Case Reports

Masquerading Pneumonia - Pulmonary Contusion

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Abstract

Pneumonia is diagnosed and treated with symptoms of fever, cough, expectoration and chest X-ray showing evidence of consolidation. When a pattern of opacification does not fit into segmental or lobar pattern, reevaluation is essential. We report a case of 18 yr old boy with symptoms of respiratory infection, and X-ray diagnosis suggestive of infective consolidation which turned out to be a case of pulmonary contusion.

Introduction

Pneumonia -- inflammation of lung parenchyma is usually due to infective causes. When symptoms are suggestive, and X-ray is supportive, provisional diagnosis is made and therapy started. However certain clinical situations and X-ray patterns need reevaluation of the patient and therefore the diagnosis.

Case Report

An 18 yr old boy was referred as a case of consolidation of right lung. He presented with fever, cough, expectoration, and dyspnea of 3 days duration. It was associated with vague pain in the right hemi thorax. No history of hemoptysis, wheeze, anorexia, PND, syncope and orthopnea. No history of major medical illness in the past. Family history, personal history, and occupational history were not contributory.

On admission patient was conscious, afebrile, not anemic, not cyanosed, no clubbing. Vitals were stable. Respiratory system examination revealed no mediastinal shift, normal movements of the chest, vocal fremitus, vocal resonance reduced on right inter scapular, mammary, axillary regions, associated with an impaired note on percussion in the same areas. Breath sound intensity was diminished over the above mentioned areas, no added sounds. His cardiovascular, abdomen, central nervous system examinations were normal.

A chest X-ray taken, revealed a dense, homogenous, circumscribed opacification in right mid zone but it was not confined to any particular lobe or segment. Silhouette sign was negative. No air bronchogram visualized (Fig. 1).

Provisional diagnosis of ? Consolidation ? Mass ? Loculated pleural effusion was made and further work up done. Patient was started on Tab. Clarithromycin 500 mg OD, with Tab. Paracetamol 500 mg SOS. Sputum culture, AFB, biochemical tests were normal. Ultrasonogram chest showed hypo echoic lesion with air pockets seen in right infra clavicular region, and was reported as consolidation right lung. HRCT chest was done, which revealed a cavity with surrounding opacification and air bronchogram, and was reported as necrotizing pneumonia (Figs. 2 & 3).

However the clinical picture and radiological reports do not correlate. Detailed reevaluation and probing elicited a history of fall from a tree one day prior to the onset of illness which was not forthcoming in initial evaluation as the patient thought it was not significant. Patient improved, X-ray shadows resolved completely within 10 days (Fig. 4). Hence a diagnosis of pulmonary contusion masquerading as pneumonia was concluded on day 10.

Fig. 1: Chest x-ray on admission

![Fig. 1: Chest x-ray on admission](image1)

Fig. 2: HRCT on admission

![Fig. 2: HRCT on admission](image2)

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Discussion

In 1761, the Italian anatomist Giovanni Battista Morgagni was first to describe a lung injury that was not accompanied by injury to the chest wall overlying it. Nonetheless, it was the French military surgeon Guillaume Dupuytren who is thought to have coined the term *pulmonary contusion* in the 19th century.

Pulmonary contusion is injury to the lung parenchyma; it may or may not be associated with chest wall trauma. Blast injuries and blunt injuries usually produce lung contusion and due to which, torn capillaries leak fluid into the tissues surrounded them. It is more common in children because of pliability of ribs. There is damage to alveolar capillary membrane and small blood vessels. This leads to leakage of fluid and blood into the alveoli and interstitial space. The extent of damage depends on the severity of the trauma. Depending on the extent the clinical picture may vary from asymptomatic X ray shadows, to frank ARDS.

Pulmonary contusion results from rapid deceleration, when the moving chest strikes a fixed object. Motor vehicle accidents and blast injuries invariably produce pulmonary contusion. Mechanisms of pulmonary contusion development include;

- In the inertial effect, the lighter alveolar tissue is sheared from the heavier hilar structures, an effect similar to diffuse axonal injury in head injury. It results from the fact that different tissues have different densities, and therefore different rates of acceleration or deceleration.

- In the spalling effect, lung tissue bursts or is sheared where a shock wave meets the lung tissue, at interfaces between gas and liquid. The alveolar walls form such a gas-liquid interface with the air in the alveoli. The spalling effect occurs in areas with large differences in density; particles of the denser tissue are spalled (thrown) into the less dense particles.

- In the implosion effect occurs when a pressure wave passes through a tissue containing bubbles of gas; the bubbles first implode, then rebound and expand beyond their original volume. The air bubbles cause many tiny explosions, resulting in tissue damage; the overexpansion of gas bubbles stretches and tears alveoli. This effect is thought to occur microscopically when the pressure in the airways increases sharply.

- Contusion usually occurs on the lung directly under the site of impact, but, as with traumatic brain injury, a contrecoup contusion may occur at the site opposite the impact as well.

- Symptoms include dyspnea, tachypnea, tachycardia, cough, wheeze, bronchorrhea and hemoptysis. Examination may reveal chest wall tenderness, cyanosis, decreased breath sounds and hypotension.

X ray appearance is similar to that of aspiration. Contusion is not typically restricted by the anatomical boundaries of the lobes or segments of the lung. Chest x ray is often not sensitive, ultra sound detects much earlier and accuracy comparable to CT. CT scanning can detect the contusion immediately after the injury, best visualized over first 24 to 48 hrs. It also helps to determine the extent of the contusion and the need for mechanical ventilation.

Management is purely supportive, keeping track of fluid balance, respiratory function and oxygen saturation. Early detection and treatment of complications such as secondary infections, respiratory failure and ARDS saves life. Prognosis is excellent with spontaneous resolution usually within 10 days. Pulmonary contusion can result in respiratory failure—about half of such cases occur within a few hours of the initial trauma. Other severe complications, including infections and acute respiratory distress syndrome (ARDS) occur in up to half of cases. Fibrosis as a squeale has been reported. Elderly people, preexisting co morbid illness are likely to stay longer in hospital, and more prone for complication.

Prevention of pulmonary contusion is similar to that of other chest trauma. Airbags in combination with seat belts can protect vehicle occupants by preventing the chest from striking the interior of the vehicle during a collision, and by distributing forces involved in the crash more evenly across the body.

Conclusion

Lung can be injured without injury to the chest wall. When a “consolidation” on chest x ray does not fit into anatomical pattern, reevaluate for mass and loculated pleural effusion. However pulmonary contusion should be thought of and history of deceleration impact must be searched for. Central air shadow in a consolidation need not be a cavitation always, but could be also clearance of contusion. When history and investigation does not match, reevaluation of history will be rewarding.

References

1. Cohn SM. “Pulmonary contusion: Review of the clinical entity”.

Fig. 3 : HRCT on admission

Fig. 4 : X-ray chest a day 10
Ultra-Short Course of Low-Dose Amiodarone-Induced Post-Operative Fatal Pulmonary Toxicity

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Abstract
A 58-year-old male diabetic patient with severe left ventricular dysfunction and pulmonary arterial hypertension successfully underwent coronary artery by pass grafting (CABG) and was extubated 48 hours after surgery. Patient had atrial fibrillation on 3rd post-operative day requiring loading dose of amiodarone followed by maintenance dose to control the arrhythmia. On 4th post operative day patient became tachypnoiec and required higher concentration of oxygen to maintain SpO2 >90%. There was new infiltrates on the x-ray, which was more on right side. Initially treated as pulmonary infection and antifailure measures. The patient did not respond and the lesions progressed to opaque hemithorax by the 10th postoperative day. On 11th POD he was reintubated due to respiratory distress. After excluding pulmonary infections, pulmonary oedema, embolism and vascular obstruction, the possibility of drug induced pulmonary toxicity was considered. Hence amiodarone was withdrawn and steroid was initiated. There was good radiological and gas exchange improvement and he was extubated the following day. After one week course of steroids the infiltrates cleared and oxygenation also improved. Post CABG patients are prone for acute amiodarone toxicity and high index of suspicion is needed to diagnose this early so that fatal complication can be averted by timely intervention.

Introduction
Amiodarone is a tri iodinated drug used to treat tachyarrythmias and the drug usually accumulates in lung and liver where its half-life is extremely long (25 to 60 days) and produces serious adverse effects. In about two third of patients, amiodarone pulmonary toxicity can present in a sub-acute or chronic form, whereas in one third it can present acutely. To avoid hazardous complications, a maintenance dose of 200 mg / day is recommended. Acute pulmonary toxicity is multifactorial as it is seen even with low dose and short duration. This is most likely in postoperative setting. There is often a delay in diagnosis as this possibility is not considered in the acute care setting and it may lead to fatal complication. Diagnosis is difficult to establish in a patient with compromised cardiac function as the clinical features and investigations are non-specific. Early diagnosis is crucial since pulmonary toxicity is reversible following discontinuation of drug and steroids.

Case Report
A 58-year-old male diabetic with triple vessel coronary artery disease was admitted for coronary artery by pass grafting. His left ventricular function was severely impaired with an ejection fraction of 25%. There was moderate pulmonary arterial hypertension. He successfully underwent coronary artery by pass with left internal mammary graft to left anterior descending artery and saphenous vein graft to ramus and posterior interventricular artery. Aorta cross clamp and cardiopulmonary bypass time was 74 and 144 minutes respectively. IABP was placed postoperatively. In addition to this ionotropes were also...
started to optimize the haemodynamics (vasopressin, adrenaline, milrinone). He was off IABP support by third postoperative day and ionotropes were also tapered off by fourth POD. 48 hours after surgery he was extubated without any difficulty and by the end of second postoperative day (POD) he was breathing comfortably with minimal supplemental oxygen. On third postoperative day patient had atrial fibrillation with rapid ventricular rate, which was reverted to sinus rhythm by amiodarone. On 5th POD, the patient became tachypnoeic, and required high concentration of oxygen to maintain SpO2 >90%. Fresh infiltrates appeared on the chest radiograph which was normal pre-operatively with cardiomegaly (Figs. 1, 2). He was afebrile and hemodynamically stable. Biochemical parameters were normal. Suspecting hospital acquired pulmonary infection; he was started on broad-spectrum antibiotics (cefaperazone + sulbactum, 2 gram IV Q12H). Over next few days patient’s condition deteriorated with worsening hypoxemia and progression of radiological infiltrates. With onset of hypoxemia, patient was placed on a bilevel positive airway pressure therapy with an EPAP of 7 cm and IPAP of 12 cm H2O (BiPAP vision, Respironics). The cardiac status was re-evaluated by the cardiologist and the drugs were optimized. Central venous pressure (CVP) was in the range of 8-12 cm of H2O. Echocardiogram showed no new wall motion abnormalities, preserved ejection fraction and no pericardial collection. A fiberoptic bronchoscopy did not reveal any obstructive lesion. No organisms were identified by microscopic examination or culture of the lavage fluid.

By 11th postoperative day he was reintubated due to worsening of respiratory distress with desaturation and bradycardia. Gas exchange was maintained on SIMV (15) + PS (10), PEEP (8 cm H2O) with a FiO2 of 0.7. He continued to be hemodynamically stable. The only biochemical abnormality was a mild elevation of blood urea (86 mg%) with a normal serum creatinine. The chest X-ray showed an opaque right hemi thorax with air alveologram and left lower zone infiltrates (Fig. 3). CT scan of the chest revealed ground glass opacification of right hemi thorax with shaggy nodules, basal consolidation with airbronchogram and bilateral minimal pleural effusion (Figs. 4, 5). After reasonable exclusion of cardiac failure, pulmonary venous obstruction, pulmonary infections and pulmonary embolism, other noninfectious mimics of pneumonia like drug induced lung disease was considered.

On reviewing the charts, it was found that patient was given a loading dose of amiodarone (5 mg / kg) followed by maintenance of 1.5 mg / kg for 24 hours. Then it was switched over to oral dose of 200 mg / day. Subsequently there was gradual deterioration in oxygenation from 5th POD (i.e. 48 hrs after administering amiodarone) requiring supplemental oxygen followed by non-invasive ventilatory support to maintain oxygenation. Chest X-ray also showed alveolar infiltrates from same day, which progressed to opacification of right hemi thorax by the 11th postoperative day. He never had temperature spikes during this period. There was no increase in sputum volume or purulence. The patient did not respond to empiric antibiotic and antifailure treatment. The possibility of hypersensitivity reaction to amiodarone with pneumonitis was considered and it was decided to discontinue the drug. Methyl prednisolone was
initiated in the dose of 60 mg IV Q8H. Other medications like carbidilol, furosemide, ACE inhibitors, digoxin, cefaperazone nebulised bronchodilators were continued as it was. There was dramatic improvement in the clinical as well as radiological status by 24 hours. He was weaned off the ventilator the following day and chest x ray showed fairly good resolution of infiltrates (Fig. 6). Thus the presumptive diagnosis of acute amiodarone toxicity was confirmed. In view of good clinical and radiological response with one-week course of methyl prednisolone and considering the postoperative status with severely impaired cardiac function and diabetes mellitus, it was decided to discontinue steroids and close observation was planned (Fig. 7).

Patient is under regular follow up. There is no clinical or radiological relapse at 10 months follow up.

Discussion

Amiodarone is a potent antiarrythmic drug, but with notorious adverse effects involving various organs like liver, neuromuscular system, thyroid, cornea, skin and lungs. Of these, the most dreaded complication is pulmonary and is seen in about 1.45% to 18% of the amiodarone treated patients. Though its potential to cause chronic toxicity is well recognized, little is known regarding the acute toxicity. The common clinical presentation is one of insidious onset of dyspnoea with non-productive cough, usually after 2 or more months of the start of the treatment. In this form, the pulmonary infiltrates are interstitial and is associated with daily dose of 400 mg / day or more. Acute presentation can be devastating, with sudden onset dyspnoea, cough, fever with patchy alveolar infiltrates on chest x-ray and may be seen in about one third of the cases within weeks of initiating the treatment. It can occur at low doses (<200 mg/day). Clinical manifestations are variable, including ARDS, bronchiolitis obliterans with organizing pneumonia, and there is a theoretical possibility of worsening bronchospasm and heart failure (via the beta adrenoreceptor blocking action and negative inotropic action of amiodarone respectively).

Toxicity is more common in elderly patients (> 40 years) with preexisting pulmonary disease (up to 9 times). There is a complex relationship between the dose and toxicity. Total dose of 140–230 gm is associated with clinically significant lung damage. The chance of toxicity is more with higher maintenance dose (400 mg /day), though the risk rises with increasing serum concentration, there is no concentration at which it is inevitable. The plasma concentrations tend to correlate with amiodarone doses administered during maintenance therapy. Plasma levels of amiodarone generally do not predict the occurrence of amiodarone pulmonary toxicity. Due to long half-life, the toxic effect may persist even after the discontinuation of the drug. The principal metabolite of amiodarone desethylamiodarone accumulates in peripheral tissues and provides a sustained release reservoir. It is presently unclear whether desethylamiodarone plays a role in the development of pulmonary toxicity. Higher desethylamiodarone plasma levels were found in patients who had amiodarone pulmonary toxicity compared with unaffected patients. The route of administration is more important as amiodarone accumulates more rapidly in the lung following intravenous administration. Lower the pretreatment DLCO, higher the risk of developing toxicity. It is believed that,
genetically determined reduced amiodarone excretion related to P—glycoprotein or cytochrome P 450 may also play a crucial role in inducing pulmonary toxicity.\(^\text{10}\)

Several competing mechanisms have been proposed to explain this pulmonary toxicity but with no conclusive evidence. Adaptive immune-mediated hypersensitivity and direct drug induced phospholipidosis.\(^\text{12}\)

There aren’t any characteristic radiological features to diagnose acute toxicity. Commonest pattern is that of asymmetric alveolar opacification with interstitial infiltrates. Other patterns include solitary or multiple masses and lobar or segmental consolidation. Pleural effusions may be seen together with other findings, but rarely alone. High attenuation density pleural and parenchymal lesion seen on HRCT is highly suggestive of chronic toxicity. Other findings include reticulonodular or alveolar infiltrates, in association with ground glass opacifications.\(^\text{13}\)

Pulmonary function testing usually reveals a restrictive pattern of disease; a decreased carbon monoxide diffusing capacity frequently is an early finding.\(^\text{10}\) Bronchoalveolar lavage (BAL) findings are also non-specific. Foamy (lipid laden) macrophages were earlier considered to be diagnostic of amiodarone toxicity; but later on such findings were noted in asymptomatic patient taking amiodarone. Hence the detection of these cells does not distinguish toxic from non—toxic patients. Amiodarone pneumonitis is considered unlikely if foam cells are not present in BAL. Neutrophils and lymphocyte counts are often increased in amiodarone pneumonitis when compared to normal (or nontoxic amiodarone patients), but they do not differ from those receiving amiodarone who have interstitial pneumonitis for other reasons. CD41/ CD81 ratio is depressed); however, these differences are not sufficiently reliable as to be diagnostically useful.\(^\text{10}\)

Histopathological findings in amiodarone toxicity can be distinctive depending on the clinical presentation. There may be characteristic lipid pneumonitis with lipid infiltration of endothelial and type 2 pneumocytes (reflects a defect in phospholipids turn over), cellular infiltration of alveolar spaces with foam laden macrophages and in some cases there may be septal thickening with or without mononuclear cell infiltrates to frank fibrosis. In ARDS type, diffuse alveolar damage with hylane membrane formation will be seen.\(^\text{14,15}\)

There is no gold standard diagnostic test. It is made based on suggestive clinical and radiographic findings, the exclusion of differential diagnostic alternatives, the demonstration of pulmonary function abnormalities compatible with amiodarone pulmonary toxicity, and typical foamy cells found in a lung biopsy specimen.

The clinical diagnosis of amiodarone-induced pulmonary toxicity\(^\text{16}\) requires two or more of the following criteria:

1. New onset of pulmonary symptoms such as dyspnoea, cough, or pleuritic chest pain
2. New chest radiographic abnormality such as an interstitial or alveolar infiltrate;
3. A decrease in the DLCO of 20% from the pretreatment value, or if none is available, a value less than 80% of predicted
4. Abnormal lung uptake with gallium-67 radioisotope
5. CD8+ lymphocytosis on BAL
6. Lung biopsy with IP, BOOP, DAD or fibrosis
7. Improvement in symptoms with drug discontinuation

Like in earlier case reports, these symptoms were initially attributed to pulmonary infection and were treated accordingly. As the patient did not show clinical and radiological response to this approach, other diagnostic possibilities like congestive failure, pulmonary embolism, atelectasis and organizing pneumonia due to non-infectious causes were considered. A presumptive diagnosis of acute amiodarone toxicity was entertained based on new onset of dyspnoea with cough, alveolar infiltrates on chest radiograph with rapid improvement of symptoms with drug discontinuation and by reasonable exclusion of other diagnostic contenders.

Most cases recover with cessation of amiodarone therapy. Efficacy of steroids, though commonly used is not proven. Anyhow it is recommended empirically in acute toxicity considering the severity of illness and the high mortality. Acute toxicity may be immune mediated and that could be the reason for excellent steroid response in this particular patient. Early treatment of bronchiolitis obliterans organising pneumonia with steroids is effective in more than 60% of subacute episodes.\(^\text{17}\) Considering the chance of relapse, at least 6 months course of steroids is recommended.\(^\text{18}\) Azathioprine and plasma exchange were tried successfully in one case.\(^\text{19}\) It might have reduced the immune complex levels as well as the serum level of amiodarone and DEA. The regression of infiltrates varies between 2 weeks to 18 months after discontinuation of the drug. Amiodarone induced pulmonary toxicity has a mortality rate of 30%.\(^\text{10}\)

Amiodarone has a potentially important, though under recognized, role in inducing an acute pulmonary toxicity in some patients, such as those undergoing cardiac surgery. The high concentration of oxygen administered during the perioperative period coupled with damage of intubation and ventilation and SIRS induced by cardiopulmonary bypass and consequent depletion of anti inflammatory mediators render the lung vulnerable to acute toxicity. Hence amiodarone should be used with utmost caution in surgical ICU patients. It can be seen as early as two days after the start of therapy and with low maintenance dose of 200 mg / day. The data on the diagnosis and intervention of acute toxicity is limited. The clinician has to depend on clinical skills rather than any definitive diagnostic tests or proven therapies. Pulmonary toxicity can be fatal. A high index of suspicion is often necessary in establishing the diagnosis of pulmonary toxicity, and most cases are reversible if detected early.

References

Abstract

Here we report a case of VACTERL ASSOCIATION in a twenty three years old married female patient primigravida with 3 months of amenorrhea admitted with history of fever and gastroenteritis along with congenital developmental defects such as scoliosis (V), small ventricular septal defect (C), right sided hemifacial dysmorphic features (right mandibular hypoplasia), small sized right sided kidney (R), bilateral hypoplastic thumb (L). For the diagnosis of VACTERL atleast three out of seven anomalies should be present while our patient had four anomalies.

Introduction

VACTERL association is a non-random association of birth defects. It specifically refers to the abnormalities in structures derived from the embryonic mesoderm.\(^1\) VACTERL is a mnemonic in which the letters V, A, C, T, E, R, and L each stand for one or more type of malformation:

- V = Vertebral anomalies; and
- A = Anal atresia;
- C = Cardiac defect, most often ventricular septal defect;
- TE = TracheoEsophageal fistula with esophageal atresia;
- R = Renal (kidney) abnormalities; and
- L = Limb abnormalities, most often radial dysplasia.\(^2\)

Defects of practically every organ system have been reported in association with VACTERL.\(^3\) This non random association has a birth prevalence varying from 1:3,500 to 1.6:10,000.\(^4\) It is seen more frequently in infants born to diabetic mothers and possibly occur in children whose mothers have taken the cholesterol-lowering statin drugs in the first trimester of pregnancy or adriamycin.\(^5\) It is rarely seen more than once in one family. The mode of inheritance is either X linked or autosomal recessive. The diagnosis is made if at least three of the seven defects mentioned above are present in an infant. Individuals with the VACTERL association do not typically have facial dysmorphic features, learning disability or abnormalities of growth, including head circumference. Many babies with VACTERL are born small and have difficulty with gaining weight although development and growth are usually normal.

Fig. 1: Roentgenogram of chest posteroanterior view showing scoliosis of thoracic vertebra with convexity to the right.

VACTERL Association

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Fig. 2: Photograph of the face showing right sided mandibular hypoplasia.

Fig. 3: Photograph of both the hands showing bilateral hypoplastic thumbs

intelligence in survivors is usually normal.

Case Report

A 23 years old married female, primigravida with 3 months of amenorrhea was admitted with history of fever with chills, headache and bodyache since 10 days and multiple episodes of vomiting, loose motion and giddiness since 3 days before admission to our hospital. There was no colicky abdominal pain, blood or mucus in the stool or bleeding / leaking per vagina. Patient had some abdominal surgery in the past but details were not available. Also patient had history of recurrent chest infection in childhood. On physical examination patient was having tachycardia, hypotension, pallor, alopecia areata and previous mid line vertical abdominal surgical scar mark. Also patient had some dysmorphic features since birth which were as follows : vertebral anomaly in form of scoliosis (V) (Fig. 1), webbed neck, right sided dysmorphic features (right mandibular hypoplasia) (Fig. 2) and bilateral hypoplastic thumb (L) (Fig. 3). Patient had loud S1, pansystolic murmur in left parasternal region grade III / VI, mild mental retardation (IQ = 60 – 65) and mild to moderate sensorineural hearing loss. Patient’s mother antenatal, intranatal and postnatal history was uneventful. Neither her mother had diabetes mellitus nor she received any lipid lowering drug or adriamycin. Patients other siblings were also normal. After birth also patient did not have difficulty in deglutition, breathing, cyanosis and passing motion.

On investigation she was found to have anaemia (Hb = 8.4) and abdominal ultrasound showed small sized right sided kidney with altered axis while rest of the investigations such as sugar, renal, liver function test and stool routine / microscopy were normal. Electrocardiogram showed sinus tachycardia with poor progression of R wave from V1 – V4. (Fig. 4 is her repeat electrocardiogram showing normal sinus rhythm, normal axis and poor progression of R wave from V1 – V4). Chest Roentgenogram done with abdominal shield showed scoliosis. 2D Echocardiography showed small muscular ventricular septal defect. Obstetrical ultrasound showed single live intrauterine gestation of average 15 weeks with no fetus abnormality.

Patient was hydrated adequately with intravenous fluids and treated with intravenous antibiotics such as Ceftriaxone, Metronidazole, antiemetics, antacids and oral haematinics. Radiologic imagings of hands could not be done to avoid radiation exposure to fetus. Patient responded well and discharged with advice to follow up in Antenatal care OPD. On her next visit to our medicine OPD (after 5 months of her discharge) we repeated her abdominal and Obstetric ultrasound which showed right sided hydronephrosis in addition to the previous finding of small sized right kidney (Figs. 5, 6, 7) with altered axis and single live intrauterine gestation of 32 weeks. The right sided hydronephrosis could be due to dextrorotation of gravid uterus.

Discussion

VACTERL association is a non random association of birth defect of structures derived from embryonic mesoderm . Defect of practically every organ system have been reported in association with VACTERL. Our patient had congenital developmental defects such as scoliosis (V), small muscular ventricular septal defect (C), right sided hemifacial dysmorphic
A case of Falciparum malaria complicated with symmetrical peripheral gangrene has been discussed. This association is not very common as reported in the literature. The special feature of this case is that the patient had bilateral hypoglossal nerve palsy and the signs of pyramidal tract involvement which improved after conservative management.

**Introduction**

Symmetrical peripheral gangrene is a rare clinical condition which manifests as symmetrical distal ischemic damage in two or more sites without any evidence of obstruction or vasculitis of the relevant artery. SPG was initially described in 1981 by Hutchinson. Though this has been reported mostly in cases with DIC but several cases has been reported in complicated Falciparum malaria also from different parts of the globe. Here we are discussing SPG in a case of Falciparum malaria with other clinical manifestations.

**Case Report**

A 30 yrs old lady, Mrs. B.B, MRD NO-00751/04, admitted at Department of Medicine, Assam Medical College and Hospital Dibrugarh, Assam, India on 12/01/2004 with complaints of fever with chills and rigors of 10 days duration, blackish discoloration of the toes and distal parts of both the feet, slurring of speech, difficulty in swallowing and weakness of the both the lower limbs of 5 days duration.

There was no history of loss consciousness, seizure, claudication pain, oral ulcers, malar rash, chest pain etc. She had 4 children, last child birth 6 months back without any history of abortion. She was treated by antibiotics for 2 days before shifting to this tertiary centre.

**References**


**Symmetrical Peripheral Gangrene and Neurological Manifestations in Plasmodium falciparum Malaria**

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![Renal ultrasonogram showing small sized right kidney with hydronephrosis and small cyst.](image-url)
On examination: The patient was found to have fever, pallor, mild dehydration and mild icterus. There was no cyanosis, oedema or clubbing. Pulse: 80/min, regular, normal volume and character without radio-radial or radio-femoral delay and all the peripheral pulses including up to dorsalis pedis were palpable bilaterally. Blood pressure was 110/70 mm of Hg; Respiratory rate was 24/min, regular, thoracoabdominal; JVP - normal.

Systemic examination: On Neurological examination, she had bilateral hypoglossal nerve palsy and signs of bilateral pyramidal tract lesion. There was no sensory deficit. On 4th day of hospital stay she developed seizure which was controlled with medication. The Cardiovascular, Respiratory and Abdominal system examination revealed no abnormality.

On local examination: There was blackish discoloration of all the toes and distal parts of both the feet which was cold, shrunken and dry with definite line of demarcation without any local rise of temperature or ulceration (Figs. 1 and 2).

The following investigations done on admission Hb%-4.1gm/dl; Total WBC count - 4,700/cmm; DLC-N50,L43,M5 E2; ESR-16mm AEFH; RBC count-1.87 million/cmm; RBC morphology - Severe hypochromia and severe anisocytosis; Nucleated red cells-2/100 WBC. Peripheral blood smear for MP - Positive imf for mp - Shows trophozoites and gametocytes of PE (+++) paracheck for pf - Positive HB typing - HbE trait; HbsAg - Negative; Anti HCV - Negative; ANA-Negative. Platelet on smear - adequate; RBS - 117 mg/100ml; Serum Creatinine - 0.9mg/100ml; Blood Urea-77mg/100ml; Urine R/E - Alb - Trace, Sugar - Absent, Pus cells - Occasional, Cast-Absent; Platelet count - 1,20,000; Rheumatoid factor - non reactive; LE Cell - absent; Blood culture - sterile; Urine culture - Sterile; BT 1 in 45 sec; CT - 2 min 30 sec; PT-11 seconds. Serum Cholesterol - 186 mg/100ml; HDL-C - 49mg%; S. Triglyceride - 252 mg%.

CT of Brain: Atrophic changes; PA view chest - Normal; Echocardiography shows normal study. Doppler study up to dorsalis pedis artery-normal.

Final Diagnosis: Symmetrical Peripheral Gangrene, Bilateral hypoglossal nerve palsy, Bilateral pyramidal tract lesion due to Plasmodium Falciparum Malaria.

Discussion

Symmetrical peripheral gangrene (SPG) is a rare clinical condition manifesting with digital ischemic damage in two or more extremities, without evidence of obstruction or vasculitis of the large artery. Most of the cases reported in the literature were associated with DIC. It accompaniies infectious diseases of various etiologies (bacterial, viral or rickettsial origin) as well as low flow states such as cardiogenic or hypovolemic shock. Less commonly it is described as a complication of paraneoplastic syndrome, ergotism, polymyalgia rheumatica, Raynaud’s phenomenon, C-protein deficiency and perhaps, sickle cell disease. Vasopressor therapy may or may not be an aggravating factor in septic patients. In non-septic patients, administration of vasopressor agents clearly can precipitate the onset of SPG.

All the cases of SPG with malaria reported in literature had evidence of DIC. A functionally active but controlled coagulatory state exists in Falciparum malaria even in uncomplicated cases. Elevations in the concentration of FDPs reflecting the ongoing fibrinolysis have been documented. Heavy parasitaemia triggering the coagulation pathway, alterations in the lipid distribution across the surface membranes of the parasitized erythrocytes activating the intrinsic coagulation cascade, and activation of the complement system have all been proposed as likely mechanisms for DIC in Falciparum malaria. Sequestration of the parasitized erythrocytes in the microcirculation by molecular interactions with endothelial receptors, chiefly intracellular adhesion molecule-1 (ICAM-1) may occur. Heavy malarial parasitemia leads to sluggish microcirculation as parasitized red cells along with uninfected erythrocytes from micro aggregates (rosettes) and attach to different endothelial cell receptors (cytoadherence).

Final Diagnosis: Symmetrical Peripheral Gangrene, Bilateral hypoglossal nerve palsy, Bilateral pyramidal tract lesion due to Plasmodium Falciparum Malaria.
Double Chambered Right Ventricle with Ventricular Septal Defect Presenting in Adulthood


Abstract

Double-chambered right ventricle (DCRV) is a form of right ventricular outflow tract obstruction. It typically presents in childhood or adolescence. Only a handful of previous cases have been described in which DCRV occurred in adulthood. We report here a case of DCRV with ventricular septal defect (VSD) presenting in adulthood.

Introduction

Double-chambered right ventricle (DCRV) is a rare form of congenital heart disease characterized by right ventricle outflow tract obstruction by an anomalous muscle band that divides right ventricle (RV) into proximal high pressure and distal low pressure chamber. The anomaly is rare, seen in only 0.5–2% of all cases of congenital heart disease. It usually presents in childhood and adolescence with most reported cases in patients less than 20 years old. Occasionally, however, patients can present with this condition in adulthood. We report here a case of DCRV with ventricular septal defect (VSD) presenting...
Case Report

A 32-year male presented with one months history of continuous fever, productive cough, breathlessness and left lower chest pain which used to aggravate on deep inspiration. There was no history of hemoptysis, orthopnea and tubercular contact.

At presentation he was febrile with respiratory rate of 24/min, pulse rate 108/ min, blood pressure 112/74 mm of Hg. On general examination there was no cyanosis, clubbing, pedal oedema and JVP was normal. Respiratory system showed normal air entry with crepitations in the left basal region. CVS examination revealed normal heart sounds with systolic murmur of grade 4/6 best heard at left sternal border in third intercostal space, which aggravated on deep inspiration and was without any radiation. Per abdominal examination was within normal limits.

A tentative diagnosis of pulmonary stenosis with left sided pneumonitis was made and the patient was investigated. His Hb was 10.4gm/dl, TC – 22,400 with N81l16. RFT and LFT were within normal limits. Chest X-Ray showed cardiomegaly, pulmonary oligemia and a pneumonitic patch in left lower lobe. ECG showed RV hypertrophy with strain pattern and right axis deviation. Echocardiography revealed severe intra-RV obstruction due to anomalous fibromuscular band in RV outflow tract causing double chambered right ventricle (Fig. 1). There was a small perimembranous VSD with right to left shunt but no vegetation/oscillating mass over heart valves or in vicinity of VSD. Doppler derived pressure gradient across the bundle was 116 mm Hg with turbulent flow (Fig. 2). 64-slice-CT angiography of thorax revealed a muscular band partly dividing RV into two chambers with dilated RA and RV with RV hypertrophy along with reduced pulmonary artery calibre and relative oligemia of lung fields (Fig. 3).

A final diagnosis of DCRV with VSD with pneumonitis was made. The patient was started on antibiotics and supportive therapy and he became afebrile after 4 days. Subsequently the patient was referred to higher centre for further management.

Discussion

DCRV is a rare form of congenital heart disease in which right ventricle is divided into a high pressure inlet portion and a low pressure outlet portion by an anomalous muscle bundle. Localisation of the obstructive muscle varies and may be either high (or horizontal), adjacent to the pulmonary valve, or low (or oblique), close to the apex. The anomaly is rare, seen in only 0.5–2% of all cases of congenital heart disease. Most cases of DCRV are diagnosed during childhood, and very few reports include cases in which the patients present in adulthood. DCRV is exceptionally rare as an isolated anomaly. Most commonly it is associated with a membranous type VSD.

The correct diagnosis of DCRV is rarely made clinically and confused clinically with tetralogy of Fallot or isolated infundibular stenosis. Because of the unique nature of this anomaly, diagnostic problems are encountered which, if not properly recognized, may lead to inappropriate surgical
treatment. Transthoracic echocardiogram although diagnostic sometime fails to demonstrate anomalous muscle band. DCRV should strongly suspected in ECHO if RV hypertrophy is present in absence of infundibular hypertrophy or valvular pulmonary stenosis. MRI or HRCT demonstrates exact anatomy of anomalous muscle bundle and help in confirmation of diagnosis.

Surgical intervention should be considered in all patients who are symptomatic or have peak gradients of ≥50 mm Hg. The surgical approach resects the muscle bundles and repairs any associated lesions. Most patients do well after surgical intervention and lead fairly unrestricted lives. There is little recurrence of obstruction after adequate surgical repair. There have been isolated attempts at percutaneous alcohol ablation of the conal branch from the right coronary artery and the use of balloon dilatation. These options may be considered in patients who are not otherwise fit for surgical treatment.

References


Unusual Presentation of Brain Abscess with Uncommon Organism in an Immunocompetent Person


Abstract

Multiple brain abscesses due to Listeria monocytogenes was detected in a 55 year old immunocompetent person who had history of progressive weakness for last three months with difficulty in micturition and abnormal behavior for the same duration. The patient was diagnosed six months back as tubercular meningitis and was put on ATD (now in continuation phase). After being diagnosed with Listeria, the patient was put on antibiotic therapy and responded dramatically. The abscess, by virtue of being relatively large and superficially located, was drained surgically.

Case Report

A 55 year old nondiabetic nonalcoholic male patient was apparently well 2 months back, when he noticed weakness of his left lower limb followed by gradual involvement of all 4 limbs over a span of 15 days. Although there were no sensory symptoms, patient had difficulty in micturition in the form of hesitancy, urgency and sense of incomplete evacuation of bladder. There was also history of deviation of angle of mouth to right side. Detailed history taking revealed altered behaviour in the form of forgetfulness, emotional lability and urination in inappropriate places. There were two episodes suggestive of GTCS during the course of illness. Significant past history, included h/o of hospitalization six months back with complaints of altered sensorium which was provisionally diagnosed as TB meningitis and patient was put on ATD. The patient showed gradual improvement with this treatment for the initial three months and again started deteriorating over the subsequent period. There was no history of fever, cough, haemoptysis, sexual promiscuity, ear discharge. There was no documented cardiac disease or symptoms suggestive of CVS abnormality. On examination patient was drowsy. General survey was essentially normal except for pallor. Detailed nervous system examination revealed left sided upper motor neuron type of facial palsy with features of spastic quadriparesis without any sensory abnormality. Bilateral papilloedema was detected on opthalmoscopic examination.

Investigations

CBC- Hb 12.3,TLC 12,200 N-82, L-17, E-1. Platelets-adequate FBS-67mg% Bil-0.8 SGPT-16,SGOT-57,Alk phos-100,Al-3.7gm%,Gl-4.5gm% HIV1 and HIV2 non reactive

Fig. 1: Multiple ring enhancing lesions, multiple brain abscess
Clinical Course

On admission patient was advised to continue ATD (continuation phase), but his condition failed to improve. Subsequently, CT scan of brain was done which showed multiple ring enhancing lesions which had the possibility of being multiple brain abscesses (Fig. 1). Antibiotic therapy was started with a regimen of ceftriaxone+ vancomycin+gentamicin. His condition gradually improved with medication. We then went for an MRI spectroscopy, which confirmed our initial diagnosis of multiple brain abscesses. A thorough E.N.T check up was done to exclude any ear pathology. His HIV screening test was negative and echocardiography was within normal limits. After fourteen days of antibiotic therapy, a repeat CT scan of brain was obtained which showed (Fig. 2) that barring the largest one, the rest of the abscesses had diminished in size. Neurosurgical consultation was taken for drainage of the abscess and eventually large amount of pus was drained (60ml).Gram stain, AF B stain and culture of the pus revealed the causative organism to be Listeria monocytogenes. Based on the culture report, we substituted ceftriaxone inj with ampicillin inj in the treatment regime. The patient showed remarkable improvement and was eventually discharged in a stable condition. Subsequent follow up visits found him to be doing well.

Discussion

Listeriosis is a food-borne disease and varying presentations include febrile diarrhea, bacteremia, meningoencephalitis and brain abscesses. Other presentations, such as endocarditis, are less frequent. Although L. monocytogenes meningoencephalitis is a common complication, less than 1% of patients with listeriosis bacteriemia develop a brain abscess or multiple abscesses. These patients commonly have impaired cell-mediated immunity, for example in the course of an HIV infection or after organ transplantation, or other risk factors such as older age, alcoholism, or diabetes. Patients with brain abscesses most commonly present with headaches, but changes in mental status, focal neurologic deficits, and seizures, are also observed. MRI is the most sensitive diagnostic tool and the preferred method of diagnosis for brain abscesses. Determination of the etiology requires fine-needle aspiration for isolation of Listeria spp. by culture, or molecular techniques such as polymerase chain reaction. The presence of Gram-positive rods in a carefully examined cerebrospinal or abscess fluid can be helpful for rapid diagnosis, but can represent contaminating diphtheroids. Blood cultures are a sensitive diagnostic tool for L. monocytogenes meningoencephalitis and can be valuable for the diagnosis of L. monocytogenes brain abscesses. Differential diagnoses of abscess include the following: (i) brain metastases that are usually multifocal and occur at the gray–white junction with a ring-like contrast enhancement on imaging; (ii) lymphoma showing dense homogeneous contrast enhancement; (iii) glioma; (iv) vascular diseases, such as infarction and arteriovenous malformation; (v) cysticercosis; (vi) multiple tuberculosis and (vii) toxoplasmosis. In our patient, intensive work up was done to eliminate these differentials as far as practicable. Listeria monocytogenes is the only Listeria species that infects humans. Immunity to Listeria relies mainly on T-cell lymphokine activation of macrophages, which clear Listeria from the blood. Interleukin (IL)-18 appears to play a pivotal role in protection against Listeria by enhancing bacterial clearance. Among 186 cases of listeriosis reviewed in 1978 by Nieman and Lorber, 102 patients (55%) had meningitis and 2 (1%) had brain abscesses. In 1986, Dec and Lorber reviewed 14 cases of listerial brain abscess and concluded that surgical intervention is indicated for establishing the diagnosis and treatment in the presence of a mass effect. Our case is an unique example of an unusual presentation of listerial brain abscess because

1. Listeria brain abscess usually occurs in immunocompromised patients but in our patient no apparent cause of immunosuppression could be detected. The patient was initially diagnosed as a case of neutrotuberculosis and was on ATD (continuation phase). We have excluded other possible causes of immunosuppression.

2. The presentation of the patient was without fever and the first manifestations of his infection were seizures. Of the 14 patients in the Dec and Lorber review, only one was afebrile at presentation, and seizures as a presenting sign was seen in a mere couple of the remaining reported cases.

It is relatively difficult to suspect Listeria infection on clinical ground but there are some clinical hints that might guide us like a) patients belonging to vulnerable groups like pregnant women, elderly persons, neonates, immunocompromised like HIV, post organ transplant, cancer, treatment with glucocorticoid, diabetes, alcoholism, renal disease, rheumatological disease and iron overload b) We can suspect Listeria as a causative organism in subcortical brain abscess with features of meningitis in elderly c) Monocytosis in the peripheral blood picture could be a hint d)The presentation might be biphasic with prodrome of fever and headache followed by cranial nerve palsy, hemiparetic and hemisensory deficits.

The purpose of presenting the case is that there may be some molecular defects in the cells of the immune system which come to the fore with age. Moreover, tuberculosis might provoke some immune dysregulation which predisposes the affected patients...
to such unusual infections.

References

CA Lung Presenting with Vasculitis

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Abstract
A 67 year old man presented to us with petechial skin rash and blebs on the legs. He was on anti-tuberculous treatment for right sided pleural effusion since 1 month. A provisional diagnosis of Rifampicin induced drug rash was made and his effusion reaspirated and sent for AFB and cytology. The pleural fluid cytology demonstrated metastasis of adenocarcinoma and biopsy of skin lesion confirmed vasculitis. There was no thrombocytopenia. CT thorax showed right lower lobe mass with right sided pleural effusion. Thus a final diagnosis of skin manifestation of carcinoma of lung was made. This was a Paraneoplastic syndrome.

Introduction
CA lung is known to present with involvement of other systems of the body. Skin manifestations have been described as one of these paraneoplastic syndromes. Vasculitis is a rare skin lesion associated with CA lung and our patient presented with petechial lesions and bullae with punched out ulcers on both shins and dorsal aspect of feet. He presented with right pleural effusion which was diagnosed as adenocarcinoma.

Case
A 67 yr old, non diabetic man presented with swelling and rash on both lower limbs and blebs with watery discharge since 8 days. He also had cough with breathlessness and right side chest pain. He had a history of right pleural aspiration done 1 month back and was on antitubercular treatment since a month.

Cutaneous Examination: of lower limbs revealed multiple petechiae with few showing vesicles. The dorsum of left foot revealed large flaccid bulla. The medial aspect of left ankle showed small punched out ulcers surrounded by necrotic tissue and flaccid bullae. There was surrounding erythema with underlying edema. Similar features over right foot with multiple small necrotic ulcers around ankle were present.

The Clinical Impression was vasculitic ulcers of connective tissue disorder or malignant etiology. The Palms, oral cavity and genitals were normal.

Chest X Ray: was suggestive of - right sided pleural effusion (Fig. 1). His haemoglobin was 11 gm% with total leucocyte count 7,400/ mm cube and platelet 2,38,000/micro litre. Pleural Fluid Cytology was suggestive of metastatic Adenocarcinoma.

CT Chest: Abnormal hypo-enhancing lesions in right lower lobe suspicious of primary bronchogenic neoplasm with right sided pleural effusion (Fig. 2).

Skin Biopsy Results: The epidermis shows spongiotic changes resulting in an intraepidermal split. The blood vessels immediately below show features of vessel wall damage which include extravasated RBC’s and deposition of fibrinoid material within vessel wall, upper reticular dermal vessel shows perivascular inflammatory cell infiltrate (predominantly lymphocyte). These features are consistent with vasculitis.

The diagnosis of Adenocarcinoma right lung [NSCLC] stage 3 b [wet] with vasculitis [paraneoplastic] was made.

Discussion
On respiratory examination, there were decreased breath sounds on right side.

Cutaneous Examination: of lower limbs revealed multiple petechiae with few showing vesicles. The dorsum of left foot revealed large flaccid bulla. The medial aspect of left ankle showed small punched out ulcers surrounded by necrotic tissue and flaccid bullae. There was surrounding erythema with underlying edema. Similar features over right foot with multiple small necrotic ulcers around ankle were present.

Changes in the skin can be a marker for internal malignant neoplasm. Some cutaneous manifestations of cancer are paraneoplastic syndromes i.e. dermatoses secondary to cancer. A true paraneoplastic syndrome has 2 essential criteria:
1. Dermatosis develops only after development of the malignant tumor.
2. Both dermatosis and malignant tumor follow a parallel
Multiple Myeloma Presenting with Hepatosplenomegaly

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Abstract
Multiple myeloma, a clonal plasma cell disorder, commonly affects adults above 50 years age and accounts for about 10% of all hematological malignancies. Anemia, bone pains, renal failure are the most common symptoms at presentation. Though extra-medullary extra-osseous disease is well known in the course of the disease, initial presentation with extramedullary disease alone is rare. Such presentation may represent poor biology of the disease and/or advanced stage. Early diagnosis and treatment may improve outcomes. We herewith report the case of a 43 year old lady who presented with hepatosplenomegaly, without any classical manifestations of multiple myeloma and discuss the relevant literature.

Case Report
A 43-year-old lady presented with vague upper abdominal pain of 2 months duration, unrelated to food intake or posture. She also complained of exertional breathlessness and loss of appetite of a month’s duration. She had no history of cough, sputum, chest pain, orthopnoea, bleeding manifestations; her bowel and micturition habits were normal. She had no other co-morbid illnesses. Examination revealed performance status- 2, pallor, massive hepatomegaly measuring 12 cm below the right costal margin in the mid clavicular line and splenomegaly of 3 cm below the left costal margin. Rest of the examination was unremarkable. Complete blood counts showed hemoglobin of 5.9 gm/dl, a total leukocyte count of 11,400 cells/mcl; platelet count of 60,000/ mcl and the peripheral blood smear examination

Fig. 2 : CT Thorax showing abnormal hypo enhancing lesion in right lower lobe with right sided pleural effusion

course in that the removal of the cancer results in the clearing of the dermatosis and recurrence of the cancer causes relapse

Often skin involvement in cancer, due to vascular and blood abnormality include flushing, palmer erythema, telangiectasia, cutaneous ischemia and thrombophlebitis and vasculitis

Leukocytoclastic vasculitis such as can be seen in Henoch-Schonlein purpura is very rarely associated with malignant neoplasms like squamous cell Ca of bronchus, renal carcinoma, leukaemia and lymphoma. Vasculitis can present as palpable purpuric eruptions in patients of cancer. Idiopathic thrombocytopenic purpura in malignant disease is often due to lymphoma. Purpura in cancer can result from a wide variety of mechanisms- thrombocytopenia, consumption coagulopathy, hyper or dysglobulinemia, vascular fragility and vasculitis.

Migratory superficial thrombophlebitis is seen in association with cancer of lung, liver, bowel, gall bladder, ovary and lymphoma and leukaemia. The migratory nature of the thrombophlebitis may be due to the generalized hypercoagulable state.

Paraneoplastic syndrome leading to colour changes of skin secondary to deposition of substances in skin include icterus, secondary to obstruction of bileduct or intrahepatic obstruction, xanthomas in multiple myeloma and leukaemia.

Acanthosis nigricans is usually of sudden onset and rapid progression in malignancy, usually causing diffuse keratoderma of palms and soles. Erythoderma, a diffuse erythema of skin surface is also known to be associated with Ca lung, liver, prostrate. Dermatomyositis and systemic scleroderma are also known to be associated with Ca lung.

Our patient presented with skin lesions and a right sided pleural effusion which was being treated with anti tubercular drugs INH, rifampicin, pyrazinamide, and ethambutol. The skin lesions included purpuric lesions, bullae and punched out necrotic ulcers on both feet. A diagnostic dilemma existed where we suspected the lesions to be rifampicin induced thrombocytopenic purpura, which was excluded since there was no thrombocytopenia.

Dermatological examination confirmed the presence of vasculitis either of Henoch Schonlein purpura type or of connective tissue disease or secondary to malignancy. On re aspiration of pleural fluid and cytological examination there was a positive result for metastatic adenocarcinoma. A CT scan then revealed a right lower lobe mass

Vasculitis associated with Ca lung is quite rare, hence we present this case.

Abbreviations
Ca- carcinoma, NSCLC-non small cell lung carcinoma INH-isoniazid.

References
showed plasma cells of 10%, suggesting the possibility of plasma cell neoplasm. Her serum creatinine and serum transaminases were normal; serum calcium was 8.6 mg/dl; and her serum globulin was 5.6 gm/dl; serum lactate dehydrogenase was 1213 u/l. A serum and urine immune fixation revealed M spike of 3.07 gm/dl with Ig-G-kappa type monoclonal immunoglobulin. A bone marrow examination showed prominence of plasma cells amounting to 85% of marrow-nucleated cells with all of them showing prominent nucleoli (Fig. 1); cytogenetic analysis was normal. Serum beta 2 microglobulin was 8.077 ng/ml; skeletal survey was unremarkable. She was diagnosed to have Multiple myeloma ISS- stage-III. A contrast enhanced CT scan of the abdomen showed gross hepato-splenomegaly with para-aortic and retroperitoneal lymphadenopathy (Fig. 2).

She was started on Thalidomide 100 mg/day and oral dexamethasone 40 mg per day for four consecutive days, repeated every other week. After initial two weeks of therapy, she responded to treatment with regression in hepatomegaly and the platelet count improved to 2.2 lakhs/mcl. She developed deep vein thrombosis of the right lower limb on 28th day of first cycle, hence Thalidomide was discontinued and she was started on anticoagulation and supportive care. In view of DVT, therapy was changed to Bortezomib and Dexamethasone. On Day 11 of first cycle of Bortezomib + Dexamethasone, she presented with a performance status of 3, increasing abdominal discomfort and a clinical examination revealed massive hepatomegaly 18 cm below the right costal margin. An ultrasonogram of the abdomen showed hepatomegaly with multiple space-occupying lesions in the liver, multiple abdominal lymph nodes, and splenic lesions (Fig. 3). Her general condition deteriorated and she developed coagulopathy and drowsiness secondary to hepatic insufficiency and she succumbed to the disease. A post mortem liver biopsy was performed which revealed hepatic sinusoidal infiltration with clonal plasma cells which stained positive for kappa light chain. (Figs. 4, 5).

**Discussion**

The reported incidence of liver involvement in multiple myeloma is 25-40 % cases in retrospective studies. However, clinically palpable hepatomegaly at the time of diagnosis of multiple myeloma is rare and the incidence has not been reported, though hepatomegaly at diagnosis or at some time later during the course was noticed in about 13% patients in one study. The transient improvement of hepatomegaly and rapid recurrence with nodular pattern of involvement following treatment with Thalidomide represents the poor response of extramedullary disease to this drug. Lack of responses of extra-medullary disease to thalidomide has been observed in some studies and it has been postulated that Thalidomide could select tumor clones with more aggressive biologic characteristics like secondary cytogenetic abnormalities, p53 mutations etc. In spite of clinically palpable hepatomegaly, our patient did not have significant hepatic...
dysfunction till the end where coagulopathy and coma have set in due to liver dysfunction. Splenic involvement in myeloma has been reported in up to 18% in autopsy series but the incidence of clinically palpable splenomegaly is not reported. Tumor nodules and diffuse infiltration of spleen by plasma cells has been described at autopsy. Retroperitoneal lymphnode enlargement has been noticed in two imaging studies.

Sinusoidal, portal and mixed patterns of plasma cell infiltration of liver has been described at histopathology and sinusoidal pattern was observed most frequently; our patient had a mixed pattern of infiltration which is the second most frequent pattern described. The rapidly fatal course of our patient may be multifactorial starting with a possible delay in diagnosis due to unusual clinical features at presentation, a thrombotic tendency to Thalidomide and biology of the disease itself.

Involvement of liver alone is rare and usually, extramedullary involvement by multiple myeloma involves several organs including para-nasal sinuses, alimentary canal, retro-peritoneum, peri-nephric region, spleen, skin and soft tissues. Liver involvement is not established as a prognostic marker and hence performing liver biopsy is not recommended. Treatment decisions are to be made on the standard existing guidelines and further studies to look at the prognostic significance of extramedullary plasma cell disease are to be made.

References

Pulmonary Embolism after Long Duration Rail Travel: Economy Class Syndrome or Rail Coach Syndrome
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Abstract
Pulmonary embolism after long duration air travel is well described. However it can also occur following a long duration rail or road transport. We present a case of 43 year old male who developed deep venous thrombosis and acute pulmonary embolism after a long rail journey. We propose to call it as rail coach syndrome and stress the need for taking the same preventive measures as recommended for airline passengers.

Introduction
Economy class syndrome describes the situation of patients who suffer pulmonary embolism following prolonged immobility in a long distance flight. But this syndrome is largely forgotten in patients who take a long overland journey especially in hot humid environment. We aim to highlight this in our case report.

Case Report
A 43 year old male, non-smoker, non-diabetic, non-hypertensive presented with complaints of breathlessness. He
had been travelling in second class non-air conditioned rail coach from Kerala to Ludhiana (Punjab). On routine clinical examination, no abnormality could be detected.

ECG showed T-wave inversion in leads II, III, aVF and V1-V4 suggestive of acute coronary syndrome (Fig. 1). However his 2D echocardiography done revealed dilated right atrium and right ventricle with hypokinetic right ventricular free wall. The main pulmonary artery was dilated and a mobile thrombus was noticed in right pulmonary artery (Fig. 2). Venous ultrasonography of the lower limbs revealed a large thrombus in right popliteal vein (Fig. 3).

The patient was thrombolysed with streptokinase infusion over 24 hours and subsequently low molecular weight heparin was given for 5 days and acitrom added. The patient improved with thrombolysis and repeat echocardiography done revealed clearance of thrombus from the right pulmonary artery with regression of dilated right atrium and right ventricle.

Discussion

Pulmonary embolism and deep venous thrombosis can occur after long duration rail travel in second class coach due to a combination of factors including prolonged immobilisation leading to venous stasis1,2 and dehydration leading to hemoconcentration.3 ECG changes can be quite deceptive. However a detailed history taking, high index of suspicion and a careful echocardiography study can clinch the diagnosis in most of the cases.

Although the literature emphasizes the risk of deep venous thrombosis and pulmonary embolism during air travel, our case, as suggested by others,4,5 demonstrates that this risk is not restricted to air transport alone.

In conclusion pulmonary thromboembolism should be considered in a patient presenting with breathlessness after a prolonged journey by any means of travel and preventive measures recommended for airline passengers should be extended to users of other means of transport.

References