ORIGINAL RESEARCH PAPER
Volume - 10 | Issue - 08 | August - 2021 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

# FETAL PRIMARY PLEURAL EFFUSION: ANTENATAL MANAGEMENT AND FOLLOW UP

**Obstetrics & Gynecology**

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## ABSTRACT

Pleural effusion in the fetus and the new born is a rare entity. Fetal pleural effusions are seen in about 1 in 15,000 pregnancies[1]. They can be divided as acquired or congenital based on etiology. The congenital causes can further be divided as primary pleural effusion wherein lymphatic malformations is the cause and secondary pleural effusion when it is associated with structural abnormalities or infections or hydrops leading to pleural effusion[1].

We present a case of congenital fetal pleural effusion and its antenatal management till delivery and neonatal follow up. With the neonatal prognosis being highly variable from spontaneous resolution, minimal symptoms to perinatal death we have highlighted the various causes and therapeutical management options.

## KEYWORDS

**INTRODUCTION:**

Pleural effusion is the accumulation of fluid in pleural space, it could be due to increased rate of filtration or decrease in lymphatic clearance or both. Incidence of pleural effusion in new bornrange from 2.2 to 5.5 per 1000 births[2].

Primary fetal hydrothorax or congenital hydrothorax is due to multiple lymphatic abnormalities or thoracic tumors or cardiovascular diseases. It can present as unilateral or bilateral pleural effusion more common in males than in females with a mortality rate of 22 to 53 %[3,4].Secondary fetal hydrothorax is due to immune or non-immune hydrops. These generally present as bilateral pleural effusion along with ascites, pericardial effusion, subcutaneous edema[5]. The prognosis in these cases is not veryfavorable.A detailed antenatal evaluation aided by ultrasound, blood tests for blood grouping and typing, antibodies for infections and, genetic testing will help in knowing the cause which will guide in the treatment and prognosis.

**Case report:**

A 29year old primigravida conceived with ovulation induction came for routine antenatal visit, she was diagnosed to have hyperprolactinemia preconceptionally and was on Tab. Cabergoline 0.5mg weekly once till confirmation of fetal heart on USG. She is married for 2 1/2years and it is a second degree consanguineous marriage. She was also hypothyroid and hence started on medication from her 4th week. Early pregnancy scan, complete blood picture and sugars were normal. Her blood group is B positive and 1st trimester biochemical markers were negative. Her 20weeks anomaly scan showed mild to moderate pleural effusion in the left hemi thorax and no other abnormalitywasdetected. She was referred to a fetal medicine center, where a repeat scan at 21 weeks showed left pleural effusion with mediastinal shift to the right and minimal effusion on the right side. No signs of hydrops or hydramnios were seen. The couple were counselled and Toxoplasmosis, Rubella, Cytomegalovirus and Herpes Simplex(TORCH) test with avidity, amniocentesis for quantitative fluorescent polymerase chain reaction (QFPCR), karyotyping and TORCH polymerase chain reaction(PCR) were done. TORCH panel was negative with CMV IgG positive with high avidity showing that she was not likely infected in the past three months, karyotyping was normal TORCH PCR negative. At 23rd week pleural aspiration was done which showed an inflammatory smear, total proteins <2, albumin <1.

A repeat scan after a week showed left sided pleural collection of about 26.8cc. She was monitored by weekly scans and the scan at 26th week showed left sided pleural effusion of about 80.7cc, right side no effusion and heart was shifted to the right. As there was significant reaccumulation of the pleural fluid. The parents were counselled regarding thoraco-amniotic shunting or serial thoracocentesis and the complications due to the procedure. The decision was taken for serial thoracocentesis and 60ml of fluid was aspirated at 30th weeks after steroid coverage and about 74ml at 34th week. She was taken up for elective LSCS at 38th week after the 4ththoracocentesis the previous day, a single dose of steroid was administered the day prior. she delivered a boy baby weighing 2.65kg with Apgar8 and 9 at 1 and 5min. Delayed cord clamping was done and baby handed over the neonatologist.

Externally baby looked normal no obvious congenital anomalies. Oxygen saturation was 100% at room air. Chest wall appeared symmetrical with mild tachypnoea, air entry was decreased in the left axillary region. Apex beat was shifted to the right side. All peripheral pulses were felt.

**Day1:** Basic blood works like CBC, CRP, blood culture, X-ray, USG abdomen and chest, ECHO, TORCH and karyotyping done. X ray showed minimal right side pleural effusion, large left pleural effusion with mediastinal shift to right. Intercoastal drain (ICD) inserted and 62ml straw colored fluid drained, sent for cytology WBC 9000cells/cumm, RBC 3500cells/cumm, lymphocytes 98%, Triglycerides 18mg/dl, cholesterol 29.2mg/dl proteins 2.8g/dl.Baby kept nil orally, IV dextrose and amino acid infusion were given.

**Day2:** Baby was started on Pasteurized human milk feeds 30ml/kg/day. Ultrasound chest showed septations with 15-20ml fluid collection. 67ml of fluid was drained through ICD. Feeds increased to 50ml/kg/day. Tachypnoea settled, baby had stable vitals on room air.

**Day3:** Around 8ml of milky yellow fluid was drained, Ultrasound chest showed minimal fluid. Feeds increased to 90ml/kg/day. Pleural fluid analysis showed RBC 3000cells/cumm, WBC 2548cells/cumm, lymphocytes 97%, neutrophils 3%, glucose 53mg/dl, proteins 2g/dl, triglycerides 331mg/dl, cholesterol 23mg/dl suggestive of chylothorax. Serum cholesterol 98mg/dl, serum triglycerides 151mg/dl, serum proteins 4.8g/dl.

**Day 4:** Feeds changed to simylmedium chain triglycerides (MCT) after diagnosing chylothorax

**Day 5:** There was no collection and MCT feed continued.

**Day6:** Fat soluble vitamin supplements were started. C reactive protein trend shifted from18.9-9.6 as baby was started on Inj. Piptaz from day1 to day5. Pleural and blood cultures were negative. TORCH IgG and IgM was negative, karyotyping normal. Baby's blood group is AB positive

**Pleural fluid analysis**

| | Day 1 | Day 2 | Day 3 | Day 4 |
|---|---|---|---|---|
| Drain | 62ml | 67ml | 80ml (chyle) | No collection |
| Total proteins | 2.8g | | 2g | |
| WBC | 9000 | | 2548 | |
| RBC | 3500 | | 3000 | |
| Lymphocytes | 98% | | 97% | |
| Triglycerides | 18mg/dl | | 331mg/dl | |
| Cholesterol | 29.2 mg/dl | | 23mg/dl | |
| Glucose | | | 53 | |
| Feed | IV dextrose | Pasteurized human milk | Semyl medium chain triglycerides started | |

The baby was discharged on day 7 and called for review after 3 days, 10 days and 17 days. Baby tolerated feeds well. Subsequently mothers milk was increased slowly weaning of MCT feeds, by 6weeks baby tolerated mother feeds well and USG chest showed no collection. Baby doing well since then.

## DISCUSSION:

Pleural effusion may be diagnosed antenatally or after birth. Though it can develop anytime during pregnancy it is mostly identified in the second or third trimester[6]. A detailed ultrasound is the cornerstone for the diagnosis of primary pleural effusion. Exclusion of co existent malformations which are seen in about 10-15% of cases[7,8] especially lung abnormalities is important.Other important considerations include a detailed prenatal examination and evaluation, identification of maternal blood type and screening serum antibodies to exclude immune hydrops, detection of infectious factors including TORCH, syphilis, parvovirus B19, performing KleihauerBetketest to exclude fetal maternal transfusion syndrome. Careful USG examination to note placenta, amniotic fluid, fetal ECHO, dopplers of umbilical artery, middle cerebral artery and ductus venosus[9,10].Karyotyping and genetic testing should be performed[11-14]. As in our case these tests were done and we ruled out infections, genetic abnormalities and other associated fetal malformations.

In the new born pleural effusions can be diagnosed with chest radiographs or ultrasound and are classified as small (less than quarter lung field is involved), moderate (quarter to half lung field involvement), severe (more than half lung field is involved) and massive (around the entire lung with mediastinal shift)[15].Criteria to define chylothorax as given by Buttikeretal: absolute white cell count >1000cells/muL with lymphocyte fraction >80% and triglycerides levels >110mg/dl in pleural fluid[16].

Congenital chylothoraxare associated with genetic diseases such as Noonan's, down's and turner's syndrome, primary congenital pulmonary lymphangiectasis. Most of the acquired chylothorax are postoperative complications mostly seen as unilateral commonly left sided and of moderate volume[17].

In the case presented x ray of the new born and other investigations of the pleural fluid showed it as a primary massive pleural effusion.

**Management:**

Primary small non hydropic effusions can be managed conservatively and most often they spontaneously regress[18]. In case of enlarging pleural effusions associated with mediastinal shift, significant hydramnios draining of the pleural fluid is indicated. This reduces the chance of pulmonary hypoplasia especially between 16 to 24 weeks of gestation during which airway differentiation begins[19]when lung expansion is not seen following decompression the risk of pulmonary hypoplasia is increased. The severity of effusion, period of exposure, when the diagnosis to delivery interval >2weeks can cause delayed pulmonary maturation[20]. Management options include several repeat thoracocentesis or thoraco-amniotic shunting, other options include pleuroamniotic shunting(PAS) and pleurodesis with sclerosants is being tried, it is less invasive and technically simpler than PAS but the safety and long term outcome for sclerent needs to be further investigated[21].Peripartum or intrapartum thoracocentesis should be considered in large pleural effusions[22-24].In the presence of a shunt, it should be immediately clamped after birth to prevent pneumothorax. In our case serial thoracocentesis was done to restrict pulmonary hypoplasia and repeated a day prior to delivery to reduce the chance of fetal distress.

Prognosis in terms of perinatal morbidity and mortality depends mostly on prematurity, presence of hydrops and pulmonary hypoplasia. Poor prognostic indicators include associated malformations, hydrops, early presentation before 33weeks, bilateral effusions, unilateral effusion with mediastinal shift and prematurity[21]. A favorable prognosis in the case presented despite the severity of the disease and its early presentation can be attributed to a close follow up and serial thoracocentesis thereby preventing pulmonary hypoplasia and good NICU facility.

## CONCLUSION:

Fetal Pleural effusion when diagnosed antenatally should be referred to a tertiary fetal medicine center for a thorough evaluation to identify the cause and manage with timely fetal interventionsfor a better perinatal outcome. Delivery should be planned in a tertiary care center with facilities for neonatal ICU and the infant should be closely followed and evaluated for respiratory outcomes.

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