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CASE REPORT

Differences in diagnostic and therapeutic procedures in the foetus and newborn depending on the clinical form of non-immune hydrops fetalis

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ABSTRACT

Hydrops fetalis is a Greek term for the pathological accumulation of fluid in the soft tissues and cavities of the foetal body. The diagnosis is made on the basis of an ultrasound examination confirming the accumulation of fluid in at least 2 foetal compartments. The paper presents the comparison of 2 clinical forms stemming from non-immune hydrops fetalis (NIHF). The following description highlights diagnostic difficulties related to the fact that the cause of the above conditions may not always be possible to determine. The continuation of multidirectional action, including both diagnostic work-up and treatment, is fundamental to the success of the treatment process, regardless of the location of the accumulated fluid and the unknown cause. The clinical condition is of key importance to the management of the newborn.

KEY WORDS:

hydrops fetalis, congenital infection, decompression.

INTRODUCTION

Hydrops fetalis is characterised by the abnormal accumulation of interstitial fluid in foetal body compartments, including the peritoneum, pleura, and pericardium, or generalised skin oedema [1]. Alternatively, it may be defined as fluid accumulation in 2 anatomical areas in the foetus or as an exudation identified within one site concomitant with subcutaneous tissue oedema [2]. Both immune and non-immune aetiologies may underlie the development of hydrops fetalis [3]. Incompatibility in the Rh system of the mother and foetus had been the most common cause prior to the use of the anti-D immunoglobulin in 1968. Currently, NIHF constitutes about 90% of cases, with the pathogenesis not being fully understood [4–8].

NIHF is associated with processes that interfere with the fluid shift between vascular and interstitial compartments, including heart abnormalities, aneuploidy, hemoglobinopathy, lymphatic system malformations, and congenital infections (Table 1) [9–11].

CASE PRESENTATION

CASE 1: HYDROTHORAX DIAGNOSED IN THE FOETUS AT 21.6 WEEKS OF GESTATION (TABLE 2)

A woman (gravida 2, para 2) delivered a male newborn at 37.0 weeks of pregnancy. On the day of caesarean section, the right pleural cavity was decompressed,

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TABLE 1. Causes of non-immune hydrops fetalis

Cardiovascular
Arrythmia, myocardiopathy, structural malformations (Ebstein anomaly, premature closure of the foramen ovale), vascular obstruction (tumour, structural, fibroelastosis), vascular malformation, and haemangioma
Genetic
Skeletal dysplasias and myopathies, metabolic diseases (Gaucher, GM1 gangliosidosis, mucopolysaccharidosis), autosomal diseases (Nooan, Prune belly, Fanconi), chromosomal abnormalities (trisomy 21, 18, 13, Turner's syndrome)
Congenital infections
Virus (cytomegalovirus, parvovirus B19, rubella, varicella, herpes, syncytial respiratory), toxoplasmosis, syphilis, Chagas disease
Hematologic
Non-immune anaemia, alpha-thalassemia, others (leukaemia)
Placental
Twin-twin transfusion syndrome, causes related to the umbilical cord
Miscellaneous
Respiratory (pulmonary sequestration, adenomatoid disease, chylothorax, tumour), genitourinary (obstructive uropathy, dysplasia, cysts, thrombosis, nephrotic syndrome), gastrointestinal (duodenal/jejunal atresia, anal imperforation, peritonitis), neurological (encephalocele, intracranial haemorrhage, cerebral aneurysm), tumoural (sacrococcygeal teratoma, neuroblastoma, hepatoblastoma), multiple causes (presence of more than one associated aetiopathic causes)
Idiopathic
Non-defined cause

TABLE 2. Details of prenatal history, tests performed, and abnormalities found

			Case 1		
Mother's blood group			A Rh D +		
Maternal diseases			Post-caesarean section, hypothyroidism, lower limb oedema		
Disorders detected in the foetus during pregnancy	Presence of fluid – location		Pleural cavity, subcutaneous tissue		
	Cardiological		Subvalvular ventricular septal defect		
	Haematological		Lack of		
	Obstetrics		Increased umbilical artery pulsatility index (> 95 percentile), agenesis of the ductus venosus (DV)		
	Osteoarticular		Lack of		
Performed procedures	Cardiotocography (CTG)		FHR approximately 135 beats/minute; oscillation normal, accelerations present, without decelerations		
	Amniocentesis		Performed – karyotype correct		
	Drugs		Levothyroxine Prenatal steroid therapy (3–4 days before delivery)		
	Foetal procedures		Decompressive puncture (33.1 weeks of foetal life)		
			Pleural shunts	Side	Gestational age (week)
				Left	23.5
				Right	24.3
				Right	30.3
				Right	32.5
				Right	33.3
Additional mother's tests	Biochemical	The day before birth	Blood count, D-dimer, INR, PT, APTT – normal		
		5 days before birth	CRP, sodium, potassium, magnesium, glucose, LDH, creatinine, ALT, AST, total bilirubin – normal		
	Towards congenital infections		Parvovirus B19: IgG–, IgM–, PCR negative *Toxoplasma gondii:* IgG+ (low titre), IgM– (previous pregnancy – no data) *Treponema pallidum:* IgG–, IgM – GBS negative CMV: IgG+, IgM–, PCR negative		
PROM/PPROM			Retained to the caesarean section		
Method of delivery			Caesarean section		

with 35 ml of fluid being removed. The newborn was born in moderate general condition. The respective APGAR scores were 6/6/8/9 at 1/3/5/10 minutes after birth. Anthropometric measurements at birth included body weight (3060 g; 50–90 pc), body length (58 cm; > 97 pc), head circumference (34 cm; 50–75 pc), and chest circumference (38 cm). The following abnormalities were identified on physical examination: oedema of the subcutaneous tissue, peripheral cyanosis, hypotonia, and low respiratory drive. Numerous traces of shunts were visible. The newborn was started on Neopuff ventilation (FiO_2 0.3–0.5–1.0). Due to the lack of improvement in the 10th minute of life, mechanical ventilation was introduced in PC-AC mode with VG. Urgent chest radiography revealed right-sided pneumothorax. Decompression drainage was inserted. On follow-up lung radiography, the right lung was expanded, and the position of the central catheter was normal. The patient was started on empirical antibiotic therapy with ampicillin and gentamicin. Enteral feeding was not implemented. The newborn was sedated with fentanyl, and parenteral nutrition was initiated. The shunt was removed from the left pleural cavity in the 5th hour of life. Normal age-specific values of arterial pressure, cardiac function, and diuresis were recorded. Trophic nutrition was introduced on the 2nd day of life. Subsequently, it was possible to discontinue the right-sided pleural drainage. As the patient's condition stabilised, the drain was removed on the 4th day of life. Follow-up laboratory results obtained on the 6th day of life revealed no positive inflammatory findings, so combination antibiotic therapy was also discontinued. The patient was then extubated and supported with non-invasive pressure-controlled continuous mandatory ventilation (PCCMV). On the 7th day of life, the ventilation mode was changed into high-flow nasal cannula (HFNC) therapy. Four days later, HFNC was discontinued, and no respiratory support was necessary from then on. On the 31st day of life, the child was discharged home in good general condition. Fluid restriction and diuretics were not recommended.

On transfontanellar ultrasound, the central nervous system was normal. Lung ultrasound performed on the first day of life revealed fluid in the epiphrenic parts of both pleural cavities: approx. 3.5–4 mm dorsally and laterally on the right side, and approx. 3.5 mm dorsally on the left side. Air was present in the right pleural cavity (Figure 1). Subsequent lung ultrasound examinations performed on the 4th (2.5 × 2 mm) and 8th (1.5 × 1.5 mm) day of life showed a systematic decrease in the amount of fluid in the epiphrenic parts of both pleural cavities, with no air in the pleural cavities on the 8th day of life. On the 18th day of life, fluid was present only in the right pleural cavity (1 mm), and on the 30th day of life, only a trace amount of fluid was identified. Abdominal ultrasound was normal. Echocardiography (ECHO) performed on days 2 and 4 showed a patent foramen ovale (Fo) and ductus arteriosus (DA), and significantly increased resistance in

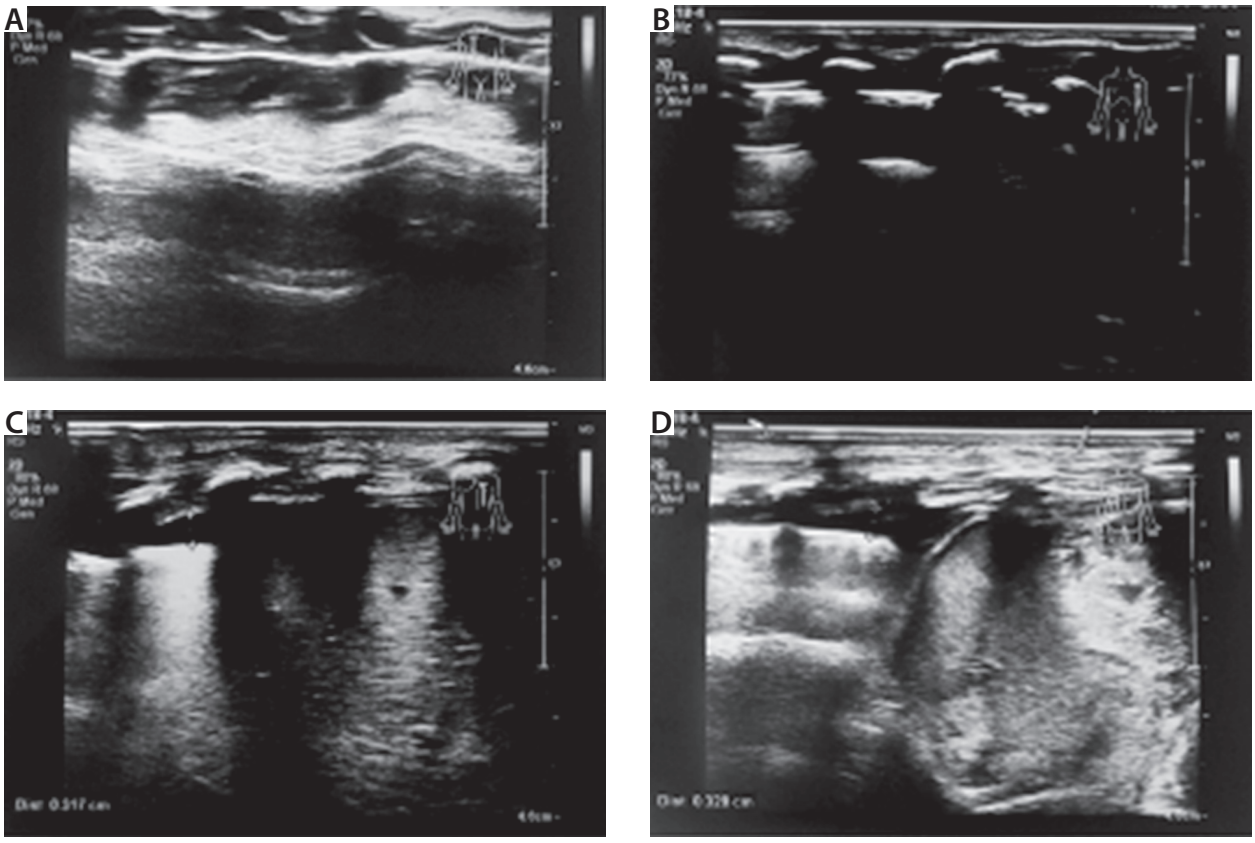

FIGURE 1. Lung ultrasound revealed the presence of fluid in the pleural cavities and air in the right pleural cavity on the first day of life. The following views are included: **A**) right anterior view, **B**) left anterior view, **C**) right posterior view, **D**) left posterior view

the pulmonary bed. At discharge, the Fo was patent, with physiological flow and the size of the left ventricle being in the upper limit of normal. Chest radiography performed on the second day of life revealed that the right lung was completely expanded without pneumothorax, with a small amount of fluid, and a trace of pneumothorax chamber in the left lung. Trace marginal left-sided pneumothorax, a trace of fluid in the right pleura, and a completely expanded lung were noted on the 3rd day of life. The laboratory tests yielded normal blood count and electrolyte parameters for the age. CRP doubled 12 hours postnatally and normalised at 72 hours of age. Otherwise, the inflammatory indices remained unincreased. Liver (ALT, AST, ALP, GGTP, bilirubin), kidney, and thyroid function tests were within normal limits for the age. Total protein concentration was slightly decreased with a tendency to spontaneous normalisation. The lipid panel was within normal limits. Normal fluid collected from the pleura at birth allowed the exclusion of chylothorax (the pleural fluid collected prenatally was negative for chylomicrons).

Tests for congenital infections in the newborn yielded the following results: parvovirus B19 IgG, IgM – negative; CMV IgG – positive, IgM – negative; *Toxoplasma gondii* IgG – positive, IgM – negative; cultures of the blood and fluid from the pleural cavity – negative.

The newborn was born in moderate general condition. Intrauterine pleural drainage was associated with the diagnosis of immune hydrops fetalis (IHF). Respiratory support with mechanical ventilation was gradually reduced to non-invasive ventilation. Such an approach was used from the first minute until the 11th day of life. A pleural effusion was seen on postnatal imaging. A pneumothorax and increased resistance in the lung bed were the complications associated with impaired lung development due to pleural fluid collection. After excluding infectious factors that could cause NIHF, we comprised any potential aetiology of NIHF in the differential diagnosis: neonatal haemochromatosis, congestive cardiac failure, twin-to-twin transfusion syndrome, hepatitis B, hypercalcaemia, hypernatraemia, hypothrombinaemia, maternal diabetes, foetal abdominal cysts, obstructed bowel, and obstructed urinary system. The aetiological factor could not be determined in the course of the diagnostic process. Therefore, although a specific infectious factor was not identified, it could be a highly likely cause.

CASE 2: HYDROPS FETALIS DIAGNOSED IN THE FOETUS AT 26.5 WEEKS OF GESTATION (TABLE 3)

A woman (gravida 3, para 3) delivered a female newborn at 36.3 weeks of pregnancy. After birth, the child was dried, and tactile stimulation and NeoPuff ventilation were applied resulting in the stabilisation of the condition. The general condition of the newborn after birth was assessed as moderate. The APGAR scores were 6/6/7/8 at 1/3/5/10 minutes after birth, respectively. Anthropometric measurements at birth included: body weight (2890 g; 50–90 pc), body length (42 cm; < 3 pc), head circumference (33 cm; 50–90 pc), and chest circumference (30 cm). The child was intubated, and mechanical ventilation was started in the 10th minute of life. On physical examination the bell-shaped chest was supported by a large symmetrical, not deformed abdomen. Moreover, the abdomen was soft with the features of hepatomegaly – the liver was palpable about 5 cm below the right costal margin. Paracetamol, fentanyl, and a single dose of sublingual midazolam were administered to relieve the patient's anxiety. A tendency to lower systemic pressure values (32–30 mmHg) was noted. Therefore, a saline bolus was administered followed by a 3-day course of dobutamine infusion. Abdominal radiography and ultrasound scans revealed free fluid in the peritoneal cavity. The patient was started on empirical antibiotic therapy with ampicillin and gentamicin. Since birth, the newborn had been fed enterally, starting from trophic nutrition and progressing to exclusive oral nutrition. Albumin was administered because the laboratory work-up revealed hypoalbuminaemia. On the 3rd day of life, it was necessary to administer albumin again due to persistent hypoalbuminaemia. The intravenous administration of 10% albumin was continued for 5 days (1 g/kg/day). Fentanyl was discontinued on the 4th day of life. The neonate was extubated, and non-invasive ventilation (NIV) was implemented. Follow-up laboratory tests revealed no positive inflammatory findings on the 6th day of life, so combination antibiotic therapy was stopped. NIV was discontinued on the 7th day of life. At the end of the 7th day of life, the patient developed tachypnoea and forced respiration, which necessitated the reintroduction of nCPAP (nasal continuous positive airway pressure) ventilation for 2 days. Subsequently, passive oxygen therapy was used for 2 days. Fluid restriction and diuretics were not used.

Transfontanellar and lung ultrasound results were normal. Abdominal ultrasound performed on the 1st day of life showed 10 mm of free fluid in the peritoneal cavity (Figure 2). The liver was slightly enlarged without focal lesions. Subsequent examinations revealed a large amount of anechoic fluid in the peritoneal cavity (3rd day – 14 mm; 8th day – 16 mm; 12th day – 7–8 mm). Most of the fluid was located in the lower abdomen and around the spleen. Slightly smaller amounts were identified in the liver area along with a small haematoma located in the left lobe of the liver. Hepatomegaly was noted. Follow-up ultrasound scans performed on day 28 showed regression of the amount of the fluid (5 mm) and the liver haematoma. Abdominal ultrasound performed on day 44 was normal. On day 2, echocardiography revealed a patent foramen ovale (FO) with bidirectional blood flow. The features of elevated resistance were noted in the pulmonary bed, and ductus arteriosus with the right-to-left shunt

TABLE 3. Details of prenatal history, tests performed, and abnormalities found

Case 2					
Mother's blood group			A Rh D +		
Maternal diseases			Condition after 2 caesarean sections, hypothyroidism, obesity, lower limb oedema, hypertension from 36.2 weeks of pregnancy		
Disorders detected in the foetus during pregnancy	Presence of fluid – location		Pericardium, peritoneum, subcutaneous tissue		
	Cardiological		Cardiomegaly, cardiac muscle hypertrophy		
	Haematological		Foetal anaemia		
	Obstetrics		Breech presentation of the foetus, 2-vessel umbilical cord		
	Osteoarticular		Shortening of the long bones		
Performed procedures	CTG		FHR approximately 140 bpm; undulating oscillation, accelerations present, without decelerations		
	Amniocentesis		Performed – karyotype correct		
	Drugs		Levothyroxine, digoxin, potassium, methyldopa, antibiotic prophylaxis (metronidazole, cefazolin, ampicillin)		
	Foetal procedures		Decompressive puncture (31.2 weeks of foetal life)		
			Intrafoetal transfusions	Amount of blood transfused	Gestational age (week)
				90 ml RCC	29.2
				58 ml RCC	30.2
			Foetal drug delivery – cisatracurium		
Additional mother's tests	Biochemical	Day of birth	blood count, INR, PT, APTT, fibrinogen, sodium, potassium, CRP – normal		
		5 days before birth	LDH, urinalysis, creatinine, uric acid, eGFR, ALT, AST – normal		
	Towards congenital infections		Parvovirus B19: IgG–, IgM–, PCR negative *Toxoplasma gondii*: IgG–, IgM– (previous pregnancies – no data) *Treponema pallidum*: IgG–, IgM– GBS – negative CMV: IgG–, IgM–, PCR negative		
PROM/PPROM			Amniotic fluid leaking 5 hours before delivery		
Method of delivery			Caesarean section		

RCC – red cell concentrate

was found. On day 33, the FO was patent with normal flow. The right ventricle was thickened and slightly enlarged. No progression was noted compared to previous examinations. Chest radiography on the 1st and 3rd day of life showed a slightly enlarged cardiac silhouette and superior mediastinal shadow. Samples collected from days 4 to 42 revealed anaemia. Iron levels were normal, and ferritin levels were significantly elevated until day 14, with a gradual decrease being observed until day 42. Transferrin concentration was decreased in a blood sample collected on day 28, and the follow-up test performed on day 42 showed its normalisation. Electrolytes were within the age-specific reference range. As regards inflammatory indices, CRP doubled 24 hours postnatally, and then it spontaneously normalised. Liver function tests (including ALT, AST, ALP, GGTP, and total and direct bilirubin) yielded elevated results. Spontaneous normalisation of the values was noted on follow-up tests performed on the 42nd day of life (Table 4).

Hypoalbuminaemia was noted for the first 4 days of life. Its normalisation was observed on day 14. Coagulation panel, total protein, and fibrinogen were within normal limits for age. The parameters of kidney and thyroid function were normal. The lipid panel was also normal for age. Tests for congenital infections in the newborn yielded the following results: parvovirus B19 IgG – positive, IgM – negative; CMV IgG – positive, IgM – negative; *Toxoplasma gondii* IgG – positive, and IgM – negative. The cultures of blood and body cavity fluids were negative.

The newborn was born in moderate general condition. Intrauterine foetal transfusions and peritoneal drainage were associated with the diagnosis of IHF. Respiratory support with mechanical ventilation was gradually reduced to non-invasive ventilation. Such an approach was used from the first minute until the 11th day of life. Postnatal imaging studies showed fluid in the peritoneal cavity. The right ventricle of the heart was thickened and enlarged on echocardiography. The complications included heart failure requiring catecholamines and increased lung bed resistance. After excluding infectious factors that could contribute to NIHF, we comprised any potential aetiology of NIHF in the differential diagnosis: neonatal haemochro-

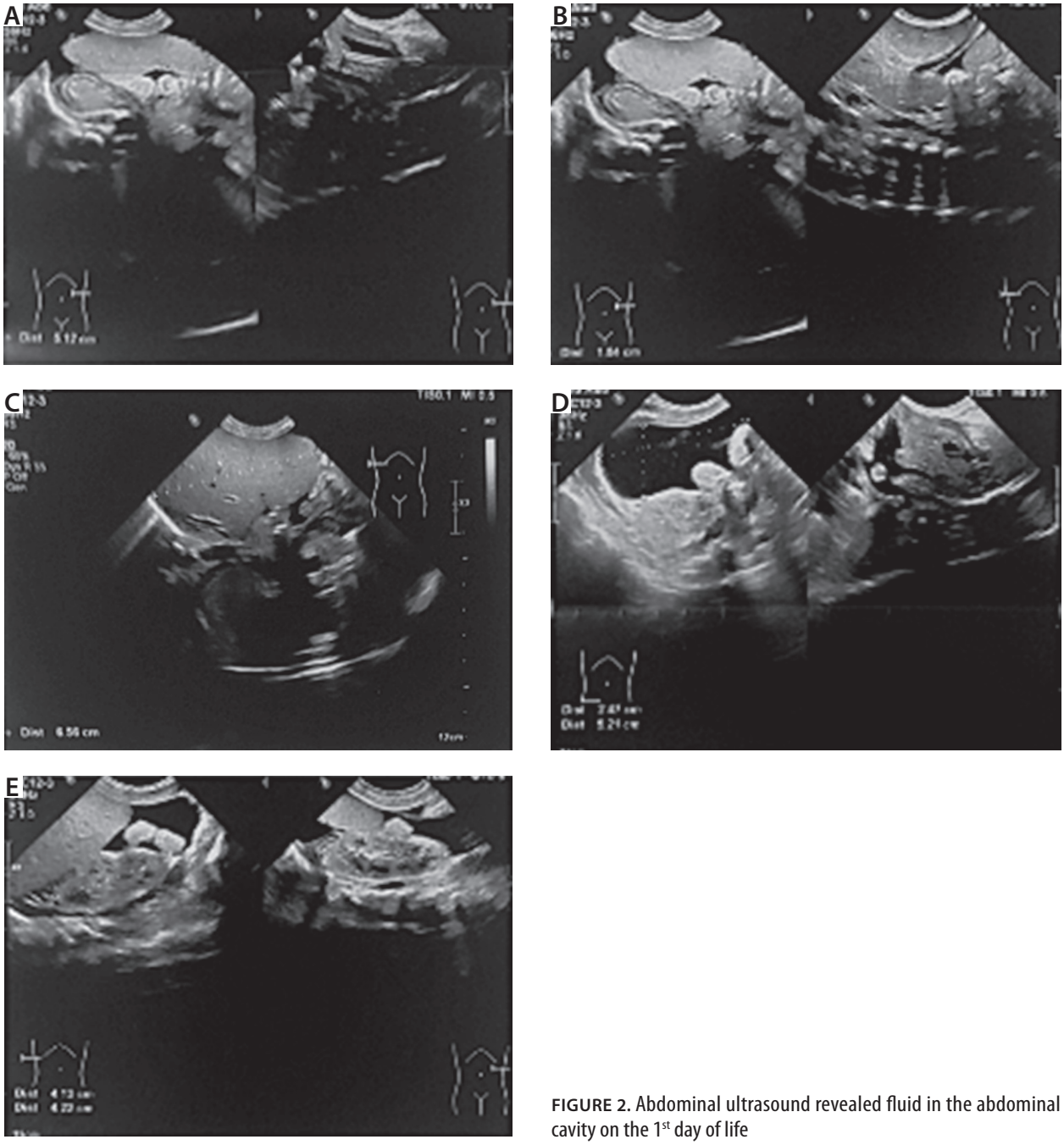

FIGURE 2. Abdominal ultrasound revealed fluid in the abdominal cavity on the 1st day of life

TABLE 4. Values of liver function tests

	ALT	AST	ALP	GGTP	Total bilirubin	Direct bilirubin
4th day	53.60 U/l	115.00 U/l	506.00 U/l	110.00 U/l	7.71 mg/dl	6.00 mg/dl
28th day	54.10 U/l	58.30 U/l	518.00 U/l	323.00 U/l	2.39 mg/dl	2.36 mg/dl
42nd day	44.50 U/l	50.90 U/l	455.00 U/l	190.00 U/l	0.97 mg/dl	0.82 mg/dl

matosis, congestive heart failure, twin-to-twin transfusion syndrome, hepatitis B, hypercalcaemia, hypernatraemia, hypothrombinaemia, maternal diabetes, foetal abdominal cysts, obstructed bowel, and obstructed urinary system. Taking account of both prenatal and postnatal data, congestive heart failure seems to be the most likely factor for the above condition, which could have developed due to a congenital infection despite the lack of an identified infectious factor. Therefore, although a specific infectious factor was not identified, it is a highly likely cause.

DISCUSSION

In the above report, we included 2 cases of NIHF newborns who met the criteria for the diagnosis of hydrops fetalis. Our aim was to present different clinical pictures and locations of the fluid in the foetuses, despite the common primary diagnosis. The case report illustrates both the differences in the management and protection of the foetus, as well as clinical problems and diagnostic and therapeutic procedures in the newborn. *In utero*, needle decompression was the common method of protecting the foetus. Other medical procedures were typically tailored to the problems of a given case. Therefore, in case 1, decompression was supplemented with intrapleural shunts, and in case 2 – with intrafoetal transfusions. Moreover, a similar general condition could be observed in both cases postnatally, i.e. the APGAR score was 6 points, meaning moderate condition. Both newborns developed respiratory distress requiring ventilation, ranging from mechanical ventilation to passive oxygen therapy. In case 1, hydrothorax was the main form of abnormal fluid collection. It constituted an obstacle as regards the normal intrauterine development of lungs, impeding normal lung expansion and respiratory function postnatally. Based on that, secondary pneumothorax was diagnosed within the first hours after birth. In case 2, the fluid was mainly located in the abdominal cavity and the pericardium. Therefore, severe circulatory failure developed requiring a catecholamine infusion and a fluid bolus.

According to the literature, NIHF was characterised by high morbidity and mortality, and heterogeneous aetiology [12]. At the same time, structural heart diseases and pulmonary hypoplasia were associated with the poorest neonatal outcomes [12]. The above statement is consistent with our observations. In both presented cases, the aetiological factor of NIHF could not be found, while the associated complications included lung hypoplasia and circulatory failure. Ergani *et al.* [13] presented similar data, stating that the aetiology was unknown in the largest percentage of patients, i.e. 46%. However, the aetiological factors may include the following: parvovirus B19, cytomegalovirus (CMV), syphilis, and toxoplasmosis. These infections may contribute to foetal anaemia with subsequent heart failure or myocarditis caused by the pathogens [14]. Therefore, Iijima *et al.* [15] and Syridou *et al.* [16] emphasised that when considering the diagnosis of NIHF detected intragestationally, it was important to screen for various infectious agents, including viruses, bacteria, and parasites such as parvovirus, CMV, syphilis, and toxoplasmosis. However, the results were negative in the study by Anghelin *et al.* [17], which is consistent with our findings. Furthermore, Chen *et al.* [18] highlighted the importance of genetic testing in the diagnosis of HF, which yielded normal results in both our cases. In the study by Watanabe *et al.* [19], the mean week of NIHF diagnosis was 29.1 ± 4.4, which is consistent with case 2, and the week of birth was 34.3 ± 2.7, which is consistent with both cases.

As regards the treatment method, Sebastián de Lucas *et al.* [20] emphasised the importance of intrauterine treatment, with particular attention paid to intrauterine transfusion (IUT) and thoraco-amniotic shunt (TAS), which were found to significantly improve the prognosis. The procedures are analogous to those performed in the cases we analysed – TAS in case 1, IUT in case 2. Similar conclusions were presented by Szewczuk *et al.* [21], who tackled the issue of no established algorithms for the management of foetal pleural effusion, with the only recommendations being dependent on the dynamics and volume of the fluid chamber. At the same time, Alkazaleh *et al.* [22] demonstrated that the presence of a valve led to effective thoracic decompression, resulting in prolonged gestation and improved neonatal outcomes. The above findings are also consistent with those of our study, in which adapting the intervention to the identified anomalies both prolonged the duration of pregnancy and improved neonatal prognosis.

CONCLUSIONS

The presented diagnostic and therapeutic approach, applied both in the prenatal and postnatal periods, demonstrates the appropriate and effective response to the identified clinical problem. Firstly, it enables foetal survival and allows for the continuation of pregnancy, thereby supporting foetal maturation and reducing the risk of prematurity-related complications. Secondly, it ensures appropriate neonatal management, including the stabilisation of vital functions and comprehensive supportive care, and a thorough search for the underlying aetiology ultimately leading to the resolution of hydrops fetalis.

DISCLOSURES

1. Institutional review board statement: Not applicable.
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

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