RESEARCH BRIEF

Mid-term Follow-up of Neonatal Pleural Effusion

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Received: November 07, 2013; Initial review: December 18, 2013; Accepted: February 22, 2014. **Objectives:** To investigate the clinical course and mid-term prognosis of neonates admitted with pleural effusion. **Methods:** Case records of 38 neonates admitted with pleural effusion were retrieved and analyzed. **Results:** 16 (42%) patients had congenital and 22 (58%) patients had acquired causes of pleural effusion. The most common causes of congenital pleural effusion and acquired pleural effusion were chylothorax (18%) and congestive heart failure (13%), respectively. Poorer outcome was observed with fetal hydrops, preterm birth (<34 weeks) and associated defects. **Conclusions:** Most of the neonates with pleural effusion have good outcome in the mid-term follow-up.

Key words: Chylothorax, Hydrops, Neonate, Outcomes,

he accumulation of fluid in the pleural space is a common manifestation of a wide spectrum of diseases in the perinatal period [1]. The incidence is low, ranging from 2.2 to 5.5 per 1000 births [2]. Most series addressing this issue have only reported short-term outcome for the neonates born with pleural effusions. The aim of this paper was to analyze the clinical features and the outcome of neonatal pleural effusion, including mid-term follow-up, in a tertiary neonatal care center in Shanghai, China.

METHODS

This study was approved by the Ethic Committee of Children's Hospital of Fudan University. We retrieved records of patients from the hospital computer database whose diagnoses were pleural effusion, chylothorax, hydrothorax, or hydrops fetalis from January 1, 2007 to December 31, 2011. All neonates admitted to the neonatal intensive care unit (NICU) with any of the above diagnoses were followed.

Pleural effusion was diagnosed on the basis of the combination of clinical manifestations chest *X*-ray and/or pleural fluid assessment. The etiology was subsequently confirmed by a neonatologist. Categorization of each patient as a congenital or acquired pleural effusion was confirmed independently by the authors based on prenatal and neonatal evaluation. The severity of the pleural effusion was then categorized as per Petersen, *et al.* [3].

Statistical analysis was performed to investigate the effect of case characteristics and prenatal intervention on survival using chi-squared test for categorical variables

and *t* test for continuous variables. Multivariate logistic regression analysis was performed to identify independent predictors for survival. A *P* value <0.05 was considered statistically significant. Analysis was performed with SPSS Version 17.0 software package.

RESULTS

In the 5-year period, pleural effusion was diagnosed in 38 (25 males) out of 15830 newborns admitted to NICU. Four children died and 34 were discharged from the hospital. The median gestational age was 34 (range 30-40) weeks. There were 23 full-term babies and 15 premature babies. The birth weight ranged from 1850 g to 4250 g (mean 2526 g). Cesarean section was the mode of delivery in 20 patients. The pleural effusion was unilateral in 13 patients (6 in the right and 7 in the left) and bilateral in 25 patients. Prenatal diagnosis was made in 6 patients: 4 had hydrops fetalis and 2 had other causes.

The etiology of neonates with pleural effusion of each category is listed in *Table I*. Chylothorax was the most common etiology in congenital pleural effusion (16) and congenital heart disease for acquired pleural effusion (22). Four patients with congenital chylothorax had a resolution of effusion after complete cessation of enteral feedings and institution of total parental nutrition (TPN) in combination with medium chain triglyceride-based formula and/or chest tube placement. Three children with congenital chylothorax did not respond to complete cessation of enteral feedings and octreotide therapy.

Four (10%) children died in this series. One patient

TABLE I UNDERLYING CAUSES OF NEONATAL PLEURAL EFFUSION (N=38)

Diagnostic category	Number (%)	
Congenital pleural effusion	16 (42)	
Congenital chylothorax	7 (18)	
Hydrops fetalis	4 (9.5)	
Twin-twin transfusion (recipient)	1 (2.5)	
Cystic hygroma	1 (2.5)	
Congenital diaphragmatic hemangioma	1 (2.5)	
Intrapericardial teratoma	1 (2.5)	
Trisomy 18	1 (2.5)	
Acquired pleural effusion	22 (58)	
Congenital heart disease	5 (15)	
Complication of thoracotomy	3 (7.5)	
Congenital diaphragmatic hernia repair	2(5)	
Patent ductus arteriosus ligation	2(5)	
Oesophageal atresia repair	2(5)	
Leakage of total parenteral nutrition	2(5)	
Congenital syphilis	2 (5)	
Capillary leakage syndrome	2 (5)	
Hypoproteinemia	1 (2.5)	
Renal failure nephrotic syndrome	1 (2.5)	

with hydrops fetalis, congenital anomaly, and congenital heart disease, who received aggressive resuscitation immediately after delivery, died 12 hours later. Two other patients with hydrops fetalis died during the course of hospitalization; they had received prenatal cordocentesis and intra-uterine transfusions. One child who had extravasation of TPN from the jugular vein catheter died of cardiac tamponade and multi-organ dysfunction.

Univariate analyses showed that, compared with neonates who were discharged from the hospital, neonates who died were smaller, more immature and sicker in the period immediately after delivery. Significantly poorer outcome was also observed with fetal hydrops, preterm birth (<34 weeks), and associated defects (*Table II*). Upon logistic regression analysis using only these variables, clinical course remained as a significant independent predictor of survival.

In the one year follow-up, 2 of the 34 survivors were lost to follow-up. We did not observe any recurrence of pleural effusion in those 32 babies who completed the follow-up. Four children had recurrent infections of the respiratory tract, and the remaining 28 had a normal clinical course after one year of follow-up. The growth

TABLE II SURVIVAL RATE ACCORDING TO DIFFERENT PROGNOSTIC FACTORS

Factors	Survival rate (%)	P value	RR (95% CI)
Location			
Unilateral	13/13 (100)	0.05	1.5 (0.3-11.9)
Bilateral	18/21(84)		
Hydrops			
Yes	1/4 (25)	0.001	13.5 (1.2-123.1)
No	34/34 (100)		
Associated defe	ects		
Yes	2/6 (33)	0.002	9.6 (1.4-67.3)
No	32/32 (100)		
GA at delivery			
<34 weeks	3/5 (60)	0.003	17.5 (1.5-196.3)
≥34 weeks	33/33 (100)		
Severity of pleu	ral effusion		
Mild	11/11 (100)	0.06	8.1 (1.159.2)
Moderate/seve	ere 24/27 (88)		

(weight and length) and nutritional status assessment did not detect any malnutrition or growth retardation.

DISCUSSION

In this series of 38 neonates with pleural effusion, 16 patients (42%) had congenital and 22 patients (58%) had acquired causes. A significantly poorer outcome was observed with fetal hydrops, preterm birth <34 weeks, and associated defects. We did not observe any recurrence of pleural effusion over one year follow-up.

The relatively small sample size was a limitation of the study. Longer period of follow-up might give us more information regarding these babies with variable underlying diseases.

Clinicians must be aware of the wide range of disorders causing neonatal pleural effusion, the different types and their clinical presentations, differential diagnosis, and their specific treatment [4]. The prognosis ascribed to pleural effusion is dependent upon patient characteristics, and may be modified by perinatal intervention. Structured approach and multidisciplinary treatment plays a key role in improving the prognosis [5].

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