Acute respiratory distress after liver transplantation in infants—looking beyond infection and hepatopulmonary syndrome: A brief report

Sir,

Acute respiratory distress after liver transplantation (LT) in infants has varied aetiology. Clinicians, at times, are so much intrigued by common complications, that rare aetiologies may be overlooked.

A nine-month old, 6-kg baby boy with biliary atresia-associated cirrhosis (paediatric end-stage liver disease score 28), decompensated with jaundice, encephalopathy, coagulopathy and hepatopulmonary syndrome (HPS) (alveolar-arterial gradient 22, PaO_2 79 mmHg) presented for living donor liver transplantation (LDLT). He had a past history of failure to thrive and recurrent respiratory distress. The blood biochemistry values included haemoglobin 8.1 gm/dl, total leucocyte count 33800/mm^3, total bilirubin 22.46 mg/dl, albumin 2.9 gm/dl, and international normalised ratio 3.09. The chest radiogram showed an enlarged cardiac shadow and clear lung fields. Echocardiography showed mild left ventricle (LV) dilatation with left ventricular ejection fraction of 62%. Saline contrast echocardiography showed presence of pulmonary arteriovenous malformation. Computed tomography showed multiple intrahepatic abscesses and clear lung fields.

After preoperative optimisation, he underwent successful LDLT. The intensive care unit course was uneventful until postoperative day (POD) 7, when he developed acute onset dyspnoea, tachypnoea (respiratory rate 65/min), desaturation (peripheral oxygen saturation 80%), tachycardia, hypercapnia ($PaCO_2$-70 mmHg), drowsiness, diaphoresis with blood pressure (BP) 134/75 mmHg, but afebrile. Auscultation revealed pulmonary crackles. He was intubated and started on mechanical ventilation. Differential diagnoses considered were acute respiratory distress syndrome/sepsis, worsening HPS and new-onset pleural effusion. Blood, urine and endotracheal aspirate cultures were sent, and empirical antibiotics were started. Chest radiogram was suggestive of pulmonary oedema. He showed quick, remarkable improvement with ventilatory strategy and was weaned off ventilator after meeting extubation criteria on the next day. However, within 1 hour, he again developed respiratory distress. This time, non-invasive ventilation (NIV) was started along with intravenous furosemide 6 mg. It was noticed that the baby had developed de novo stage-2 accelerated hypertension (BP-143/96 mmHg). The systolic pressure was 33 mmHg more than the 99th percentile. Class-4 acute heart failure was diagnosed according to modified Ross classification. Echocardiography was consistent with preoperative findings except for newly developed mitral regurgitation. Infusions of labetalol and nitroglycerine were started, along with tablet amlodipine (5 mg BD) via Ryle's tube. Patient showed improvement in respiratory parameters once the BP was controlled [Figure 1]. Renal doppler, serum lactate and C-reactive protein were normal and cultures were sterile. Antimicrobials were stopped. Graft functions remained normal. NIV was replaced by high-flow nasal cannula while the baby's BP was gradually controlled to <95th percentile (103/56 mmHg). There were no more similar episodes and he was shifted to ward on POD 14 with regular immunosuppression and antihypertensive. BP was high during both events.

At the first event, we thought that hypertension was a sequela of distress but in fact, sedation led to BP normalisation and the heart failure improved rapidly. During the second event, we considered hypertension as a significant finding, which could have led to the current status of the baby and he quickly responded to antihypertensives and supportive therapy.

Hypertension and pulmonary oedema are known complications after adult LT, but rarely reported after paediatric LT.[1] In fact, BP measurement, though important, is often overlooked in small children.[2] Nevertheless, it is necessary to improve patient safety and quality of anaesthesia care.[3]

Hypertensive crisis presents commonly with neurological manifestations (46%), followed by cardiopulmonary manifestations (29%) presenting as dyspnoea and pulmonary oedema.[4] The management consists of treating the primary aetiology and phased control of BP. The initial 6-hour phase consists of lowering the BP to 25% of base level, whereas the next gradual phase lasting 24–48 hours

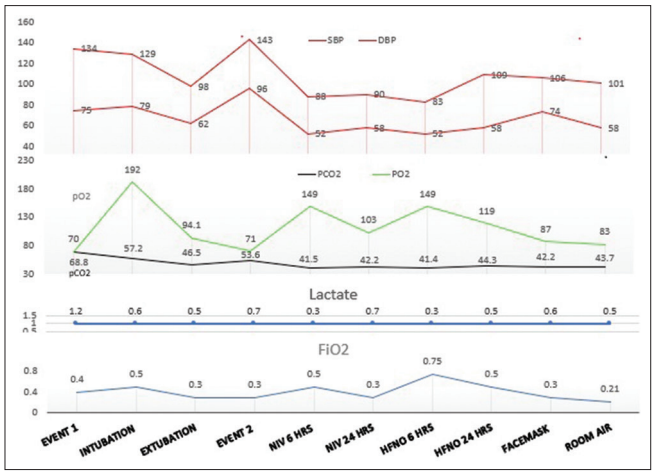

Figure 1: Parameters against time

targets BP to be <95th percentile.[5] The causes of de novo hypertension include normalisation of systemic vascular resistance after LT, effects of drugs (tacrolimus/steroids) and relative volume overload.[6]

Acute increase in afterload may lead to acute LV failure and pulmonary oedema.[7] This type of respiratory distress requires rapid differentiation from noncardiac causes (infection, worsening HPS, massive pleural effusion). Although early post-operative complications are common due to infections, immunological or technical aetiologies, once graft function is ensured to be normal, the threshold to suspect uncommon aetiologies, must be low for much-needed timely intervention.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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