMA/RECURRENT HYPOGLYCEMIA

72 years/Male, complaints of decreased response followed by uprolling of eye ball, bilateral lower limb swelling, abdominal distentionpresent.On examination in ER, on auscultation bilateral AE decreased in view of tachypnea patient started on BIPAP support and shifted to CCU.RWMA present, severe LV systolic dysfunction, aortic valve sclerosis, mitral regurgitation (Grade II), severe pulmonary arterial hypertension, right sided pleural effusion present, EF - 25%, In view of lower back pain orthopaedician surgeon opinion was sought and orders carried out. MRI lumbar spine showed wedge compression fracture of D10 vertebral body in its anterior 2/3rd with fluid cleft and mild patchy marrow oedema,CT chest showed large right pleural effusion with near complete collapse consolidation of right lung with ipsilateralmediastinal shift to the right side, mild left pleural effusion, severe cardiomegaly.patient taken up for pleural tapping, under aseptic condition right pleural fluid aspiration done under USG guidance, pigtail inserted .venous doppler study showed no evidence of DVT, Diffuse severe subcutaneous edema in bilateral lower limbs.He was on ventilator support, gradually weaned off.Pigtail catheter removed. patient was mobilized with taylor`s brace hence getting discharged.

CASE REPORT - 7

POSTERIOR CIRCULATION STROKE/CORTICAL VISUAL DISTURBANCE/ LEFT VERTEBRAL ARTERY STENOSIS 40-50%

Complaints of headache, blurring of vision, face numbness for 2 days.complaints of giddiness and blurred vision initially, vomiting, right side facial and left hand numbness.MRI brain done, shows Ill-defined patchy acute infarcts in bilateral cerebellar hemisphere (L>R), Bilateral occipital,

Parieto- occipital, Tempero- occipital cortical & sub cortical regions and right thalamus. A 6.5 x 4mm short segment acute eccentric thrombus in the intra cranial V4 segment of left vertebral artery with a linear 1cm long intraluminal hypointense area distal to the thrombosed segment and just proximal to the origin of PICA, probably dissection with eccentric thrombus. CT CAROTID/ CEREBRAL ANGIOGRAM done suggested proximal left vertebral artery with focal concentric asymmetric mixed calcified plaque in the V4 segment of Left Vertebral Artery causing 40-50% stenosis. Hypoplastic Right Vertebral Artery.Patient visual fields had a greater improvement and hence getting discharged with advice medications and follow up.

CASE REPORT - 8

RIGHT RECURRENT PNEUMOTHORAX /RIGHT LOWER LOBE CONSOLIDATION/COPD WITH EMPHYSEMA/S/P RIGHT ICD AND PLEURODESIS

43years/ Male right sided chest pain for the past 3 - 4 days associated with difficulty in breathing. CT chest scan done elsewhere showed gross hydropnuemothorax with passive atelectasis.Right ICD drain tube inserted.CT chest showed moderate right pneumothorax with ICD tube in-situ (post procedure) and complete collapse with consolidation of right lower lobe.Bronchoscopy done and BAL sent for further study. BAL culture showed growth of streptococcus pneumoniae and klebsiella species. Patient was treated with IV antibiotics, IV analgesics, Nebulisation and other supportive medications. Hence patient discharged.

CASE REPORT - 9

IDIOPATHIC THROMBOCYTOPENIC PURPURA

Leg swelling associated with left leg pain since 4 days,Intially patient went to Anand Hospital with platelet and FFP for decreased platelet count (34,000). Platelet count improved to 62,000 on next day. Which further decreased to (3000).Hence came here for further management.Left lower limb anterior venous doppler done shows no significant stenosis or occulsion in left lower limb anterior, no evidence of deep vein thrombosis, In view of severe thrombocytopenia ,Hematologist opinion obtained and orders carried out. In view of decreased platelet count 4 FFP transfused and 1 unit SDP transfused.platelet count improved gradually. Bone marrow biopsy done shows negative reactions. Patient was monitored regularly.Hence getting discharged.

CASE REPORT - 10

SYNCOPAL HEAD INJURY/RIGHT FRONTAL CONTUSION/LEFT SYLVIAN SAH/ SICK SINUS SYNDROME

TACHY- BRADY SYNDROME

56 years/Male,syncope and fall in his residence CT-brain shows right frontal contusion, left sylvian fissure SAH with no midline shift or mass effect .ECG showed atrial fibrillation.inview of atrial fibrillation, advised for Holter monitoring.Holter report showed significant sinus paused and frequent atrial fibrillation episodes suggestive of sick sinus syndrome.reviewed with Holter reports and advised for permanent pacemaker implantation.underwent permanent pacemaker implantation under local anaesthesia. Patient symptomatically felt better hence discharged.

OVERVIEW

Sick sinus syndrome, also known as sinus node dysfunction (SND), is a disorder of the sinoatrial (SA) node caused by impaired pacemaker function and impulse transmission producing a constellation of abnormal rhythms. These include atrial bradyarrhythmias, atrial tachyarrhythmias and, sometimes, bradycardia alternating with tachycardia often referred to as "tachy-brady syndrome." These arrhythmias may result in palpitations and tissue under-perfusion leading to fatigue, lightheadedness, pre-syncope, and syncope.

CASE REPORT - 11

FRACTURE RADIAL HEAD WITH LEFT ELBOW DISLOCATION

26 years/Male, history of slip and fall while playing with friends in Agricultural land, sustained injury over left elbow tenderness and contusion present. CT left elbow report showed posterior dislocation of the elbow joint and a displaced fracture of the radial head with medial and inferior displacement of the fracture fragment. patient underwent open reduction of elbow dislocation. Patient underwent ORIF left radial head with Herberts screw. Pre and post operative procedure uneventful.Hence being discharged.

CASE REPORT - 12

RTA –HEAD INJURY –BILATERAL BASIFRONTAL CONTUSION/HYPONATREMIA

30years/ Female,complaints of vomiting and history of giddiness associated with complaints of headache for past 2 days, similar episodes 2 weeks back. serum electrolytes done on 31.01.2025 showed (serum sodium- 120) was started on IV 3% sodium chloride continuous infusion and encourages normal diet with extra salt. CT brain showed near complete resolution of the hemorrhagic contusion in bilateral basifrontal region. Repeat serum electrolytes done on 02.02.2025 showed (Serum sodium + -141).hence discharged.

OVERVIEW

Symptoms of hyponatremia (low sodium) can come on suddenly or slowly, over time.

What is hyponatremia?

Hyponatremia is when your blood sodium (salt) level is lower than it should be. Healthcare providers consider sodium (Na+) levels below 135 milliequivalents/liter (mEq/L) to be lower than normal.

It might seem like very little salt would be a good thing. But like many things in life, it's all about balance. You need the right balance of water and sodium in your body to stay healthy. They can become unbalanced due to medical conditions or medications. When this happens, water moves into your tissues, causing them not to work properly. This can be especially dangerous in your brain.

If your kidneys are working as they should, drinking too much water almost never causes your blood sodium to fall too low unless you're very dehydrated (like after running a marathon) and you don't replace electrolytes (important minerals that keep your body working) at the same time.
Hyponatremia can be chronic or acute. Chronic hyponatremia can happen over a long time, with symptoms coming on slowly and your body making adjustments for the low sodium levels. With acute hyponatremia, blood sodium levels fall quickly, and your symptoms may become severe suddenly.

What are the types of hyponatremia?

Types of hyponatremia include:

- Euvolemic hyponatremia/dilutional hyponatremia. This is when the amount of sodium in your body stays the same but the amount of water in your body increases.
- Hypervolemic hyponatremia. This is when the amount of water and sodium in your body both increase, but the amount of water increases more.
- Hypovolemic hyponatremia. This is when the amount of water and sodium in your body both decrease, but the amount of sodium decreases more.

CASE REPORT - 13

HYPOGLYCEMIC SEIZURES/ DM/NECTROTIZING FASCIITIS LEFT FOOT

complaint of seizure jerky moment (4 limb) at around 4.15am followed up-rolling of eye presents, decreased urine output, complaints of chest discomfort, generalized myalgia.NECT brainrevealed small ischemic vessels .CT angiogram of lower limb vessels ,revealed secure long segment left superficial femoral artery. Left popiteal and bilateral mild moderate below knee diseases. Doppler left lower limb arteries revealed chronic thrombotic occlusion of superficial temporal artery. Patient underwent left SFA+ popliteal angioplasty .The event went uneventul and was planned for left mid foot amputation under spinal on 10.01.25. she was managed with antibiotics and other medications.hence discharged.

CASE REPORT - 14

OPEN FRACTURE MIDDLE PHALANX LEFT INDEX FINGER/ EXTENSOR TENDON INJURY ZONE 2

aged 43 years / Male, came with alleged history of accidental cut injury to right 2nd, 3rd fingers and abrasion to 4th finger while working with a cutting machine at his residence. Xray finger shows fracture middle phalanx normal index finger.Patient underwent debridement / K wire fixation middle phalanx left index finger + Tendon repair under general anesthesia + Nerve block .Post operative period uneventful. Hence getting discharged following advice and follow up.

OVERVIEW

Extensor tendon lacerations of the hand are common injuries that can lead to significant functional impairment if not properly managed. These injuries often occur in working-age individuals, contributing to a notable economic burden. The severity of the injury depends on the location and extent of the tendon damage, and diagnosis is typically made through physical examination, provocative tests, and sometimes advanced imaging. Treatment may be nonoperative or surgical, depending on the extent of the functional deficit. Prompt and appropriate management is crucial to preserving hand function and preventing long-term disability.

CASE REPORT - 15

ACUTE IWMI (LYSED) WITH MILD LV DYSFUNCTION/ HYPOTHYROIDISM

42 years / Male, presented with history of Retrosternal - chest pain Yesterday morning) around 9am, followed by sweating and giddiness. ECG showed inferior wall myocardial infarction. He was thrombolysed Inj. Tenecteplase, loading dose with heparin given. 2D - ECHO was done. Patient underwent coronary angiogram through right radial artery revealed occluded proximal RCA, advised for PCI to RCA.Procedure uneventful discharged with stable hemodynamical status.

CASE REPORT - 16

EFFORT ANGINA CLASS 3/OLD IWMI/CAD-TVD/SHTN

69years / Male,Complaints of chest pain on exertion (NY Class III) sweating on and off since 1 week. ECG showed Q with T inversion in inferior leads and ST segment depression with T inversion in V3 - V6.ECHO showed RWMA present involving Mid and Apical Anteroseptum, Inferior, Basal anterior, LV Apex segments are Hypokinetic, Mild Concentric LVH, Dilated LV, Moderate LV Systolic Dysfunction (EF - 38%), Type II LV Diastolic Dysfunction.Patient was taken up for CAG on the same day which showed Triple Vessel Disease. Patient was given the option of CABG / PCI to LAD and LCX and the risk explained in view of renal dysfunction. However, patient opted for PCI to LAD and LCX. After adequate hydration, patient was taken up for PCI with informed consent. Patient underwent successful PCI to LAD with 2.75 x 24mm PROMUS PREMIER stent and LCX with 2.5 x 20mm PROMUS PREMIER stent. His renal function were monitored daily, hospital stay was uneventful. Patient is being discharged in a stable hemodynamic.

CASE REPORT - 17

THROMBOCYTOPENIA WITH VIRAL FEVER

29 Years/ Female, come with complaints of fever since 3 days, associated with generalised body pain, vomiting. Platelets 33000, 4 units of RDP Transfusion was done. Following that the patients condition improved and the repeated platelets. Count values are improving trend. He had one episode of involuntary movements of lower limb. MRI brain was normal. Patient's platelet count gradually improved to 84000.Hence patient discharged.

OVERVIEW

Several other infections can cause both a fever and low platelets. Some of the most common ones include the following.

MALARIA

Malaria is a mosquito-borne illness common in warm, wet climates around the world. It causes a high fever, chills, shaking, and gastrointestinal symptoms such as nausea, vomiting, and diarrhea.

Thrombocytopenia is common among people with malaria infections. The authors of a 2021 studyTrusted Source reviewed 73 confirmed cases of malaria from a hospital in Ethiopia and found that 79.5% of people with malaria also had some form of thrombocytopenia.

DENGUE

Dengue fever is another viral infection that mosquitoes commonly transmit. It's mostly prevalent in tropical regions and can cause a high fever, body aches, and a skin rash, among other symptoms.

Thrombocytopenia is a common complication of severe dengue that can sometimes also lead to bleeding in the nose, gums, or intestines.

LEPTOSPIROSIS

Leptospirosis is a virus common among animals such as dogs, rodents, and livestock. Humans can get it through contact with the urine of an infected animal.

Although leptospirosis often causes a mild, flu-like illness, symptoms may worsen over several weeks. Thrombocytopenia is more common with severe cases of leptospirosis.

TYPHOID

Typhoid fever is a bacterial infection that spreads through contaminated water and food. It causes a high fever along with weakness, pain, fatigue, and other symptoms.

Without treatment, typhoid fever can lead to severely low platelet levels, as well as life threatening symptoms such as intestinal bleeding and sepsis.

SEPTICEMIA

Septicemia is a severe blood-related complication of a bacterial infection in another part of your body, such as pneumonia or a urinary tract infection. It occurs when bacteria enter the bloodstream and start to spread rapidly, triggering widespread inflammation and sepsis.

Septicemia doesn't always cause low platelets. But when it does, it's often a sign of a less favorable outlook.

CASE REPORT - 18

CAD/ ISCHEMIC VT –REVERTED WITH CARDIOVERSION/CAG-TVD/DM

85 years/Male, presented with complaints of acute onset of retrosternal chest pain (Non radiating) associated with palpitations, Breathing difficulty x past 1 hour.In ER, he developed VT, which was Reverted with synchronised cardioversion 150 Joules, Nephrologist opinion sought for elevated renal parameter, Patient underwent coronary angiogram through right ulnar artery under LA which revealed distal left main with triple vessel disease, RCA RE-OCCLUDED. ICD was initially considered for VT episode, but in view of LM with TVD, RCA RE-OCCLUDED-Recommended for revascularization at a later date. If VT persists, EP study was suggested. Holter monitor showed transient VT, IV amiodarone infusion tappered and started on oral medications.Patient is hemodynamically stable and being discharged with advice medicine.

OVERVIEW

Ventricular tachycardia (VT) is a wide complex arrhythmia of ventricular origin, defined as three or more consecutive beats at a rate of more than 100 beats per minute. Sustained ventricular tachycardia is defined as tachycardia that continues for more than 30 seconds or leads to hemodynamic compromise within 30 seconds and requires intervention. On the other hand, non-sustained ventricular tachycardia lasts less than 30 seconds and does not cause hemodynamic instability. Ischemic heart disease is the most common cause of ventricular tachycardia, and VT has a wide range of clinical presentations, including palpitations, chest pain, shortness of breath, syncope, and cardiac arrest. This activity reviews the evaluation and management of ventricular tachycardia and highlights the importance of an interprofessional team in managing patients with ventricular tachycardia.

Ischemic heart disease is the most common cause of ventricular tachycardia, and 5 to 10% of patients with acute coronary syndrome are found to have ventricular arrhythmias. Ventricular tachycardia in acute coronary syndrome is usually polymorphic, while monomorphic ventricular tachycardia is a sign of a myocardial scar.Accelerated atrioventricular rhythm (AIVR) is a monomorphic ventricular tachycardia, referred to as a sign of successful reperfusion, and it has a strong association with infarct size.

Ventricular tachycardia is a major contributor to sudden cardiac death in patients with ischemic and non-ischemic cardiomyopathy. Ventricular tachycardia in cardiomyopathy is usually monomorphic due to scar-related reentry, and its degeneration into ventricular fibrillation may result in cardiac arrest or even sudden cardiac death. The clinical presentation of ventricular tachycardia varies from palpitation to sudden cardiac death. For appropriate management of VT and prevention of sudden cardiac death, it is essential to understand the pathophysiology of ventricular tachycardia and underlying structural heart disease. In this chapter, we summarize the etiology and epidemiology of ventricular tachycardia and discuss the evaluation and management of patients present with ventricular tachycardia.

CASE REPORT - 19
TAKAYASU ARTERITIS

19 years / Female came with history of giddiness with syncope aggravated on walking and sitting up since 5-6 months. Aortagram showed Takayasu arteritis Type- I, patient was started on IL-6 – inhibiting monoclonal antibody. Observation done hemodynamically stable, hence discharged.

OVERVIEW

Takayasu arteritis, aka pulseless disease, is a systemic inflammatory condition which leads to damage of the medium and large arteries and their branches. It occurs predominantly in young Asian women. It usually involves the aorta and its major branches, particularly the renal arteries, carotid arteries, and subclavian arteries, and leads to stenosis, occlusions, or aneurysmal degeneration of these large arteries. An abnormality in cell-mediated immunity seems to be its main pathogenesis, but its etiology is still largely unknown. Diagnosis is based on suspicion as well arteriographic findings. Treatment usually begins with medical management using corticosteroids; however, surgical management has become more common recently due to findings of an overall lack of disease regression and high rates of relapse with just medical management alone.

CASE REPORT - 20

COPD/CARDIOGENIC SHOCK/B/L PLEURAL EFFUSION/S/P LEFT PLEUROCENTESIS/ SEVERE PAH/ CONGESTIVE HEPATOMEGALY

62years/ Male dyspnea for past 2 months, presented with complaint of dyspnea for 2 month aggravated in last 10 days at rest.ECG Tachycardia, Echo Severe PAH, Normal LV dilated RA-> RV, chest X-ray shows right side pleural effusion, congestive hepatomegaly. CT Pulmonary Angiogram was done reports no pulmonary thromboembolism , presence of pulmonary arterial hypertension with dilated right heart with right ventricular strain and mild to moderate pericardial effusion, moderate bilateral pleural effusion more over left with sub-segmental atelectasis , extensive centrilobular emphysematous changes in both lung and presence of fibrotic strands in both upper lobe.In CCU left Pleurocentesis was done. In view of low GCS and increased PCO2 patient was intubated. Cardiologist &Pulmonologist opinion was obtained. Patient was strictly monitored in CCU.Patient GCS improved and extubation was done .Patient was on 6LO2 VM24% and overnight BIPAP. patient was gradually tappered from venturi mask to nasal prongs.hencedischarged.

CASE REPORT - 21

RIGHT CEREBELLAR METASTASIS MULTIPLE PULMONARY METASTASIS SYSTEMIC HYERTENSION CA CERVIX ON CHEMOTHERAPY S/P HYSTERECTOMY

63 years/Female, presented to emergency with history of altered sensorium and history of decreased response associated with complaints of vomiting and giddiness for 2 days. S/P EVD status, patient was intubated and referredhere.CT brain done showed non enhancing partially enhancing cyst lesion in left cerenellar with hydrocephalus with brain stem edema. CT chest showed multiple pulmonary metastasis.after getting well inform consent patient underwent right sub occipital craniectomy excision of tumor under general anaesthesia. Both intra and post operative events were uneventful. Repeat CT brain showed post operative changes. Patient was symptomatically better and neurologically stable.Hence discharged.

CASE REPORT - 22

ACUTE PORTAL VEIN THROMBOSIS CHRONIC EXTRA HEPATIC PORTAL VEIN THROMBOSIS PORTAL BILIOPATHY ACUTE PANCREATITIS – RECOVERING

43 years/Female, complaints of abdomen pain for 1 week, Radiation to bilateral loin region. increased pain while deep breathing. Consumed outside food.high coloured urine since 3 days, giddiness on & off.History of Ayurvedic medications intake for 1 month (for chest burn).USG abdomen was done, which revealed portal vein thrombosis with periportal pancreatic collateral, GB sludge. MRCP acute edematousinersitial pancreatitis.Portal biliopathy with pericholedochalvarices.Early portal biliopathy.OGD scopy and ERCP showed gastric antral vascular ectasia (GAVE). Prominent and bulging ampulla.ERCP was done, which revealed choledocholithiasis and CBD stone delivered. Biliary sphincterotomy followed by ballonsphincteroplasty done.Re-look endoscopy was done, biliary stent with full flow of bile done.Hence discharged.

CASE REPORT - 23

DISTAL ESOPHAGITIS/ HIATUS HERNIA/ EROSIVE GASTRITIS/ ALZHEMIER'S /BPH/NEUROGENIC BLADDER/S/P SIGMOIDOSCOPY -HEMORRHOIDS

84 years / Male, came with complaints of abdomen distention since 2 weeks duration associated with constipation (+), black tarry stool since 3 days duration. History of bilateral lower limb swelling (+), loss of appetite (+) since 20 days. NECT abdomen was done. UGI scopy done under MAC Sigmoidoscopyrevealed Anal Verge – Haemorrhoids.

Significally over distended urinary bladder with mass effect displacing the bowel loops as described.

Large sliding hiatus hernia

Fecal loaded colonic loops with suggestion of mild diffuse wall.

OVERVIEW

Esophagitis refers to inflammation or injury to the esophageal mucosa. One of the most common causes is gastroesophageal reflux, which can lead to erosive esophagitis. Other etiologies include radiation, infections, local injury caused by medications, pill esophagitis, and eosinophilic esophagitis (EoE). The most common symptoms in patients with esophagitis are chest pain, odynophagia, and dysphagia. Patients with EoE may present with food impaction. If the esophagitis is severe and leads to strictures, fistulization, and perforation, patients may present with symptoms related to those entities.

Multiple etiologies for esophagitis have been identified such as reflux esophagitis, Medication (Pills) induced esophagitis, infectious , eosinophilic and radiation esophagitis.

- Reflux or erosive esophagitis that occurs because of the reflux of gastric contents into the stomach leading to mucosal injury is one of the most common causes of esophagitis.
- Infection esophagitis can be caused by bacteria, viruses, fungal and parasitic microorganisms with the least common being bacteria and the most common being fungal.
- Pill induced esophagitis is most commonly associated with oral bisphosphonates like alendronate, some antibiotics like tetracycline, doxycycline, and clindamycin. NSAIDs, aspirin, ferrous sulfate, potassium chloride, and mexiletine has also been reported as a cause of pill-induced esophagitis.
- Eosinophilic esophagitis (EoE) is now thought to be a chronic immune antigen-related esophageal disease with almost similar symptoms of esophagitis but histologically have squamous mucosal inflammation caused by predominant eosinophilic infiltration.
- Radiation-induced esophagitis is associated with radiotherapy related toxicity and can present as both in acute and chronic forms.

CASE REPORT - 24
ACS – STEMI/ CAD -TVD

A 68 years male, came with chest pain and profuse sweating, diagnosed with ACS / AWMI – lysed with STK. CAG under LA through right radial artery access showed Left main Triple vessel disease, advice – Multi Vessel PCI. Post procedure patient developed acute pulmonary edema, patient was intubated and ventilated and shifted to CCU. On 08/01/2025 patient extubated, weaned off ventilator. Hemodynamically stable and comfortable. Hence discharged.

CASE REPORT - 25
SEVERE AECOPD WITH DECOMPENSATED TYPE 2 RESPIRATORY FAILURE/ COR PULMONALE/ ATRIAL FIBRILLATION WITH RVR/HYPONATREMIA/ IMPACTED RIGHT NECK OF FEMUR FRACTURE ? RECENTLY

A 91 years male came with shortness of breath associated with drowsiness with lower limb weakness since 2 days. H/o fall last week left knee swelling, altered sensorium, slurring of speech, limb weakness, general weakness. After pre dilatation patient developed flash pulmonary edema which was managed with NIV and parental diuretics. Angio showed TIMI III flow. In view of heavily calcific lesion and patient in acute pulmonary edema. Patient intubated and connected to mechanically ventilated, managed with NTG infusion, ABG showed severe respiratory acidosis with co2 narcosis. Followed by inotrophs support was tapered down and gradually weaned off ventilation and extubated on 11/10/24. 1 episode AF which was managed IV cordarone bolus. Patient hemodynamically stable.Hence discharged.

CASE REPORT - 26
TYPE 2 RESPIRATORY FAILURE, SECONDARY INFECTION

A 69 years male came with decreased response from yesterday evening. C/o breathing difficulty H/o decreased appetite, DM / HTN. Patient was immediatelt intubated due to reduced responsiveness associated with Co2 narcosis. TC, RFT elevated and started on IV antibiotics. Patient improved by 19/09/2024, hence extubated on 20/09/2024 and placed on BIPAP support. Have low uribe output. Treated with IV antibiotics / LMWH and Bipap support.

OVERVIEW

What is respiratory failure?
Respiratory failure is a condition where you don't have enough oxygen in the tissues in your body (hypoxia) or when you have too much carbon dioxide in your blood (hypercapnia). You might also hear people use the term "acute hypoxemic respiratory failure (AHRF)" to describe it.

What are the types of respiratory failure?
Respiratory failure can come on suddenly (acute) or over time (chronic). There are two common types: hypoxemic respiratory failure (type 1) and hypercapnic respiratory failure (type 2). Other types include perioperative (related to surgery) respiratory failure (type 3) and respiratory failure due to shock (type 4).

Hypoxemic respiratory failure
Hypoxemic respiratory failure happens when you don't have enough oxygen in your blood (hypoxemia). Heart and lung conditions are the most common causes. Hypoxemic respiratory failure is also called hypoxic respiratory failure

Hypercapnic respiratory failure
Hypercapnic respiratory failure happens when you have too much carbon dioxide (CO2) in your blood. If your body can't get rid of carbon dioxide, a waste product, there isn't room for your blood cells to carry oxygen.
The most common causes of hypercapnic respiratory failure include heart, lung, muscle and neurological (brain and spinal cord) conditions. Certain medications can also cause it. Hypercapnic respiratory failure is also called hypercarbic respiratory failure.

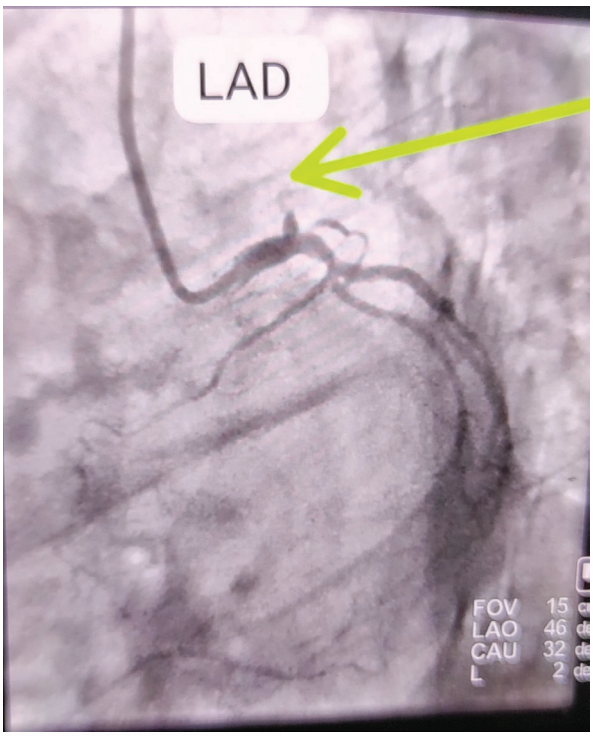
Perioperative respiratory failure
Perioperative respiratory failure can happen when you have surgery. Anesthesia (medication that keeps you asleep) can keep you from breathing properly. Sometimes, air sacs in your lungs can collapse (atelectasis) and keep oxygen from getting into your blood.

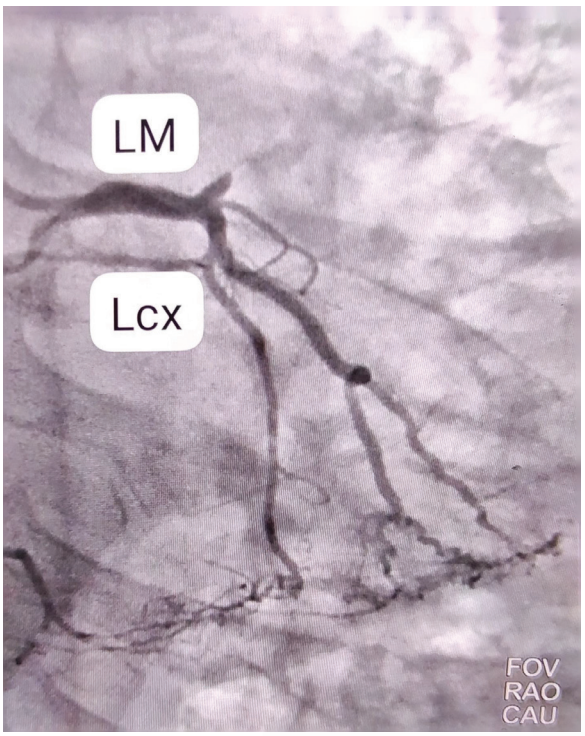
Respiratory failure due to shock
Shock is a condition that causes low blood pressure, fluid in your lungs (pulmonary edema) and other issues that can lead to respiratory failure. Sepsis, cardiac events (like a heart attack) and blood loss can cause shock.

CASE REPORT - 27

INTRA-AORTIC BALLOON PUMP (IABP): A LIFE-SAVING CARDIAC DEVICE (R.A.PURAM)

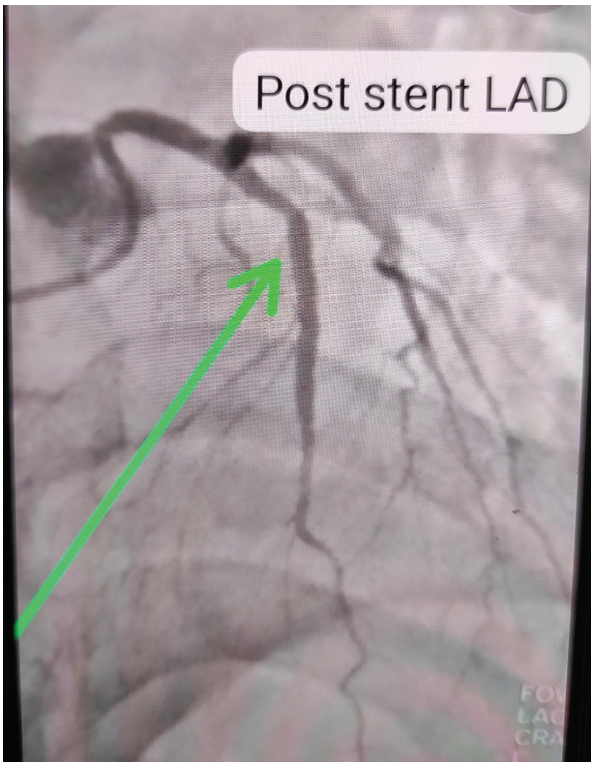
54 year make presented to the Emergency Room with severe chest pain raditing to the shoulder Patient is a known case of Uncontrolled diabetes, coronary Artery Disease, had PCI to RCA few years back and had stopped the antiplatelets . Trop I strongly positive. Creatinine was 1.32. Patient was immediately transferred to the cath lab after giving the loading dose antiplatelets. Coronary angiogram showed,





LAD – Ostial 100% occluded
LCX - Non dominant . Normal
RCA - Dominant
Proximal 100% occluded
Distal vessel filling through Hethocollatarls.

Patient underwent PCI to the culprit vessel, he had sudden bradycardia, pulmonary oedema, and cardiogenic shock.



He was transferred to ICU and started on multiple vasopressors, Diuretic and after CTVS opinion, IABP was inserted and started on 1:1 with full augmentation.

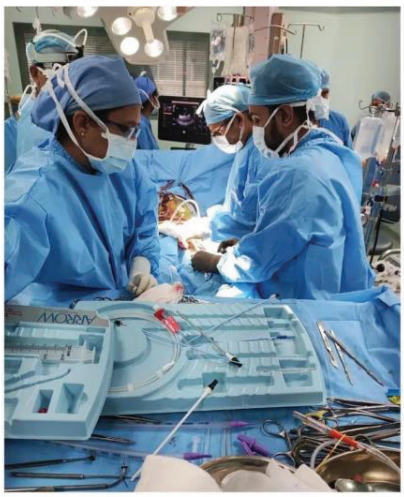
Patient was started on heparin infusion with monitoring. After 48 hours of initiating IABP , Vasopressors were gradually tapered off and after weaning IABP was removed and observed.

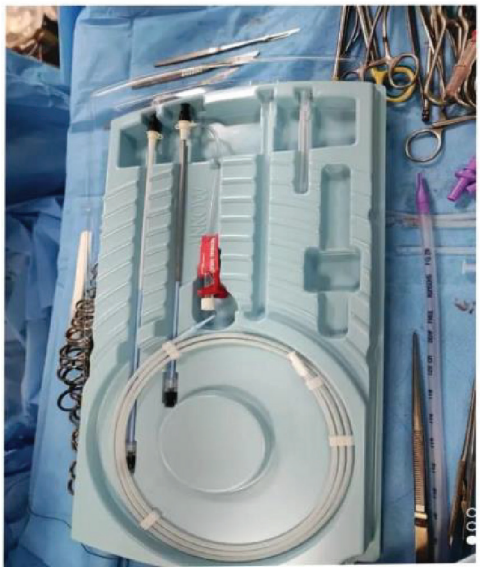
He was transferred to ward with stable haemodynamics.

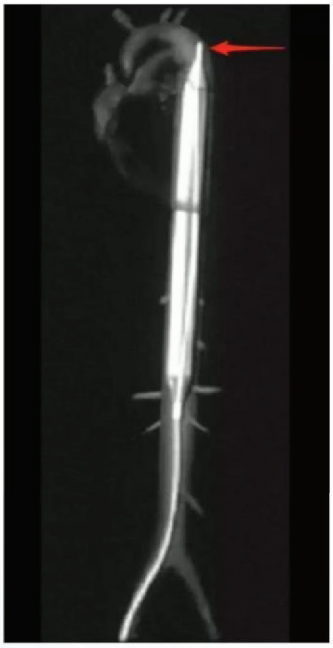
INTRODUCTION

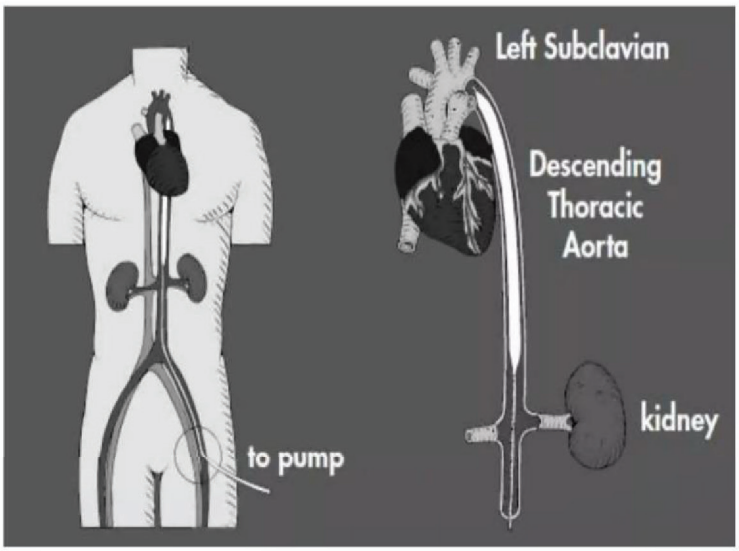
The Intra-Aortic Balloon Pump (IABP) is a crucial life-saving device used in critical cardiac conditions to support the heart's function. It is widely utilized in patients with cardiogenic shock, acute heart failure, and high-risk cardiac procedures. By improving coronary perfusion and reducing the heart's workload, IABP enhances cardiac output and stabilizes patients until further definitive treatment can be administered.

How IABP Works









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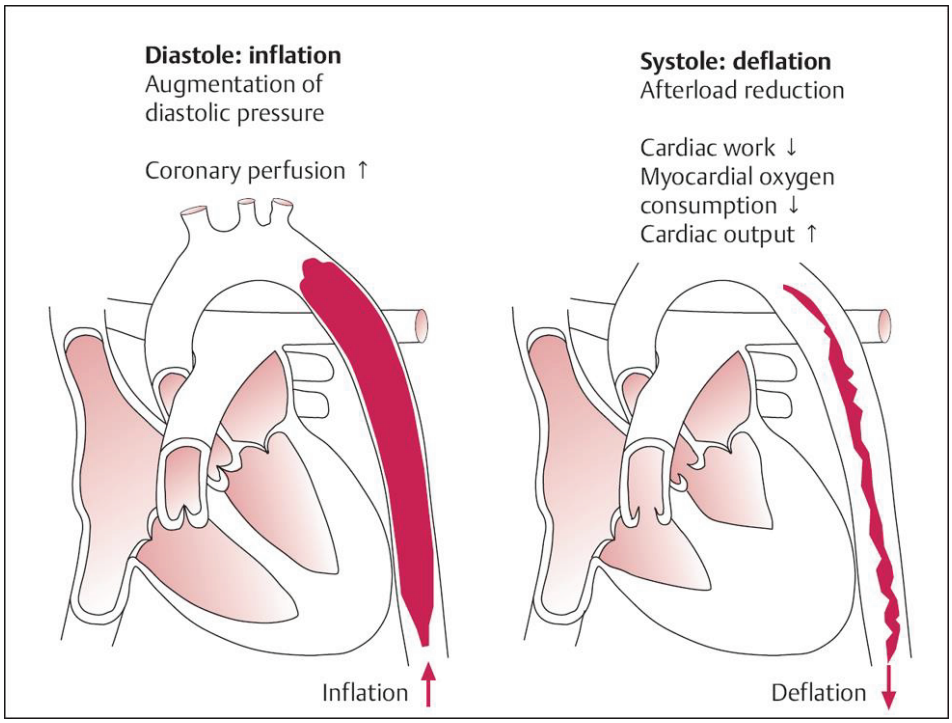
The IABP consists of a catheter with an inflatable balloon that is inserted into the descending thoracic aorta via the femoral artery. It functions in sync with the heart's cycle:

Inflation during Diastole (Heart Relaxation Phase):



The balloon inflates, increasing coronary artery perfusion, which improves oxygen delivery to the heart muscle.

Deflation during Systole (Heart Contraction Phase):
The balloon rapidly deflates, reducing afterload (resistance against which the heart pumps), thus decreasing myocardial oxygen demand and enhancing cardiac output.

Indications for IABP
IABP is used in various life-threatening cardiac conditions, including:
1. Cardiogenic Shock – Commonly after a myocardial infarction (heart attack) where the heart fails to pump effectively.
2. Acute Left Ventricular Failure – Seen in severe heart failure and post-cardiac surgery patients.
3. High-Risk Percutaneous Coronary Intervention (PCI) – Used in patients with poor left ventricular function undergoing angioplasty.
4. Unstable Angina Not Responding to Medications – Helps improve myocardial oxygen supply.
5. Bridge to Definitive Treatment – Stabilizes patients awaiting heart transplant, ventricular assist device (VAD), or coronary artery bypass grafting (CABG).

Contraindications

Despite its benefits, IABP is not suitable for certain patients, including those with:

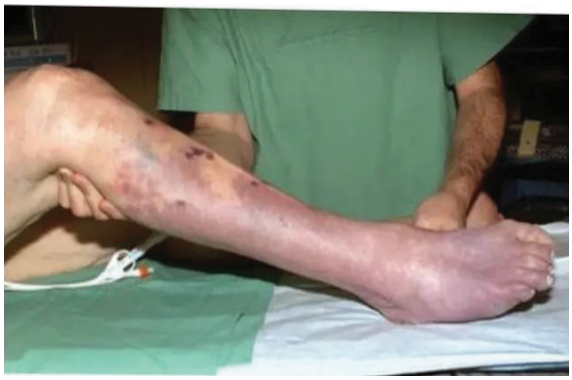
Aortic regurgitation (as balloon inflation can worsen the condition)

Aortic dissection (risk of rupture)

Severe peripheral arterial disease (can lead to limb ischemia)

Uncontrolled sepsis (risk of infection and complications)

Potential Risks and Complications



While IABP is life-saving, it does carry some risks:

Limb Ischemia – Reduced blood flow to the leg due to catheter insertion.

Balloon Rupture – Rare but can lead to embolism.

Infection – Due to prolonged catheter placement.

Aortic Injury – Improper placement may damage the aortic wall.

Conclusion

The Intra-Aortic Balloon Pump (IABP) remains a vital life-saving device in critical cardiac care. By enhancing coronary perfusion and reducing cardiac workload, it serves as an essential bridge to definitive interventions like CABG, PCI, or heart transplant. Despite some risks, its benefits in managing cardiogenic shock and acute heart failure make it an indispensable tool in modern cardiology.

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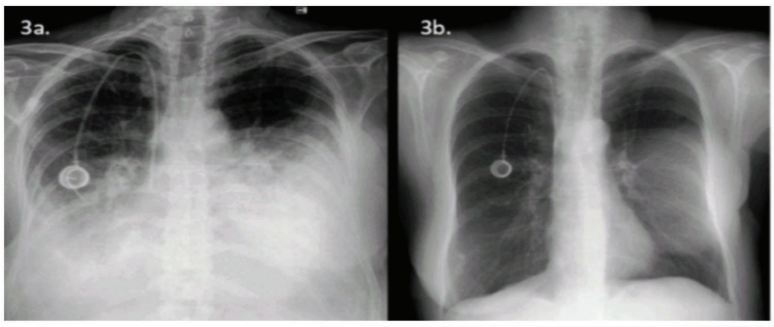
CASE REPORT - 27

A CASE OF METASTATIC CA BREAST ON ALTERNATIVE MEDICINE WITH REPIRATORY DISTRESS AND HYPOXIA (R.A.PURAM)

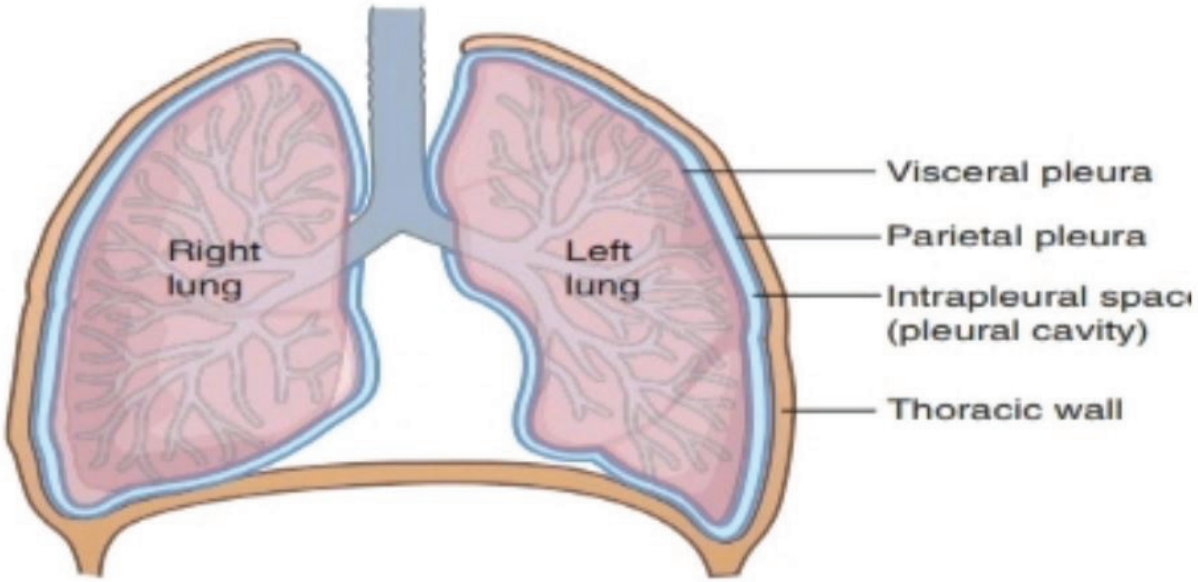
A 45 YEARS OLD FEMALE, A KNOWN CASE OF CA BREAST diagnosed 5 months back, who was on acupuncture treatment, got admitted with complaints of severe breathing difficulty , gradually worsening since one month. Patient was diagnosed to have Bilateral Pleural effusion secondary to metastatic breast carcinoma. Patient required Non invasive ventilation in view of respiratory distress and hypoxia. Patient was seen by pulmonologist and she underwent therapeutic pleurocentesis on both sides. Pt developed Left sided Pneumothorax secondary to ruptured bulla of the underlying bullous lung disease. The CT surgeon was involved and was inserted with Bilateral Intercostal Drainage tube in view of exudative effusion and Hydropneumothorax on the left side. Patient good lung expansion on the right side but had residual pneumothorax on the left side with sustained air leak. Patient was connected with Heimlich value in view of sustained pneumothorax. Patient got stabilised and relieved of breathlessness and was weaned of Non Invasive Ventilation. Medical oncologist opinion was obtained and started on Chemotherapy with first cycle completed uneventfully.

In view of Malignant pleural effusion, patient was planned for pleurodesis. She underwent pleurodesis on the Left side with Bleomycin. The procedure was completed uneventfully and ICD was successfully removed with no further re-accumulation. As the right side lung had continuous fluid collection through the ICD, Pleurodesis was deferred until the fluid collection was less than 100ml in 24 hours. Once the Fluid collection got reduced, Pleurodesis done with Steri-talc with remarkably good results leading to removal of ICD tube on both sides. Patient recovered well and was shifted to the wards for futher management. This case study is to highlight the ignorance of patient in view of breast carcinoma and the treatment strategies. Patient had presented with life threatening hypoxia secondary to the complications of the metastatic disease. Patient was treated for the complications and and is on regular follow up with oncologists.



INTRODUCTION

Pleurodesis is a procedure that obliterates the pleural space with the aim of preventing recurrent pleural effusion or recurrent pneumothorax. It is performed by inducing inflammation of the pleurae by either instilling a chemical sclerosant or by performing mechanical abrasion. This inflammation leads to fibrosis and symphysis of the parietal and visceral pleurae. This method is used to treat recurrent pneumothorax and malignant pleural effusion.



TYPES OF PLEURODESIS

CHEMICAL PLEURODESIS : This is done by inserting a sclerosing agent into the pleural cavity via a chest tube. The drug used causes the pleural surfaces to become sticky and bond together, therefore closing the pleural space.

SURGICAL PLEURODESIS : This is done through medical thoracoscopy, video-assisted thoracoscopy (VATS), or open thoracotomy, where either a sclerosis agent is placed in the pleural cavity or mechanical abrasion (also termed dry abrasion) is achieved by draining the pleural fluid using a tunneled catheter (induces pleurodesis without instillation of a sclerosing agent).

INDICATION

MALIGNANT PLEURAL EFFUSION

This is the most common indication for pleurodesis. Patients diagnosed with malignant pleural effusion (MPE) are candidates for pleurodesis if they satisfy the following criteria:

- The effusion should be symptomatic (producing dyspnea and impairing the quality of life).
- There should be improvement in dyspnea and re-expansion of the lung after removal of the pleural fluid
- The effusion should be rapidly reaccumulating (i.e., requiring therapeutic thoracentesis more than once a month)

PRIMARY SPONTANEOUS PNEUMOTHORAX

- Pleurodesis for PSP is indicated in the following circumstances
- Second episode of PSP (chance of pneumothorax recurrence >60%)
- Persistent air leak following any episode of PSP (lasting >3–5 days)
- Bilateral pneumothorax

SECONDARY SPONTANEOUS PNEUMOTHORAX

- Pleurodesis is indicated in all patients with secondary spontaneous pneumothorax (SSP) even in the first episode as there is a high chance of recurrence (>50%) and the recurrence if occurs leads to more physiologic imbalance and can also be life-threatening due to the presence of underlying lung disease.

NON-MALIGNANT REFRACTORY EFFUSION

Chemical pleurodesis has also been tried with varying degrees of success (50%–80% success rates) in patients with refractory nonmalignant effusions when the treatment of the underlying disease fails to control the rapid reaccumulation of fluid. Some indications include hepatic hydrothorax, uremic effusions, chylothorax, and continuous ambulatory peritoneal dialysis pleural effusions.

CLINICAL MANIFESTATIONS

• Breathlessness • Cough • Chest Pain • Fever

SCLEROSING AGENT

An ideal sclerosing agent would be cost-effective, widely accessible, easy to administer, highly effective at achieving pleurodesis and associated with minimal side effects. Over time, various agents such as autologous blood, doxycycline, iodopovidone, Streptococcus pyogenes A3 (OK-432), and silver nitrate have been utilized. Talc is widely regarded as the most effective and safest option in the current literature. Talc pleurodesis has demonstrated success rates ranging from 80% to 95%, influenced by factors such as dosage, frequency of application, the underlying condition, and the patient's overall health. While other agents are generally effective, none have shown superiority over talc.

Agents	Advantages	Disadvantages	Remarks
Talc	Cheap, easily available, highest efficacy	Reports of ARDS, renal failure	+++
Bleomycin	Efficacy similar to talc	Very costly, chest pain, fever nausea	++
Povidone iodine	Cheap, easily available	Anaphylaxis, randomized study required involving larger number of patients	++
Tetracycline/ doxycycline	Easily available	Very painful, ARF	++

Following are the sclerosing agents that can be used as chemicals for chemical pleurodesis:

•Talc, Tetracyclines (minocycline, doxycycline), Silver nitrate, Iodopovidone, Bleomycin, Corynebacterium parvum with parenteral methylprednisolone acetate, Erythromycin, Fluorouracil, Interferon beta, Autologous blood, Mitomycin C, Cisplatin, Cytarabine, Doxorubicin, Etoposide, Bevacizumab (intravenous or intrapleural), OK-432.

TECHNIQUES AND PROCEDURE

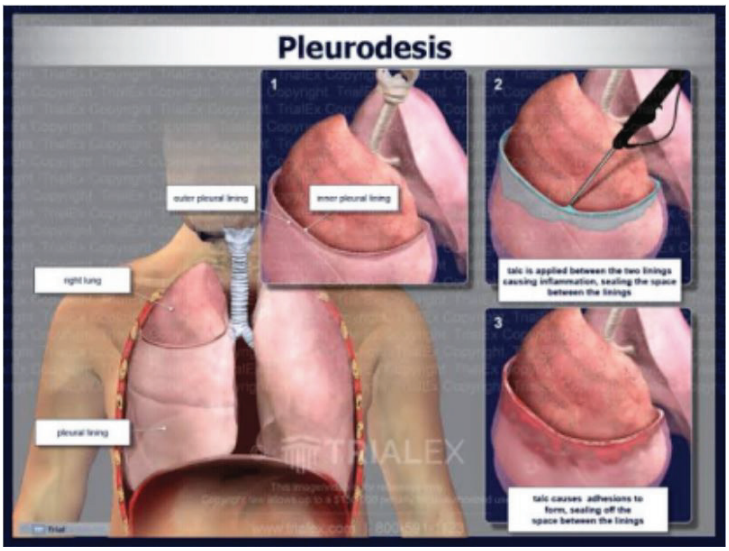
PREPARATION

Proper preparation for pleurodesis is essential to optimize outcomes, minimize complications, and ensure patient safety. The process begins with thorough patient selection and evaluation. Informed consent from the patient should be obtained with an explicit discussion of the procedure's indications, alternatives, risks, and complications. The procedural site should be marked, and the status of nil per os should be confirmed. Team preparation involves ensuring the availability of necessary equipment, including chest tubes, sclerosants, and imaging tools, as well as confirming the readiness of the multidisciplinary team. An appropriate plan for sedation and anesthesia should be established. For chemical pleurodesis, a patent chest tube should be in situ, the lung should be expanded to the chest wall, and fluid output from the chest drain should be less than 100 mL in the last 24 hours.

The procedure for chemical pleurodesis is as follows:

The procedure for instilling a sclerosant for pleurodesis begins with sterile preparation. Wear sterile gloves and clean the chest tube and surrounding area with povidone iodine or another sterilizing agent. Position a sterile dressing beneath the chest tube and 3-way stopper. Remove the cap from the chest tube and clean it with the 3-way stopper. Prepare 1% lidocaine in a syringe, attach it to the 3-way stopper, and instill the lidocaine into the pleural cavity to provide local anesthesia. Remove the syringe and turn off the stopper.

Next, prepare the sclerosant slurry by mixing the agent (eg, talc) with 40 mL of normal saline in a 50 mL syringe. Shake well, as talc is difficult to dissolve, and keep the syringe moving continuously to prevent precipitation. Instill the prepared slurry into the pleural cavity via the chest tube and flush it immediately with 10 mL of normal saline to ensure delivery. Close the 3-way stopper and keep it closed for 3 hours to allow the sclerosant to act effectively.



After pleurodesis, additional analgesia may be necessary to manage pain; however, NSAIDs should generally be avoided as they can inhibit the inflammatory response required for effective pleurodesis. After 3 hours, the chest drain should be reopened to resume drainage. A chest radiograph should be obtained 24 hours postprocedure to confirm the absence of pneumothorax or fluid reaccumulation. The chest tube can be safely removed if the radiograph is satisfactory and there is no further fluid output. Follow-up, including a chest radiograph, is recommended after 4 to 6 weeks to assess the procedure's success and ensure patient stability.

Pleurectomy

Pleurectomy involves the radical resection of the visceral and parietal pleura (total or subtotal) and may include decortication, which removes a fibrous pleural rind. This procedure can be employed to control malignant pleural effusions in cases where chemical pleurodesis has failed. Pleurectomy and decortication may also serve as primary therapeutic options for patients with malignant pleural effusions due to mesothelioma. However, these interventions do not improve overall survival and are associated with significant complications. Given the invasiveness and morbidity of this thoracotomy-requiring procedure, it is reserved for patients with good surgical candidacy and a reasonably prolonged life expectancy.

Mechanical Pleurodesis

Mechanical pleurodesis induces pleural adhesion through physical abrasion of the pleural surfaces, typically during thoracoscopy or thoracotomy. This method is primarily used for recurrent pleural effusions, pneumothoraces, or in conjunction with procedures for malignant pleural effusion. For thoracoscopy, a thoracoscope is inserted through one or more small incisions, while thoracotomy requires a larger incision to expose the pleural cavity. Any pleural fluid or air is removed to collapse the lung partially, improving visibility and access to the pleural space. The pleural cavity is then carefully inspected for lesions, adhesions, or abnormalities. During mechanical abrasion, a sterile surgical pad, gauze, or specialized pleural abrader gently rubs the parietal pleura, creating uniform abrasions. This induces an inflammatory response that promotes the release of cytokines and fibrin deposition, leading to pleural adhesion and fibrosis. Care must be taken to avoid damaging underlying structures, such as the intercostal nerves and blood vessels. After ensuring adequate abrasion, the pleural cavity is inspected for complications like bleeding or lung injury. The lung is then reexpanded to its full volume to ensure contact between the visceral and parietal pleura, facilitating adhesion formation.

A small-bore chest tube is inserted to drain any remaining pleural fluid or air and is typically left in place until fluid output is minimal and pleural adherence is confirmed.

Tunneled pleural catheter placement is a highly effective palliative strategy for managing malignant pleural effusion, offering significant symptom relief while facilitating outpatient care. The minimally invasive nature of this catheter and the low complication risk make it an optimal choice for patients with malignant pleural effusion, particularly those with limited life expectancy or recurrent effusions. Tunneled pleural catheter insertion should be considered a primary treatment option for these patients to enhance quality of life and minimize hospital stays.[22]

Postprocedure Care

After pleurodesis, additional analgesia may be necessary to manage pain; however, NSAIDs should generally be avoided as they can inhibit the inflammatory response required for effective pleurodesis. After 3 hours, the chest drain should be reopened to resume drainage. A chest radiograph should be obtained 24 hours postprocedure to confirm the absence of pneumothorax or fluid reaccumulation. The chest tube can be safely removed if the radiograph is satisfactory and there is no further fluid output. Follow-up, including a chest radiograph, is recommended after 4 to 6 weeks to assess the procedure's success and ensure patient stability.

COMPLICATIONS

Pleurodesis, while effective for managing recurrent pleural effusions and pneumothoraces, carries risks of complications and variable success rates influenced by patient factors, tumor characteristics, and procedural nuances.

Complications of Pleurodesis

- Pain and inflammation
- Pleural inflammation induced by chemical agents or mechanical abrasion can cause significant chest pain, often requiring NSAIDs or opioids.
- Fever is a common inflammatory response.
- Infection
- Failure to maintain an aseptic technique can result in pleural infection or empyema.

Respiratory complications

Potential complications include pneumothorax, hemothorax, reexpansion pulmonary edema, and acute respiratory distress syndrome (ARDS). Due to systemic absorption, ARDS has been reported with talc use, especially when smaller talc particles or large doses are administered.

Systemic Effects

Systemic inflammation, coagulation cascade activation, and pulmonary embolism have been documented, particularly with chemical pleurodesis.

Failure of Pleurodesis

Trapped lung, inadequate lung expansion, or insufficient inflammatory response may lead to procedural failure.

Failure of the procedure may be due to increased tumor burden, which causes a decrease in the mesothelial cells and, therefore, an inadequate inflammatory response.[27]

The type of tumor may also play a role in the process; diffuse mesothelioma and metastatic carcinomas have an inadequate response. This is because the healthy mesothelial cells secrete the inflammatory mediators necessary for fibrosis.

Surgical Complications

Surgical pleurodesis carries risks related to general anesthesia, wound infection, and atelectasis.

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- ***Dr. Vinod*** *MBBS.,DA*

CASE REPORT - 28

DIAGNOSIS: TYPE II RESPIRATORY FAILURE SECONDARY INFECTION (R.A.PURAM)

A 42yrs male came with decreased response from yesterday evening, difficulty in breathing, decreased appetite.In view of low GCS, patient was intubated, later weaned off from mechanical ventilator.patient continued to have low urine output along with altered renal function test.patient was escalated to intermittent BIPAP. Patient improved symptomatically hence discharged.

- ***Dr.Jeykumar** MBBS.,MD (anaesthesia)*
- ***Dr. Senthil** MBBS.,DA*
- ***Dr. Vinod** MBBS.,DA*

CASE REPORT - 29

Diagnosis: Sepsis/diabetic ketacidosis/hyperkalemia/acute kidney injury/ Diabetes mellitus/ systemic hypertension. *(R.A.PURAM)*

A 74yrs female came with decreased response associated with right hand stiffness and reduced intake of food,vomit,headache. In view of poor GCS. Patient was intubated and mechanically ventilated. ABG showed severe metabolic acidosis. Patient was started on supportive treatment - insulin infusion, calcium supplement.Under aseptic precaution USG guided right IVJ catheter inserted.patient hemodynamically improved. Hence discharged in stable condition.

OVERVIEW

DKA is a serious complication of diabetes that can be life-threatening. DKA is most common among people with type 1 diabetes. People with type 2 diabetes can also develop DKA.

DKA develops when your body doesn't have enough insulin to allow blood sugar into your cells for use as energy. Instead, your liver breaks down fat for fuel, a process that produces acids called ketones. When too many ketones are produced too fast, they can build up to dangerous levels in your body.

Causes:-

Very high blood sugar and low insulin levels lead to DKA. The two most common causes are:

1. Illness. You may not be able to eat or drink as much as usual, which can make blood sugar hard to manage.
2. Missing insulin shots, a clogged insulin pump, or the wrong insulin dose.

Other causes of DKA include:

- Heart attack or stroke.
- Physical injury, such as from a car accident.
- Alcohol or drug use.
- Certain medicines, such as some diuretics (water pills) and corticosteroids (used to treat inflammation in the body).

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CASE REPORT - 30

Diagnosis: Acute pulmonary edema Old AWMI CAD - Triple vessel disease Systemic hypertension *(R.A.PURAM)*

A 72yrs old male came with complaints of giddiness while walking for past 1 month. CAG done showed calcification Triple vessel disease recanalized. Adviced PCI to LCX and RCA for PTCA to POBA to LCX Under local anaesthesia through femoral artery access, 6F EBU 3.5 guiding catheter was used to engage LM.Lesion was crossed with guide wire then pre- dilated with 2.75*12 mam Accu-force balloon 12atm for 15secs. After pre-dilatation patient patient developed flash pulmonary edema which was managed with NIV and diuretics. In view of heavily calcific lesion and patient in acute pulmonary edema, PTCA was defered and stop with POBA. No evidence of thrombus or residual stenosis.In view of respiratory distress was was intubated and connected to mechanical ventilator.patient had high BP 180/120mmHg which was managed with NTG infusion.ABG showed respiratory acidosis with CO2 narcosis. Patient was gradually weaned off from ventilator and extubated. Post which patient developed one episode of atrial fibrillation which was managed with anti- arrhythmic drug and gradually tapered and. Stopped. Patient hemodynamically improved hence discharged.

OVERVIEW

Pulmonary edema can be defined as an abnormal accumulation of extravascular fluid in the lung parenchyma. This process leads to diminished gas exchange at the alveolar level, progressing to potentially causing respiratory failure. Its etiology is either due to a cardiogenic process with the inability to remove sufficient blood away from the pulmonary circulation or non-cardiogenic precipitated by injury to the lung parenchyma. It is an important pathologic feature in many disease processes, and hence learning the underlying disease process is crucial to guide its management. Clinical features include progressive worsening dyspnea, rales on lung auscultation, and worsening hypoxia.

Pulmonary edema can be broadly classified into cardiogenic and noncardiogenic pulmonary edema.

Cardiogenic or volume-overload pulmonary edema arises due to a rapid elevation in the hydrostatic pressure of the pulmonary capillaries. This is typically seen in disorders involving left ventricular systolic and diastolic function (acute myocarditis including other etiologies of non-ischemic cardiomyopathy, acute myocardial infarction), valvular function (aortic/mitral regurgitation and stenosis in the moderate to the severe range), rhythm (atrial fibrillation with a rapid ventricular response, ventricular tachycardia, high degree, and third-degree heart block).

Non-cardiogenic pulmonary edema is caused by lung injury with a resultant increase in pulmonary vascular permeability leading to the movement of fluid, rich in proteins, to the alveolar and interstitial compartments. Acute lung injury with severe hypoxemia is referred to as acute respiratory distress syndrome (ARDS) and is seen in various conditions directly affecting the lungs, such as pneumonia, inhalational injury, or indirectly, such as sepsis, acute pancreatitis, severe trauma with shock, multiple blood transfusions.

CASE REPORT - 31

Diagnosis: Severe acute exacerbation of COPD with decompensated type 2 respiratory failure/ for pulmonale/ atrial fibrillation with FVR/ hyponatremia/? Right neck of femur fracture. ***(R.A.PURAM)***

A 91year old male came with c/o shortness of breath associated with drowsiness,slurring of speech and lower limb weakness and swelling , followed by fall at home last week.

ABG showed metabolic acidosis, ECG showed Atrial fibrillation. In view of low GCS and threatened airway patient was intubated and connected to mechanically ventilator and was started on anti- arrhythmic drug. CT brain showed age related changes and small ischemic disease. Pt was gradually weaned off from ventilator and extubated. Adviced conservative management Hence discharged.

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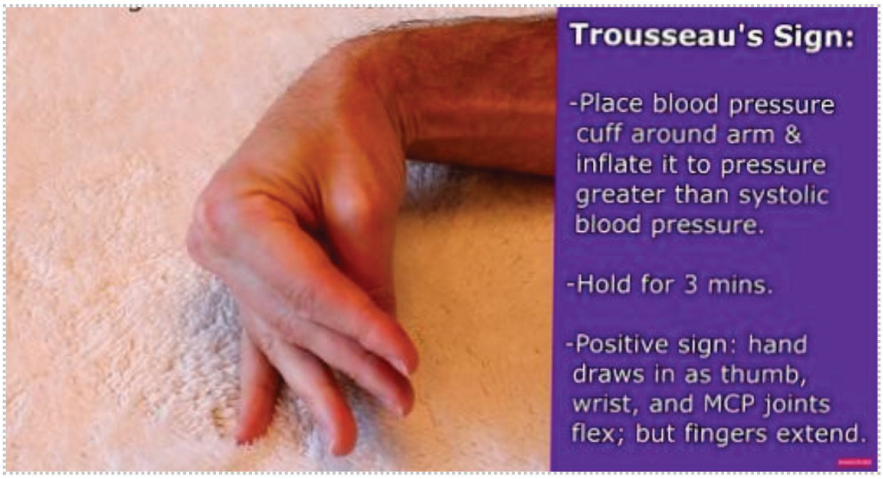
CASE REPORT - 32

RESISTANT HYPOCALCEMIA FOLLOWING PARATHYROIDECTOMY (R.A.PURAM)

68 year old female patient presented with history of pain in the legs and swelling of the neck. She was evaluated and PET scan showed Right superior pole Parathyroid Adenoma.

S.calcoim wa 10.9
PTH – 239.7
S.magnesium.- 2.5.

After assessment, cardiologist, endocrinologist and Anesthesiologist opinion, patient underwent comprehensive parathyroidectomy with Total thyroidectomy. Patients serum calcium, PTH levels, serum magnesium was monitored post operatively and started on calcium supplement and thyroxine. Patient was observed and transferred to the ward.



In the wards patient developed severe pain in both the upper limbs with tingling.

Trousseaus sign was positive.

Patient was transferred to the ICU and stated on calcium infusion, magnesium. Serum calcium improved .she was discharged. The patient got readmitted after a week with loss of appetite, severe vomiting, diarrhea, generalized body pains and hypotension.

Investigations showed
S.calcium- 5.3
Creatinine 1.4
Patient had blood pressure 80/50 mmHg. She was started on iv fluids, vasopressors, calcium gluconate infusion, after taking samples for blood culture started on carbapenem. Patient gradually improved, and was discharged. Patient had hungry bone syndrome due to multiple brown rumours secondary to chronic hypoparathyroidism.

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RESISTANT HYPOCALCEMIA FOLLOWING PARATHYROIDECTOMY (R.A.PURAM)

Resistant hypocalcemia is a potential complication following parathyroidectomy, particularly in patients with primary or secondary hyperparathyroidism. This condition occurs when calcium levels remain persistently low despite standard supplementation.

CAUSES OF RESISTANT HYPOCALCEMIA POST-PARATHYROIDECTOMY

1. Hungry Bone Syndrome (HBS)
After removal of overactive parathyroid glands, bones rapidly absorb calcium, magnesium, and phosphate, leading to prolonged hypocalcemia. More common in patients with severe hyperparathyroidism, chronic kidney disease (CKD), or high preoperative bone turnover.

2. Inadequate Calcium and Vitamin D Supplementation
Standard calcium and vitamin D doses may not be sufficient, particularly in patients with preexisting bone disease.

3. Hypomagnesemia
Magnesium deficiency impairs PTH secretion and action, contributing to persistent hypocalcemia.

4. Delayed Recovery of Parathyroid Glands
Transient hypoparathyroidism may occur due to gland trauma, ischemia, or removal of all parathyroid tissue.

5. Chronic Kidney Disease (CKD) or Vitamin D Deficiency
Reduced renal conversion of 25(OH)D to active $1,25(OH)_2D$ may impair calcium absorption and exacerbate hypocalcemia.

6. Alkalosis or Excessive Phosphate Binding
Metabolic alkalosis or excessive use of phosphate binders (especially in CKD patients) can worsen hypocalcemia.

MANAGEMENT STRATEGIES

1. Aggressive Calcium Supplementation
Oral calcium: 2–4 g/day (as calcium carbonate or citrate).
IV calcium gluconate: In severe cases, continuous infusion may be required.

2. Active Vitamin D (Calcitriol or Alfacalcidol)
Calcitriol (0.5–2 µg/day) to enhance intestinal calcium absorption.

3. Magnesium Replacement
If hypomagnesemia is present, magnesium sulfate (IV) or oral magnesium should be given.

4. Phosphate Management
In CKD or HBS, phosphate levels should be monitored and controlled to avoid worsening hypocalcemia.

5. Monitoring and Long-Term Follow-Up
Frequent monitoring of calcium, phosphate, magnesium, PTH, and vitamin D levels is essential, especially in high-risk patients.

- ***Dr.Jeykumar*** *MBBS.,MD (anaesthesia)*
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ACCIDENT & EMERGENCY MEDICINE/ CRITICAL CARE UNIT CONSULTANT

EMERGENCY MEDICINE DEPT - SHENOY NAGAR

CRITICAL CARE UNIT - SHENOY NAGAR

CRITICAL CARE UNIT - R A PURAM

Billroth Hospitals

24/7 READY TO SAVE LIVES

EMERGENCY CARE DEPARTMENT

Emergency Department team - Shenoy Nagar
Dr.Chakravathi Alapati MBBS.,MD(anaesthesia).,IDCCM (HOD)
Dr.T.Lavanya Saisurendar MBBS.,DEM.,FICM.,MRCEM
Dr.Mohammed Ghouse Khan MBBS.,MEM
Dr.Hemalatha MBBS.,MEM
Dr.Mohammed Mustafa Shahi MBBS.,FEM.,MRCEM

Critical Care unit team - Shenoy Nagar
Dr Chakravarthi MD (anaesthesia).,IDCCM(HOD)
Dr.Preethi MD (anaes)
Dr.Santhini DNB (anaes)
Dr.Aswathy MD (anaes)
Dr.Manoj MD (anaes)
Dr.Umakanth MD.,FICCM
Dr.Sivaram MD.,IDCCM
Dr.Balaji MD (anaes)

Critical Care unit - R.A.Puram
Dr.Jeykumar MBBS.,MD
Dr.Senthil MBBS.,DA
Dr.Vinod MBBS.,DA

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Dr.vinith
Dr.Sam Paul Wesley
Dr.Syed Mhd Abdul Huq

SECOND YEAR
Dr.Pradeish
Dr.Srinivas
Dr.Jawahar Jeyesh
Dr.Dhinesh kumar
Dr.Nandhakumar
Dr.Muralidharan

FIRST YEAR
Dr.Karthick
Dr.Alwinsam
Dr.Madhan Kumar
Dr.Kaushik

NETAXIS PVT LTD BLOOD DONATION DRIVE: A REMARKABLE SUCCESS!

We are thrilled to share the success of our recent Netaxis PVT LTD Blood Donation Drive. This incredible achievement was made possible by the generosity and enthusiasm of our donors and dedicated staff members.

Blood donation is a simple yet powerful act that saves lives. Every unit collected contributes to providing critical care to patients in need. Your support and participation in this initiative highlight the spirit of compassion and community service that drives us forward.

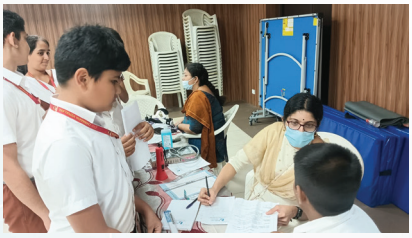
Together, we care, we give, we save lives.

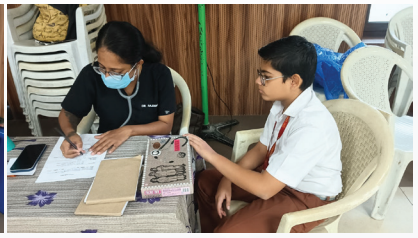
BILLROTH HOSPITALS CONDUCTS FREE HEALTH CHECKUP FOR CHINMAYA SCHOOL STUDENTS

In an effort to promote health and well-being among the younger generation, Billroth Hospitals organized a comprehensive free health checkup camp for the students of Chinmaya School. The initiative, held on 3rd to 6th Feb, aimed at ensuring the overall health of the students and raising awareness about the importance of regular medical checkups.
The camp, conducted by a team of experienced doctors from Billroth Hospitals, included a thorough medical examination covering various aspects such as vision, dental health, physical fitness, and general wellness. The team also provided valuable advice on maintaining a balanced diet and staying physically active.

The management and staff at Chinmaya School expressed their gratitude to Billroth Hospitals for their proactive approach in addressing the health concerns of the students. Parents and teachers also appreciated the initiative, which provided an opportunity for the students to receive early detection of any potential health issues.

The free health checkup was well-received, with many students benefiting from expert consultations and personalized health advice. Billroth Hospitals continues to work towards its mission of providing accessible and high-quality healthcare to the community through such outreach programs.





BILLROTH HOSPITALS CONDUCTS FREE HEALTH CHECKUP FOR KOLAPERUMAL SCHOOL STUDENTS

Billroth Hospitals, known for its commitment to community healthcare, organized a free health checkup camp for the students of Kolaperumal School from 27th January to 31st January 2025. This initiative aimed at promoting early detection of health issues and encouraging a healthy lifestyle among young students. A team of expert doctors, including pediatricians, general physicians, dentists, and ophthalmologists, conducted comprehensive medical examinations covering vision screening, dental health, general wellness, and nutritional guidance. The camp provided students with valuable health insights and preventive care tips to ensure their well-being. The school management expressed gratitude to Billroth Hospitals for their proactive healthcare initiative. Teachers and parents also appreciated the effort, emphasizing the importance of such programs in maintaining students' overall health.

The initiative was well-received, with a large number of students benefiting from expert consultations and personalized health advice. Billroth Hospitals remains committed to its mission of delivering quality healthcare and will continue conducting similar outreach programs in the future.

PRIORITIZE YOUR HEALTH – EXCLUSIVE HEALTH CHECKUP CAMP FOR PNB EMPLOYEES!

We are pleased to announce a General Health Checkup Camp for Punjab National Bank employees on 7th and 8th February. This camp aims to promote preventive healthcare by offering essential screenings, expert consultations, and personalized health guidance. Employees can get their Blood Pressure, Sugar Levels, ECG, BMI checked, along with a doctor consultation for further recommendations. Early detection plays a crucial role in maintaining long-term health.

LAMP LIGHTING CEREMONY & CONVOCATION CELEBRATION AT OUR NURSING COLLEGE

On February 3rd, the prestigious Lamp Lighting Ceremony took place, where Dr. D. Saritha and Prof. Dr. C. Rajendiran graced the occasion with their inspiring presence. This momentous event marked the beginning of the nursing journey for the new batch of students, instilling in them a sense of pride, responsibility, and commitment to the noble profession of nursing.

The following week, on February 10th, the college celebrated the Convocation Ceremony, presided over by the esteemed Dr. G.Kumaragurubaran. The event saw our graduates don their robes with pride as they stepped into the professional world, ready to take on the challenges and opportunities in the healthcare sector. The ceremony was a true reflection of the dedication and hard work of both students and faculty, celebrating the culmination of years of academic excellence.

These events were filled with inspiration, joy, and a deep sense of accomplishment. We extend our heartfelt congratulations to all the graduates and thank everyone who contributed to making these events a resounding success!

BILLROTH HOSPITALS CELEBRATES PONGAL WITH ENTHUSIASM AND UNITY

Billroth Hospitals recently hosted a vibrant Pongal celebration, bringing together the hospital staff for an unforgettable experience. The event was filled with traditional folk performances that echoed the rich cultural heritage of Tamil Nadu, and exciting games that engaged attendees of all ages. The festive atmosphere was further enhanced by a variety of stalls offering delicious local delicacies and crafts, capturing the true spirit of Pongal.

BILLROTH HOSPITALS CELEBRATES PONGAL WITH ENTHUSIASM AND UNITY

The celebration emphasized the importance of unity and joy, as people from all walks of life gathered in harmony to mark this auspicious occasion. It was a beautiful reminder of the cultural richness that binds us together, making the day both meaningful and memorable for all.

EMERGENCY CALL

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FOR APPOINTMENTS

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R. A. Puram: 044-2464 1111

Shenoy Nagar: 43, Lakshmi Talkies Road, Chennai - 600030

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