Case Report of Hepatopulmonary Syndrome in a Cirrhotic Patient: The Anaesthetic Concern in Orthopedic Surgery

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ABSTRACT

INTRODUCTION: Hepatopulmonary syndrome (HPS) is a pulmonary complication observed in patients with chronic liver disease, attributable to an intrapulmonary vascular dilatation that induces hypoxemia. Every anesthetist should be aware of the possibility of HPS and of related peri-operative risks in hypoxemic patients with liver disease.

A 57 years old male patient with chronic liver disease and hepatopulmonary syndrome, secondary polycythaemia, tubercular brain abscess (on antitubercular treatment and phenytoin for seizure disorder) on domiciliary oxygen had intertronchenteric fracture of left hip. He underwent closed proximal femur nailing under subarachnoid block with high risk consent. Intraoperative period was uneventful. On second post operative day he developed hepatic encephalopathy grade II –III and was managed with laxatives, diuretics and other supportive measures. His symptoms improved, and medication were adjusted accordingly and discharged on 10th post op day.

CONCLUSION: An accurate identification of HPS and proper evaluation is important to prepare the patient and the anesthetist himself for a safe anesthesia during the peri-operative period.

KEY WORDS: Hepatopulmonary syndrome, hepatic encephalopathy, orthopedic surgery, spinal anesthesia.

INTRODUCTION

Hepatopulmonary syndrome (HPS) is characterized by the triad of liver disease, intrapulmonary vascular dilatations and arterial deoxygenation.1 Cirrhosis of liver irrespective of its etiology is the commonest disorder leading to HPS but it can occur in patient with mild liver dysfunction. Platypnea (dyspnea worse on sitting position) and orthodeoxia (arterial deoxygenation on sitting from supine position) in patient with chronic liver disease are highly specific for HPS.2 The intravascular pulmonary dilations can be of two types- type I is diffuse dilations of pulmonary vasculatures at pre-capillary level near to gas exchange units of lung whereas type II is localized, large arteriovenous communications situated distant to gas exchange unit and responds poorly to supplemental oxygen.3 Diagnosis of HPS in a patient with liver disease requires demonstration of arterial hypoxia and intrapulmonary shunts. It is staged as mild, moderate, severe and very severe depending on PaO2.1,2 There is still search for pharmacological agent to treat HPS though a few agents have been tried with variable success. When surgery is required in patients with HPS anesthetist should be aware of issues of oxygenation, fluid status, mechanical ventilation, coagulopathy, altered drug metabolism and risk of infection.4 So we report our experience of a patient with HPS who underwent orthopaedic surgery.

CASE REPORT

A 57 years old male patient, diagnosed case of cirrhosis of liver with hepatopulmonary syndrome presented with complain of alleged history of fall on the ground. He had intertrochanteric fracture of left hip. He was on domiciliary oxygen therapy and was also getting
antitubercular drugs for tubercular brain abscess and phenytoin for seizure disorders.

He was conscious, cooperative, oriented to time place and person. He was cyanosed and had clubbing. His peripheral oxygen saturation was 70% with oxygen and 66% on room air on sitting but his oxygen saturation improved on lying down up to 83% with oxygen. He was afibrile with pulse rate 88/min, BP 130/80 mmHg and respiratory rate 22/min. His chest examination was normal. Evaluation of cardiovascular system and abdomen did not reveal any abnormalities.

Laboratory reports: CBC : Normal, Haemoglobin-15.8, Platelets-150000 per cubic mm, serum bilirubin Total-6.1 and Conjugated – 1.9, PT 25 and INR- 1.78. Renal function tests and serum electrolytes were within normal range. His arterial blood gas report on room air was as follow: pH 7.475, PCO2: 30.4mmof Hg, PO2: 45.6mmHg, HCO3 22.1 mmol/L, alevolar arterial (A-a) gradient was 47.9mm of Hg.

Chest X-Ray showed prominent hilar shadow and ECG was normal with sinus rhythm with heart rate 82/min. Echocardiography revealed normal systolic function (ejection fraction of 60%) and normal cardiac chambers with pulmonary arterial systolic pressure of 20mm Hg.

CT Chest findings were cirrhotic liver with portal hypertension, large lienorenal shunt with dilated left renal vein and splenomegaly

X-ray of pelvis and left leg was done which shows that intertrochanteric fracture of left hip,

He belonged to ASA-PS III and high risk consent was taken for closed proximal femur nailing under subarachnoid block for the next day. He received four units of fresh frozen plasma. Repeat INR was 1.29.

Preloading with 500ml of ringer’s lactate solution was done. With all standard monitoring and under all aseptic precautions, subarachnoid block was given with 2.8 ml injection Bupivacaine (Heavy) 0.5% and 0.2 ml (20 microgram) of fentanyl with 27 G pencil point needle and T8 level block was achieved. O2 supplementation was done with ventury mask (FiO2 0.6%) and SpO2 was 80-84% during whole intra operative period. Intraoperatively BP dropped once to 80/50 mm Hg which was managed with Mephenteramine 6+6 mg and infusion of 500ml colloid solution. He received 1500 ml fluid during intraoperative period and urine output was 300ml. Heart rate was 62 to 88/ minute. After two hours of surgery the patient was shifted to surgical ICU.
–III on 2nd post-operative day and was managed with laxatives, diuretics, calcium supplementation and other supportive measures. Postoperative pain was managed with ketorolac injection. He was shifted to ward on the 4th post-operative day when started recovering from encephalopathy and discharged on 10th post-operative day with significant recovery.

**DISCUSSION**

The term HPS was first described in 1977 by Kennedy and Knudson\(^{13}\). While its pathogenesis not yet completely clear, HPS is widely prevalent (4% to 32%) among cirrhotic patients\(^{14}\). The arterial deoxygenation in a patient due to intrapulmonary vascular dilatations with evidence of liver disease constitutes hepatopulmonary syndrome (HPS). A detailed history and thorough clinical examination is essential and other causes of hypoxia such as pleural effusion, ascites, pulmonary atelectasis, ascites and co-existing cardiorespiratory diseases need to be excluded. \(^3\) Presence of digital clubbing, spider naevi, cyanosis and severe hypoxia (PaO2 < 60 mmHg) are strong pointers of presence of HPS, \(^1\) most of these clinical features are present in our case.

The characteristic clinical features are worsening dyspnea on sitting or standing position (platypnea) which improves on recumbency and orthodeoxia defined as decrease in PaO2 by ≥ 5% or ≥ 4mmHg from the supine to upright position. The syndrome is graded into mild, moderate, severe and very severe based on PaO2. \(^2\) The common pathological changes in the pulmonary circulation causing hypoxia are diffuse dilatation of pre-capillary and capillary vessels predominantly in lower two-thirds of lungs leading to decrease in diffusion of oxygen in the centre of large capillaries. The result is ventilation perfusion mismatch which worsens on erect posture due to effect of gravity. Less common are a few pleural and pulmonary arteriovenous shunts and portopulmonary venous communications. \(^5\) Various agents have been studied for the cause of vasodilatation. Larger quantities of nitric oxide, a potent vasodilator has been found in expired air in patients with HPS which normalizes after liver transplantation. \(^3\)

Diagnosis of pulmonary vasodilatation can be demonstrated either by contrast enhanced echocardiography (CEE) using intravenous agitated physiological saline producing microbubbles which are trapped in pulmonary vasculature under normal condition. In intracardiac right to left shunts they appear within three cardiac cycles into the left atrium whereas in HPS microbubbles are seen after third cycle. Another method is perfusion lung scanning with \(^{99m}\) technetium-labelled macroaggregated albumin but differentiation between intracardiac and pulmonary shunt is not possible. Pulmonary angiography is reserved for patients with severe hypoxia (PaO2 < 60 mmHg) and who respond poorly to 100% oxygen. \(^5,6\)

In our case HPS was diagnosed by hypoxaemia (PO2: 45.6mmHg) and intracardiac right to left shunt. It was proven by detection of saline microbubbles appearing in the left atrium after its injection in peripheral vein during contrast enhanced echocardiography.

Various pharmacological agents such as nitric oxide antagonists, somatostatin analogues, almitrine bimesylate have been tried but the results are inconsistent. \(^7,8\) Modest improvement in arterial oxygenation with garlic powder capsule when given daily for minimum of six months has been demonstrated. \(^5,9\) Orthotropic liver transplantation remains the only definitive treatment. \(^1,2\)

With the increasing prevalence of liver disease in the population and improved survival of these patients, a growing number of hepatopathic patients will undergo surgery and consequently will require attention of both surgeons and anesthesiologists. Every anesthesiologist should be aware of the possibility of HPS and of related peri-operative risks in hypoxemic patients with liver disease. As no clearly effective medical therapy for HPS has been demonstrated, the anesthetist has no specific medication available for pre-operative use in these patients before and during surgery. Surgical procedures- both hepatic and extrahepatic in patients with HPS is associated with an increased perioperative risk. \(^10,11\) Lower pre-operative oxygenation, use of inhalational general anesthesia, volume overload and pulmonary atelectasis may precipitate respiratory failure. Patients frequently need prolonged mechanical ventilation. \(^4\) Use of non-invasive ventilation following extubation has shown better oxygenation than oxygen therapy by mask. \(^12\) In our case, surgery was performed in regional anesthesia to minimize the perioperative risk of respiratory failure.
CONCLUSION

Every anesthetist should be aware of the possibility of HPS in hypoxic patients with liver disease. Identification of HPS is important for the planning of a rational anesthesiological technique and for the safety of the patient during the overall peri-operative period. Pre-operative clinical evaluation is the first step in the management of hypoxic patients undergoing surgery. It helps the anesthetist to select pre-operative diagnostic tests, to identify operative risks, to obtain informed consent and to prepare the patient and the anesthetist himself for a safe anesthesia.

REFERENCES