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CASE REPORT

A Case of Non-Immune Hydrops Fetalis in an HIV-Positive Mother

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Abstract
Mother-to-child transmission (MTCT) remains the primary route of HIV infection in children, with an estimated 30–40% risk in untreated pregnancies. We report a case of a pregnant woman with a high HIV viral load who developed non-immune hydrops foetalis (NIHF), a rare and life-threatening foetal condition characterized by excessive fluid accumulation in at least two compartments. Ultrasound examinations revealed left ventriculomegaly, cardiomegaly with pericardial effusion, pleural effusion, and ascites with an enlarged spleen, confirming NIHF. Extensive investigations ruled out common infectious and non-infectious causes, raising the possibility of a link between maternal HIV and NIHF. This case highlights the need for further investigation into the potential relationship between maternal HIV, congenital HIV, and NIHF, as well as the importance of early detection and multidisciplinary management in high-risk pregnancies.

Keywords: *HIV-Positive Mother, hydrops foetalis, non-immune hydrops foetalis.*

Introduction

Hydrops foetalis (HF) is an abnormal accumulation of fluid in at least two foetal compartments, including the peritoneal cavity (ascites), pleura (pleural effusions), pericardium (pericardial effusion), and generalized skin oedema (defined as skin thickness >5 mm). Additionally, placental thickening (greater than 4 cm in the second trimester or greater than 6 cm in the third trimester) and polyhydramnios are often associated with hydrops foetalis.[1]

Hydrops foetalis can be immune or non-immune. Immune hydrops result from blood type incompatibility between the foetus and the pregnant mother. In contrast, non-immune hydrops foetalis (NIHF) occurs when fluid accumulation is not caused by maternal antibodies attacking foetal blood cells. Due to the widespread use of anti-D immunoglobulin, the prevalence of immune hydrops associated with red cell alloimmunization has declined significantly, making NIHF account for over 90% of all HF cases. The prevalence of NIHF ranges from 1 in 1,500 to 1 in 4,000 births.[2]

We present a case of a neonate born with NIHF who demised shortly after birth. The maternal HIV infection was diagnosed antenatally. An extensive evaluation for known causes of NIHF, both infectious and non-infectious, was negative.

Case report

A 28-year-old Malaysian woman, previously diagnosed as HIV-positive, became pregnant for the third time (gravida 3, para 2). She was diagnosed with HIV seven years ago, having acquired the infection from her first husband. Since then, she had a history of poor adherence, with multiple defaults on follow-up and treatment. At the time of presentation, she was married to her second husband, who was HIV-negative. Her obstetrics history included two previous emergency lower-segment caesarean sections (LSCS). Bothe of her children from previous pregnancies were HIV-negative.

In this pregnancy, she performed a self-urine pregnancy test at six weeks of amenorrhoea due to pregnancy symptoms. After confirming the pregnancy, she visited a healthcare centre, where a medical officer performed baseline viral load and CD4 tests and referred her to the infectious disease team.

At eight weeks of amenorrhea, her initial investigations showed a viral load of 50,322 copies/mL and a CD4 cell count of 205 cells/mm³. Consequently, she was started on highly active antiretroviral therapy (HAART) with Tenofovir-Emtricitabine and Dolutegravir. A repeated blood investigation at 24 weeks of pregnancy showed an improved but still high viral load of 27,290 copies/mL and a CD4 count of 238 cells/mm³. This poor response was likely due to initial poor compliance with medication during the first trimester due to nausea and vomiting. After receiving proper education and counselling, the patient understood the necessity of HAART and adhered to the treatment.

An ultrasound scan (USS) at 27 weeks' gestation identified foetal ascites, prompting referral to a tertiary foetal medicine centre. The USS confirmed the presence of left ventriculomegaly (Figure 1), cardiomegaly with pericardial effusion, hypoplastic ventricles, pleural effusion, and ascites with an enlarged spleen (Figure 2). The Middle Cerebral Artery Doppler peak systolic velocity was within normal limits, ruling out foetal anaemia. The Umbilical Artery Doppler was also normal, excluding increased vascular resistance (Figure 3).

Further investigations to determine the cause of hydrops foetalis were conducted. The patient's blood type was O positive, with negative direct and indirect Coombs tests. Screening tests for syphilis (Venereal Disease Research Laboratory test, VDRL), hepatitis C antibody, and hepatitis B surface antigen were negative. TORCHES screening showed positive IgG but negative IgM ELISA results for toxoplasmosis, rubella virus, cytomegalovirus, herpes simplex virus, and parvovirus, implying a previous infection.

The patient was informed about the diagnosis of hydrops foetalis and its poor prognosis to assist in decision-making. Given her underlying retroviral

disease (RVD) with a high viral load, history of two previous caesarean scars, and the poor prognosis of the foetus, the patient was offered an elective LSCS and bilateral tubal ligation at 32 weeks of pregnancy.

A female baby was delivered, but she was not vigorous, with Apgar scores of 5 at one minute, three at five minutes, and two at ten minutes (Figure 4). No resuscitation was provided due to the poor prognosis. The baby was pronounced dead at 13 minutes of life. The parents declined a post-mortem examination and permission for HIV testing of the newborn.

Discussion

NIHF results from an imbalance in foetal interstitial fluid dynamics due to increased venous pressure and reduced lymphatic return. Even minor increases in venous pressure can impair lymphatic flow, leading to fluid accumulation and NIHF.[3][4]

NIHF has serious perinatal implications, regardless of gestational age at diagnosis. The leading causes include chromosomal anomalies, foetal cardiovascular disorders, and congenital infections.[5]

Standard NIHF assessments include obstetric and family history reviews, infection screenings, maternal red cell antibody testing, and detailed foetal ultrasound. The Middle Cerebral Artery Doppler is used to exclude foetal anaemia. In this case, Doppler findings were normal.[6]

Congenital infections such as parvovirus B19, toxoplasmosis, cytomegalovirus (CMV), congenital syphilis, and herpes simplex virus (HSV) are part of the routine evaluation for infectious causes of NIHF.[6] An extensive infectious workup was negative for our patient. However, congenital HIV infection is not included in routine screening despite mother-to-child transmission being a significant concern in several countries.

To the best of our knowledge, there are very few reported cases of congenital HIV infection associated with NIHF. The first reported case in 2006 described an infant born to an HIV-positive mother with NIHF and hepatitis. The hepatitis resolved after the initiation of HAART, suggesting a secondary cause.[7] Another report, a retrospective review of a case series involving 28 infants with NIHF, identified one case of congenital HIV infection, although the details were not described.[8]

In our case, the mother was HIV-positive, but we were unable to determine whether the baby had congenital HIV as the parents refused testing. However, a case report described a pregnant Zimbabwean woman with HIV and hydrops foetalis whose baby tested negative for HIV.[9] This suggests that NIHF can occur regardless of the infant's HIV status, yet it is distinctly observed in mothers with HIV. This raises an important question regarding the underlying oetiology of NIHF in HIV-negative infants born to HIV-positive mothers.

In this case, the foetal echocardiogram revealed left ventriculomegaly, cardiomegaly with pericardial effusion, and hypoplastic ventricles. After infectious causes, cardiac anomalies represent the next largest oetiological group for NIHF (12.7%).[4] Cardiovascular anomalies can cause haemodynamic disturbances that impair venous return, leading to cardiac failure. Cardiac failure increases central venous pressure and interstitial fluid, which results in NIHF.[10] Interestingly, a study of 173 foetuses from 169 HIV-infected mothers reported that these foetuses can manifest abnormal cardiovascular structure and function and increased placental vascular resistance independent of their HIV status once born. [10] This report suggests that, in infants born to HIV-positive mothers, NIHF may not only be linked to possible congenital HIV infection but also to cardiovascular abnormalities that could be indirectly associated with maternal HIV.

Although our case and other reports provide valuable insights, the link between congenital HIV and hydrops foetalis remains unclear. This case highlights the need for further reporting or investigation into the potential relationship

between maternal HIV, congenital HIV, and NIHF. Since congenital HIV is not part of the routine screening for NIHF, we propose that congenital HIV be considered a possible infectious oetiology of NIHF. Clinicians should also take maternal HIV status into account when evaluating NIHF, as it may contribute to the condition through indirect mechanisms.

Pre-pregnancy counselling is essential to ascertain the optimal medical conditions of HIV-positive women for pregnancy. Aggressive preconception assessment and ART initiation can drastically decrease maternal viral load and mother-to-child transmission (MTCT). In this case, late commencement and inadequate antenatal attendance in early pregnancy resulted in unprecedentedly high viral load, potentially influencing foetal outcomes. Optimal prenatal care that includes adequate planning, achieving viral suppression before conception, and multidisciplinary management is critical to preventing complications and, possibly, associations with non-immune hydrops fetalis (NIHF). This case highlights the need for the integration of HIV care and reproductive planning to optimize safer pregnancies.

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Conflicts of interest

All authors declare no conflicts of interest.

Ethical

The patient provided verbal consent for the use of case details and images for publication.

Authors' contribution:

NKC: conceptualization, data curation, writing – original draft. AHA and CP: supervision, writing – review and editing. All authors have read and agreed to the published version of the manuscript.

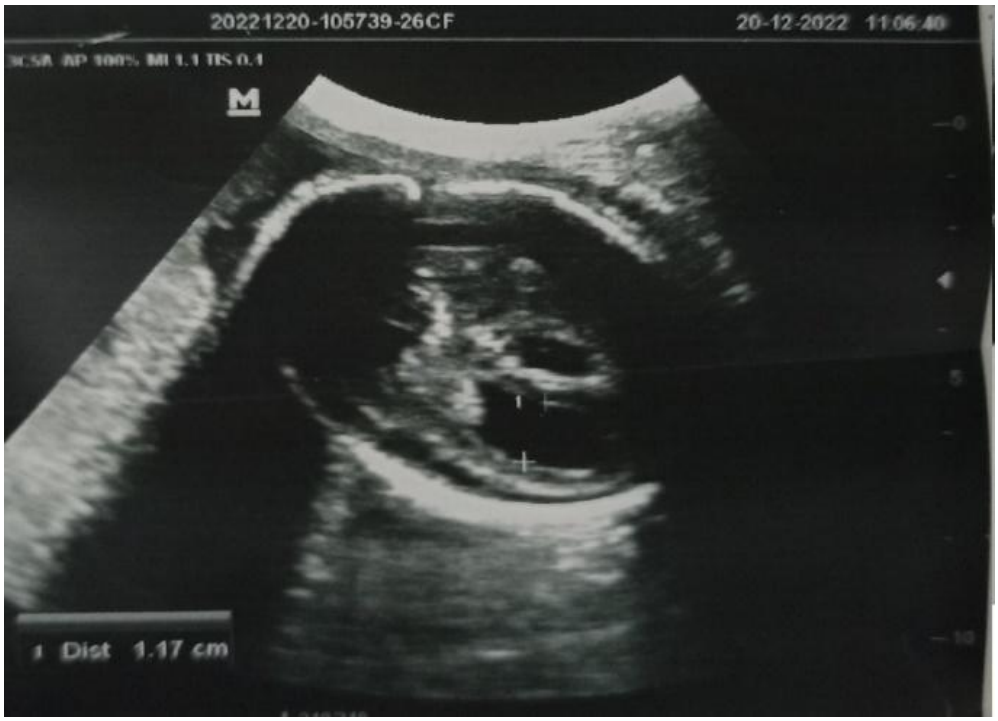

Figure 1. Left ventriculomegaly

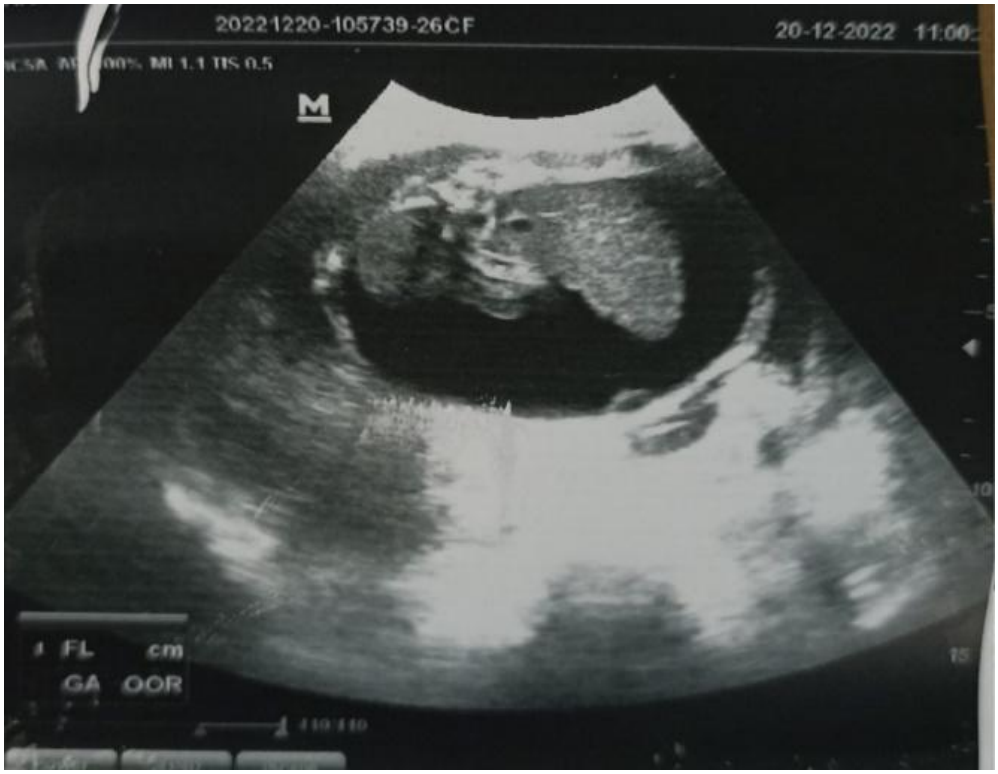

Figure 2. Ascites with enlarged spleen.

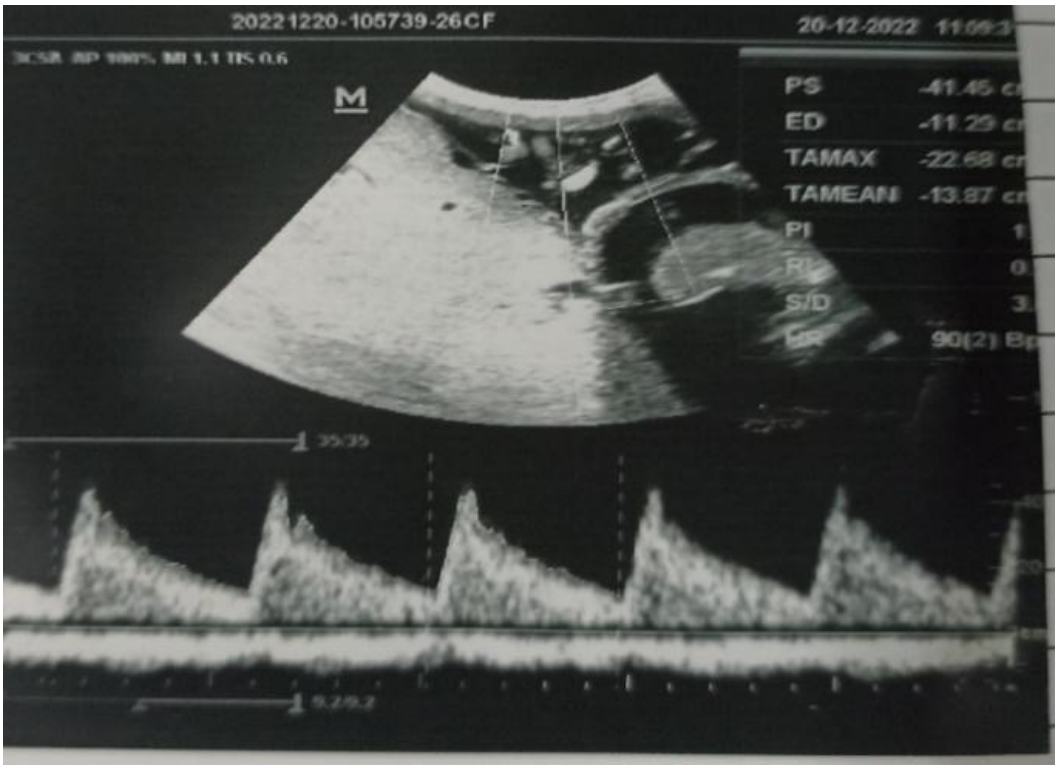

Figure 3. Umbilical artery doppler

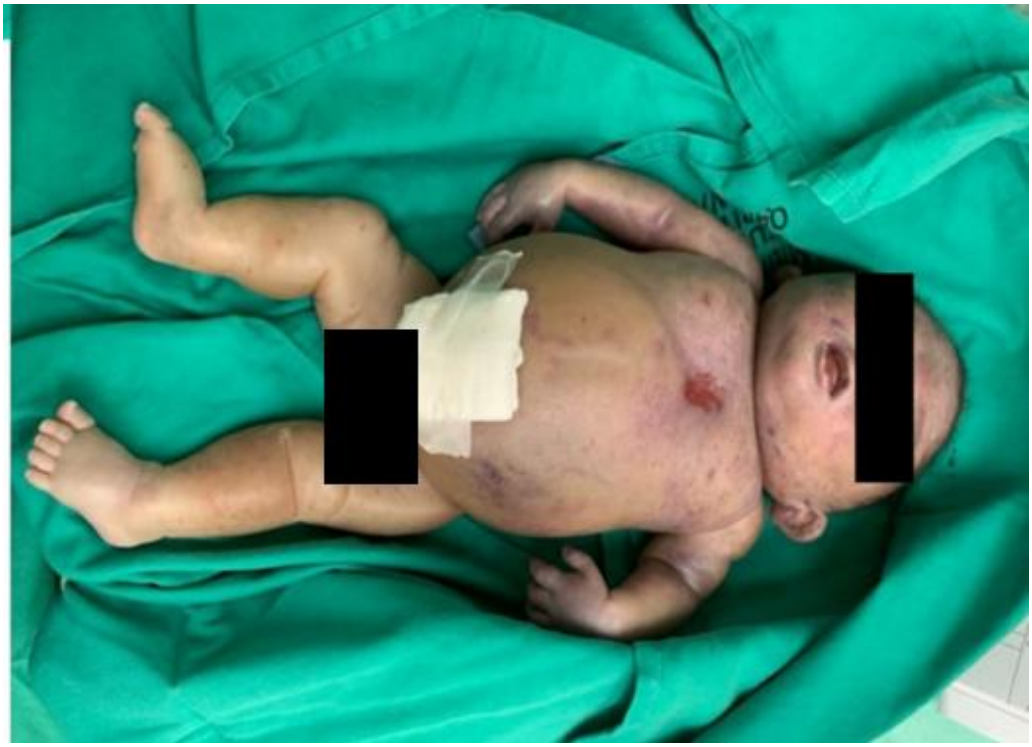

Figure 4. The new-born presented a hydrops fetalis

References

[1] Vanaparthy, R., Vadakekut, E. S. & Mahdy, H. *Nonimmune Hydrops Fetalis*. (2025).

[2] Hall, V., Vadakekut, E. S. & Avulakunta, I. D. *Hemolytic Disease of the Fetus and Newborn*. (2025).

[3] Norton, M. E., Chauhan, S. P. & Dashe, J. S. Society for Maternal-Fetal Medicine (SMFM) Clinical Guideline #7: nonimmune hydrops fetalis. *Am J Obstet Gynecol* **212**, 127–139 (2015).

[4] Khairudin, D., Alfirevic, Z., Mone, F. & Navaratnam, K. Non-immune hydrops fetalis: a practical guide for obstetricians. *The Obstetrician & Gynaecologist* **25**, 110–120 (2023).

[5] Bellini, C. & Hennekam, R. C. Non-immune hydrops fetalis: A short review of etiology and pathophysiology. *Am J Med Genet A* **158A**, 597–605 (2012).

[6] Sileo, F. G. *et al.*Non-immune fetal hydrops: etiology and outcome according to gestational age at diagnosis. *Ultrasound in Obstetrics and Gynecology* **56**, 416–421 (2020).

[7] Kadrofske, M. M. P., Parimi, et al. NONIMMUNE HYDROPS FETALIS AND HEPATITIS IN A. *Pediatr Infect Dis J* (2006).

[8] Wafelman, L. S., Pollock, B. H., et al. Nonimmune Hydrops fetalis: Fetal and Neonatal Outcome during 1983–1992. *Neonatology* **75**, 73–81 (1999).

[9] Davis, J. & Fernando, J. J. R. Successful management of prenatal hydrops fetalis in a mother with HIV. *Int J STD AIDS* **19**, 281–282 (2008).

[10] Hornberger, L. K. *et al. Cardiac Structure and Function in Fetuses of Mothers Infected with HIV: The Prospective P 2 C 2 HIV Multicenter Study*. (2000).