VERSITA

Cent. Eur. J. Med. • 8(4) • 2013 • 420-423
DOI: 10.2478/s11536-013-0189-y

Central European Journal of **Medicine**

Critical cardiac tamponade in newborn, life-saving emergency interventions and possibility of parvovirus B19 congenital infection

Case Report

Radoslaw Jaworski[1], Ireneusz Haponiuk[1], Ninela Irga-Jaworska[2], Jolanta Zablocka[3], Maciej Chojnicki[1], Mariusz Steffens[1], Jacek Juscinski[1]

1 Department of Pediatric Cardiac Surgery, Mikołaj Kopernik Pomeranian Centre of Traumatology, 1-6 Nowe Ogrody Street, 80-803 Gdansk, Poland

2 Department of Pediatric Oncology, Medical University of Gdansk, Poland

3 Department of Pediatric Cardiology, Pediatric Province Hospital in Olsztyn, Poland

Received 18 December 2012; Accepted 15 March 2013

Abstract: The presence of pericardial effusion in neonates usually indicates a poor prognosis. Here, we report a case of isolation of cardiac tamponade in a newly born. This may be related to vertical human parvovirus B19, an infection with atypical clinical manifestation. Any neonate with unexplained fetal pericardial effusion should always be tested for parvovirus B19 infection, even in the absence of known and proved fetal exposure. Despite the etiology of a tamponade the only reasonable procedure is a surgical evacuation during diagnosis.

Keywords: *Cardiac tamponade • Newborns • Pediatric cardiac surgery • Parvovirus B19*

1. Introduction

Pericardial effusion causes collection of fluid into the pericardial sac which surrounds the heart which,- if substantial- can lead to a cardiac tamponade, i.e. critical condition impairing cardiac function. The presence of pericardial effusion in neonates usually indicates a poor prognosis as is often related to cardiac failure. Restrictive cardiomyopathy in fetal life, associated malformations, chromosomal abnormalities, fetal infection, intrauterine growth restriction, nonimmune hydrops and erythroblastosis can all be causes of pericardial effusion in the neonates [1,2]. Human parvovirus B19 (PV-B19) infection is the cause of erythema infectiosum, a disease common in children, but it can also be related to a non-immune hydrops fetalis, which affects pregnant woman [3]. The risk of vertical transmission is described approximately as 30% and of hydrops, about 1% [3].

We report a case of isolated cardiac tamponade in the newborn, which may be related to vertical parvovirus B19 infection with atypical clinical manifestation.

2. Case report

Female newborn (body weight 4070g) was delivered by cesarean section to a 38-year-old mother (GIII, PIII) at 39 weeks' gestation in a regional hospital (Apgar 7), and a vast amount of amniotic fluid was noted at birth. Previously, a routine prenatal ultrasound in the first trimester was ordinary. There was not a maternal history of infection, fever, arthritis or other illnesses that might suggest an infection with PV-B19. At birth, the girl manifested severe respiratory distress and was transferred to a district hospital only few hours after delivery. Physical examination revealed dyspnea and a cardiac failure, with dysmorphic physical features, with lower situated ears, mongoloid shaped eyes, small head circumference and a flat nasal base. The initial chest radiography of the child, echocardiography (ECG), as well as computed tomography pointed out an immense accumulation of fluids within the pericardium (Figure 1). Thus, critical life-threating tamponade was diagnosed. Despite medical therapy, the pericardial puncture was performed,

* *E-mail: radicis@go2.pl*

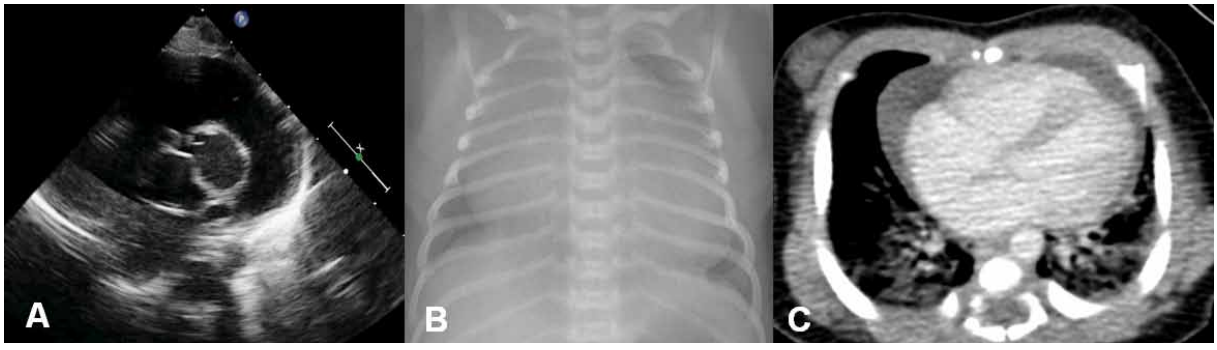

Figure 1. Initial echocardiography (A), chest radiograph (B), and computed tomography image (C) showing massive fluid accumulation within the pericardium.

with aspiration of the small amount of yellow and clear fluid. Control ECG showed a pericardial fluid volume that remained at the same level for the next 2 weeks, without life-threating signs of cardiac tamponade. At this stage the cytomegalovirus infection, bacterial infection or toxoplasmosis were excluded. There were not an evidence of anemia amongst the laboratory parameters ; blood tests revealed normal liver and renal function. The C-reactive protein level were in a normal range. Echocardiography revealed interruption of vena cava inferior without intracardiac congenital heart defects. There were not recorded any abnormalities in the head or abdomen following an ultrasound exam. The normal 46,XX cariotype without any genetic disorders was stated, and metabolic diseases were not diagnosed.

At day 17 following birth, baby was transferred to the Department of Pediatric Cardiac Surgery in Gdansk, Poland, with the diagnosis of chronic pericardial effusion. On admission, the physical examination has revealed a phenotypically abnormal, crying neonate with the reddish skin (Figure 2) and a phenotype-based suspicion of a congenital infection. The baby has not presented with any clinical signs of hydrops fetalis or organomegaly. Pericardial drainage surgery was performed under the general anesthesia, with exploration of the pericardial sac using a transxiphoid approach. The total volume of 100 ml of a yellow liquid was evacuated and sent for laboratory analyses, which have revealed the following parameters: density 1.03, pH 8, white blood cells 0.36 G/l with 94% of lymphocytes; 54 g/dl of protein, 27 mg/dl of triglyceride and 90 mg/dl of cholesterol. The histopathological examination has revealed no tumor cells in the fluid. The qualitative polymerase chain reaction (PCR) for the human PV-B19, performed using serum and tamponade fluids was negative (cut off at 251 mEq/ml). PV-B19 IgG antibodies have tested positive by enzyme-linked immunosorbent assay in the serum, although PV-B19 IgM antibodies remained negative. The baby was examined using thoracic ultrasound that showed a complete resolution of pericardial fluid following a surgical intervention. The baby was thus released from hospital on the 4th postoperative day and referred to the pediatric cardiology department for recovery. The follow-up observation revealed recurrence of the pericardial fluid 2.5 months after the first intervention as a result of chickenpox infection. At this point pericardial fluid was again successfully drained without further complications.

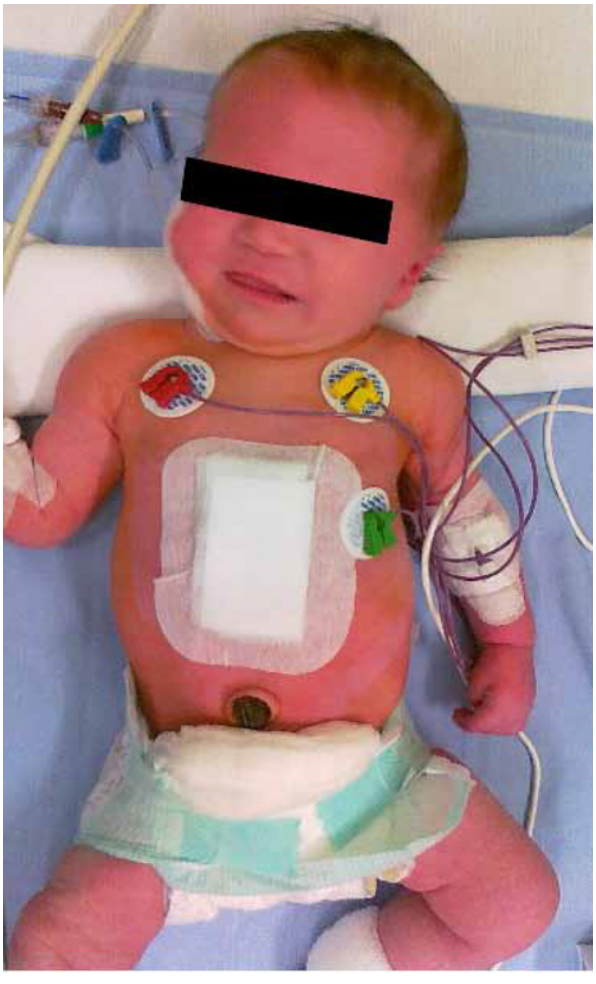

Figure 2. Physical examination of baby revealed a phenotypically abnormal neonate with reddish skin.

3. Discussion

Pericardial tamponade is a life-threating condition, especially in newborns, babies and infants. Except for major cardiac morbidities - an idiopathic pericardial tamponade in newborns is rarely reported in the literature. Alongside previously described isolated pedicardial effusion due to a restrictive cardiomyopathy or dilated and hypertrophic cardiomyopathies in fetal life, we have also considered a possibility of congenital pleural effusions occurring with hydrops fetalis, or congenital chylothorax [1,4]. Congenital PV-B19 infection may present with isolated fluid effusion as a sole manifestation [4].

Here reported baby underwent pericardial puncture in the first day after birth, and forthcoming surgical pericardial drainage sixteen days later. Both procedures are extremely rarely performed in the newborns thus requesting unique clinical settings for the life-threating circumstances. The use of fluoroscopy imaging for initial withdrawal of interventional fluid is undoubtly less invasive, although it was difficult to obtain due to lack of advanced technical equipment. Despite unknown etiology, both surgical interventions appeared safe and effective, gaing the baby life-saving support with retrieval of the fluid samples for further laboratory investigations.

Human PV-B19 is a small single-stranded DNA virus and could cause a wide spectrum of clinical manifestations from erythema infectiosum in immunocompetent children, to lethal cytopenias in the immunocompromised patients [5]. Erythrema infectiosum („fifth disease") is the most common clinical manifestation of PV-B19 infection. It is primarily seen in children aged 3-15 years, although it can occur at any age [6]. Common clinical symptoms are headache, coryza, low-grade fever, pharyngitis, and malaise, bright red erythema of the face and extremities, as well as polyarthropathy. During pregnancy PV-B19 infection can be asymptomatic or can cause a variety of signs of fetal damage (severe anemia, hydrops fetalis, death) [5]. In hydrops fetalis, nonchylous fluid accumulates in the different serous cavities (ascites, pericarditis, pleura), but widespread edema of subcutaneous fetal tissue is the main clinical manifestation [4]. It is thought, that hydrops fetalis caused by PV-B19 results from destruction of erythroid cells leading to subsequent fetal anemia which causes cardiac failure [5]. To the best of our knowledge there are only few publications reporting the association of isolated pleural or pericardial effusions and PV-B19 congenital infection [4,7,8]. Following the above we have suspected the PV-B19 fetal infection in our patient.

Maternal symptoms in the setting of parvovirus B19 infection are uncommon during pregnancy [9]. Available data suggests that in approximately 15% of pregnant women infected with PV-B19 the fetuses had adverse outcomes. The interval between the onset of the maternal infection and the diagnosis of non-immune hydrops fetalis ranges from 2 to 6 weeks. The risk of adverse outcome in pregnancy, due to e PV-B19 infection was estimated to 10.2% increasing to 12.4% when maternal infection occurred during the first 20 weeks of gestation [3]. Only few cases in which maternal infection has occurred after 20 weeks of gestation have been described in the prospective studies. When maternal PV-B19 infection was asymptomatic the interval between likely time of maternal infection and fetal manifestations ranged from 3 to 15 weeks [8]. The vertical transmission rate is estimated as 24.1% [3].

In our case, upon the analysis of pregnancy, retrospectively it has been suspected that mother of baby was infected with PV-B19 after 20 weeks of gestation. Mild clinical presentations of this were seen in here described infant although no signs of anaemia were recorded. The lack of non-immune hydrops fetalis can be explained by the fact that the site of haematopoesis after 20 weeks has moved from the liver to the bone marrow, and the erythroid lineage cells which were derived from the fetal liver during active haematopoesis were probably the target of PV-B19 [3]. Viral transmission may be suspected due to presence of an adverse outcome in baby with PV-B19 IgG present in the fetal serum. The presence of PV-B19 IgG in fetal serum can also be due to placenatal transport of maternal PV-B19 IgG antibodies [10]. There were no PV-B19 IgM antibodies in the neonatal blood of our patient; however this fact does not exclude PV-B19 infection in the fetus. The most likely possibility is immaturity of the fetal immune system, and has been described by other authors, or because PV-B19 IgM antibodies are only detectable for a limited period of time [10]. Moreover PV-B19 IgM antibodies become detectable in the serum at 7-10 days after infection, with the peak at 10-14 days and slow decline afterwards [8,11]. There is a considerable possibility of false negative results, because the sensitivity of IgM antibodies detection between 8 and 12 weeks after maternal infection varies between 63 - 70% [12,13]. Low sensitivity of the assays, transient low-titer viremia not inducing an immune response and immaturity of the fetal immune system have all been suspected as reasons for the absence of specific PV-B19 IgM antibodies [4,14,15]. In contrast, PV-B19 IgG antibodies increase from 14 days after infection and reach a plateau level after 4 weeks; these antibodies are supposed to be present for a long period [11].

The golden standard in diagnostics of PV-B19 infection still remains to be a polymerase chain reaction (PCR). PCR should be performed in maternal and neonatal blood whenever clinical suspicion of PV-B19 infection has occured, although the method is not free from limitations [4]. There are some studies that report on the

clearance of PV-B19 DNA from peripheral blood after acute infection; in some patients this clearance is registered after one month already, and in 67% of patients in the second month after the onset of illness [14,16]. In our patient PV-B19 DNA was negative, possibly due to long period from the onset of a fetal infection until the diagnosis approaches in our Department; all the tests were performed more than one month after suspected onset of infection and more than two weeks after birth. There is also a possibility, that there are low PV-B19 DNA serum levels in PV-B19 IgG antibodies positive fetuses [10]. Moreover, some authors report that PV-B19 DNA levels in the fetal blood are significantly low in the presence of PV-B19 IgG (viral load inversely correlated with IgG levels). This fact is especially important because lack of PV-B19 IgG antibodies in fetal blood might be associated with an increased risk for adverse fetal outcome [10]. What is positive, among the infected fetuses who survived after birth, there are neither congenital abnormalies nor serious neurodevelopement problems related [3].

4. Conclusion

Cardiac tamponade in newborns is a very rare emergency. Although we cannot be sure about the etiology of pericardial tamponade in our patient, we suggest that any neonate with unexplained fetal pericardial effusion should always be tested for the PV-B19 infection, even in the absence of known and proved fetal exposure. Regardless of the etiology of cardiac tamponade the only reasonable procedures are surgery or interventional evacuation, upon diagnosis.

References

[1] Sekar P, Hornberger LK, Smallhorn JS. A case of restrictive cardiomyopathy presenting in fetal life with an isolated pericardial effusion. Ultrasound Obstet Gynecol. 2010; 35:369-372

[2] Yezewski KA, Ferroni KM. Very large isolated fetal pericardial effusion with spontaneous resolution. Journal of Diagnostic Medical Sonography. 2004; 20:194-197

[3] Yaegashi N, Niinuma T, Chisaka H, Watanabe T, Uehara S, Okamura K, et al. The incidence of, and factors leading to, parvovirus B19-related hydrops fetalis following maternal infection; report of 10 cases and meta-analysis. J Infect. 1998; 37:28-35

[4] Sarafidis K, Drossou-Agakidou V, Evdoridou I, Petridou S, Hatzisevastou-Loukidou H, Dadamojas C, et al. Hydrothorax as a sole manifestation of congenital parvovirus B19 infection. Am J Perinatol. 2008; 25:551-554

[5] Savarese I, De Carolis MP, Costa S, De Rosa G, De Carolis S, Lacerenza S, et al. Atypical manifestations of congenital parvovirus B19 infection. Eur J Pediatr. 2008; 167:1463-1466

[6] Keeler ML. Human parvovirus B-19: not just a pediatric problem. J Emerg Med. 1992; 10:39-44

[7] Parilla BV, Tamura RK, Ginsberg NA. Association of parvovirus infection with isolated fetal effusions. Am J Perinatol. 1997; 14:357-358

[8] Dijkmans AC, de Jong EP, Dijkmans BA, Lopriore E, Vossen A, Walther FJ, et al. Parvovirus B19 in pregnancy: prenatal diagnosis and management of fetal complications. Curr Opin Obstet Gynecol. 2012; 24:95-101

[9] Beigi RH, Wiesenfeld HC, Landers DV, Simhan HN. High rate of severe fetal outcomes associated with maternal parvovirus B19 infection in pregnancy. Infect Dis Obstet Gynecol. 2008; 2008:524601

[10] Weiffenbach J, Bald R, Gloning KP, Minderer S, Gärtner BC, Weidner A, et al. Serological and virological analysis of maternal and fetal blood samples in prenatal human parvovirus B19 infection. J Infect Dis. 2012; 205:782-788

[11] Anderson MJ, Higgins PG, Davis LR, Willman JS, Jones SE, Kidd IM, et al. Experimental parvoviral infection in humans. J Infect Dis. 1985; 152:257-265

[12] Donders GG, Van Lierde S, Van Elsacker-Niele AM, Moerman P, Goubau P, Vandenberghe K. Survival after intrauterine parvovirus B19 infection with persistence in early infancy: a two-year follow-up. Pediatr Infect Dis J. 1994; 13:234-236

[13] Peters MT, Nicolaides KH. Cordocentesis for the diagnosis and treatment of human fetal parvovirus infection. Obstet Gynecol. 1990; 75:501-504

[14] Dieck D, Schild RL, Hansmann M, Eis-Hübinger AM. Prenatal diagnosis of congenital parvovirus B19 infection: value of serological and PCR techniques in maternal and fetal serum. Prenat Diagn. 1999; 19:1119-1123

[15] Nunoue T, Kusuhara K, Hara T. Human fetal infection with parvovirus B19: maternal infection time in gestation, viral persistence and fetal prognosis. Pediatr Infect Dis J. 2002; 21:1133-1136

[16] Erdman DD, Usher MJ, Tsou C, Caul EO, Gary GW, Kajigaya S, et al. Human parvovirus B19 specific IgG, IgA and IgM antibodies and DNA in serum specimens from persons with erythema infectiosum. J Med Virol. 1991; 35:110-115