PEDIATRIC ONCALL
CHILD HEALTH CARE

CASE REPORTS

CONGENITAL IDIOPATHIC NON-CHYLOUS PLEURAL EFFUSION PRESENTING WITH RESPIRATORY DISTRESS IN A PRETERM NEONATE: A CASE REPORT

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ABSTRACT

Congenital pleural effusion is a rare neonatal condition with an incidence of around 1 case per 12,000–15,000 births, that usually presents as chylothorax.[1] We report an unusual case of isolated non-chylous, unilateral pleural effusion in a neonate who developed respiratory distress at birth requiring mechanical ventilation and chest drainage. Pleural fluid analysis showed clear straw colored fluid with low triglycerides and proteins. A comprehensive workup including imaging, pleural fluid analysis, infection screen, and genetic studies failed to identify a definitive etiology. The baby was managed with therapeutic thoracentesis, respiratory support, and empirical antibiotics. The pleural effusion eventually resolved, and the infant was discharged in a good condition.

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Neonates, Pleural effusion, Chylothorax.

Case report

A 33+5 week gestation, 2390 grams male baby, born out of non-consanguineous marriage to a G3P1A1L1 mother was delivered on 29/04/2023 through caesarean section (indication: breech presentation with antenatal fetal distress) at a Government community health centre. APGAR scores at birth were 5, 7 and 7 at 1 min, 5 min and 10 min respectively. Baby had a weak cry at birth and required positive pressure ventilation and was referred to our tertiary care unit in view of respiratory distress and was initiated on CPAP support upon admission.

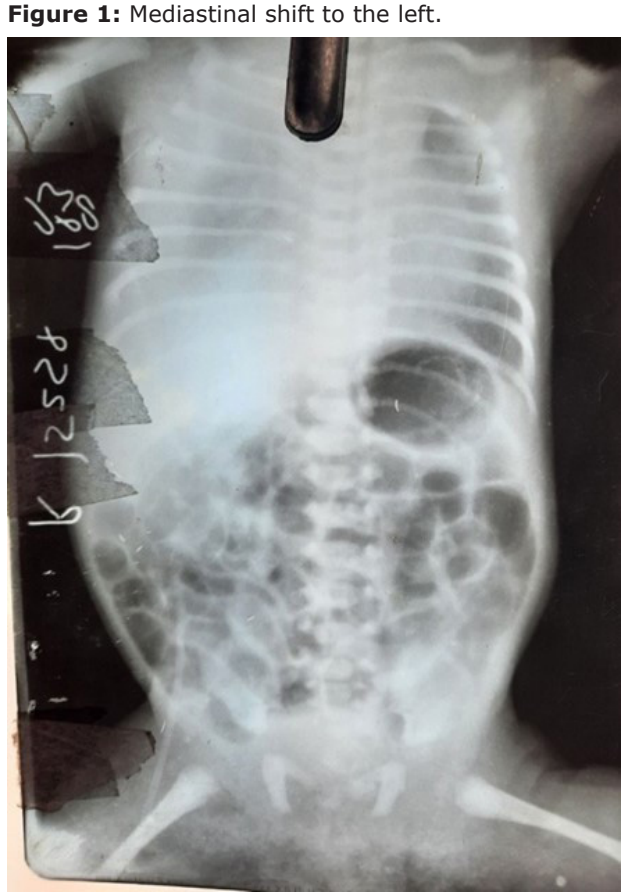
Obstetric history revealed one previous first trimester spontaneous abortion and one living healthy male sibling aged 18 months. There was no history of fever or rash in mother in first trimester. Viral markers and VDRL status of mother were negative.

Antenatal scans: Nuchal translucency scan was normal; TIFFA scan was normal; double marker showed decreased PAPP-A level; growth scan during third trimester showed fetal growth restriction, moderate polyhydramnios (AFI 32 cm), increased placental thickness with ?fetal hydrops; fetal 2D echocardiography showed cardiomegaly. Mother was investigated for TORCH infections and was IgM positive for CMV and managed symptomatically.

Head to toe examination was normal. There were no dysmorphic features or generalized edema. Baby had respiratory distress with tachypnea, retractions, decreased air entry in right hemithorax and audible grunt. Silverman Anderson Score was 7/10.

In view of worsening respiratory status, the neonate was intubated and started on mechanical ventilation. CXR revealed a large right-sided opacity with mediastinal shift to the left (Figure 1). Bedside ultrasound confirmed massive right pleural effusion with atelectatic lung changes. Therapeutic thoracentesis was performed and intercostal tube was placed in right 5th intercostal space in mid axillary line. There was a gush of straw colored serous fluid (around 50 mL). Over the next 24 hours an additional 150 mL of serous fluid was evacuated. Laboratory workup included CBC, CRP, blood cultures and venous blood gas; VBG showed respiratory

Figure 1: Mediastinal shift to the left.



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acidosis. Initial sepsis screen and blood cultures were negative. Pleural fluid was sent for analysis. Baby was evaluated for TORCH infections and received empirical broad-spectrum antibiotics.

Ventilation parameters improved after drainage and baby was extubated on DOL 3. Gavage feeds were introduced. The pleural drain fluid never turned milky after feeds. By DOL 4, chest drain output diminished to <5 mL/day and right lung appeared fully expanded on CXR. ICD tube was clamped after pediatric surgeon opinion. On DOL 6 there was increased respiratory distress requiring re-intubation. CXR showed re-accumulation of pleural fluid and new lung infiltrate. Sepsis screen and blood cultures were repeated. Blood culture was positive for Klebsiella pneumonia and baby was diagnosed with ventilator acquired pneumonia. Antibiotics were upgraded as per sensitivity. Subsequently, clinical and radiographic improvement was noted. Baby was extubated on DOL 15 and connected to NIPPV and respiratory support was tapered to room air. ICD tube was removed on DOL 15 after ensuring complete lung expansion and no re-accumulation. On DOL 17, CECT chest showed mild right pleural effusion with sub-segmental atelectasis in lower lobe secondary to pneumonia.

Pleural fluid analysis

The drained fluid was serous and did not appear milky. Laboratory analysis showed protein level 1014 mg/dL (low), glucose 45 mg/dL, triglyceride not detected. Cell count was 80 cells/mm³ (20% neutrophils, 80% lymphocytes). Gram stain and culture were sterile. CBNAAT was negative. These findings confirmed a non-lymphatic transudative cause of pleural effusion.

Additional evaluation

Bedside echocardiogram showed closing PDA and mild tricuspid regurgitation with good ventricular function. 2D ECHO at 4 months showed small ASD (4 mm) and mild pulmonic stenosis. Abdominal and cranial ultrasounds were normal. Kidney and liver function tests were normal. TSH was normal. TORCH serology showed no IgM antibodies; IgG antibodies to toxoplasma, rubella and CMV were detected. Urine PCR for CMV was negative. Clinical geneticist advised karyotyping which revealed normal male genotype (46,XY). MLPA revealed no microdeletions. Whole exome sequencing showed MYH8 gene heterozygous missense mutation (variant of uncertain significance).

Outcome and follow up

The infant was discharged after 41 days of NICU stay. Infant had adequate weight gain and breastfeeding was established. Due to stormy NICU course, the child was followed up for neurodevelopment and was found to have mild gross motor delay. MRI brain showed periventricular leukomalacia and thinning of corpus callosum. Fine motor, language and social milestones appeared normal. Improvement in motor milestones was noted after physiotherapy. Child developed bilateral inguinal hernia and underwent herniotomy.

Discussion

Pleural effusion in a neonate can result from excess production or decreased absorption of pleural fluid due to damaged lymphatics. Neonatal pleural effusion may be congenital or postnatally acquired. Congenital effusions act as a space occupying lesion and may lead to hypoplastic lungs postnatally.[1]

The absence of fluid in other cavities or edema rules out hydrops. Congenital heart diseases usually present with heart failure and effusions along with hepatomegaly. Lymphangiectasia and lymphatic obstruction may initially present as simple effusions and turn into chylothorax after fat feeds.[2] However, in this case the effusion remained non-chylous after feeds were initiated. CECT chest was done to rule out pulmonary hypoplasia and bronchopulmonary sequestration or CPAM.[3] Antenatal infections like HSV, CMV and parvovirus are reported to cause pleural effusion; a likely etiology could be antenatally acquired intrauterine infection.

Postnatal causes include injury to thoracic duct after thoracic surgery or extravasation of TPN fluid from central catheters or parapneumonic effusion. The neonate did not undergo thoracic surgery and no central lines were inserted. Development of ventilator acquired pneumonia may explain prolonged persistence of ICD drain output.

This case had no identifiable etiology and is therefore an idiopathic pleural effusion. There is lack of definitive literature regarding therapeutic options apart from thoracocentesis. Somatostatin analogues have been tried in congenital chylothorax; their benefit in non-chylous pleural effusion is unknown.[2]

Compliance with Ethical Standards
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