

Early Stabilization and Supportive Management of Congenital Ascites in Non-Immune Hydrops Fetalis: A Rare Case Report

Reny Widayanti^{1*}, Martono Tri Utomo², Risa Etika², Dina Angelika², Kartika Darma Handayani², Wurry Ayuningtyas², Manggala Pasca Wardhana³

¹Resident, Department of Child Health, Faculty of Medicine, Universitas Airlangga, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

²Lecturer, Department of Child Health, Faculty of Medicine, Universitas Airlangga, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

³Lecturer, Department of Obstetrics and Gynecology, Universitas Airlangga, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

Abstract

Background: Congenital ascites, considered part of the spectrum of non-immune hydrops fetalis (NIHF), is a rare clinical condition characterized by diverse underlying etiologies and variable outcomes, posing significant challenges in both diagnosis and management

Case Presentation: We report a term neonate who presented with massive ascites associated with respiratory compromise. Ascitic fluid analysis demonstrated an exudative profile, characterized by elevated protein levels and marked mononuclear cell predominance, raising suspicion of an underlying lymphatic disorder or chronic inflammatory process. Imaging studies revealed isolated ascites accompanied by bilateral hydroceles, with no other significant structural abnormalities. Cardiac and endocrine evaluations were unremarkable. During hospitalization, the patient developed pneumonia and subsequently received appropriate antimicrobial therapy.

Discussion: Management was performed using a structured, stepwise, and predominantly non-invasive approach, including escalation of respiratory support, fluid restriction, diuretic therapy, and gradual nutritional optimization. The patient showed progressive clinical gfhimprovement with resolution of ascites without invasive intervention. These findings support evidence that selected cases of isolated fetal ascites may respond favorably to conservative management. In addition, timely intrauterine interventions such as amnioreduction and fetal paracentesis have been associated with improved survival rates exceeding 90% in cases presenting after 28 weeks with surgically correctable etiologies.

Conclusion: This case highlights the importance of individualized, physiology-based management in neonates with congenital ascites, particularly when the underlying etiology remains uncertain. Careful integration of clinical findings, laboratory results, and imaging may facilitate appropriate therapeutic decision-making and contribute to favorable clinical outcomes.

Keywords: Congenital Ascites, Non-Immune Hydrops Fetalis (NIHF), Postnatal Stabilization.

Introduction

Background

Congenital ascites, also referred to as fetal ascites, is defined as the pathological accumulation of fluid within the fetal abdominal cavity and may present either as an isolated condition or as part of the spectrum of non-immune hydrops fetalis. Although relatively uncommon, this condition is clinically significant due to its association with a broad range of underlying etiologies and variable perinatal outcomes [1].

Recent evidence suggests that a structured diagnostic approach enables clinicians to identify the underlying cause of isolated fetal ascites in approximately 92% of cases [2]. Furthermore, timely intrauterine therapeutic interventions, such as amnioreduction and fetal paracentesis, have been associated with improved neonatal outcomes and survival rates exceeding 90%, particularly in cases presenting after 28 weeks of gestation and involving gastrointestinal or intra-abdominal etiologies amenable to surgical correction [3]. Overall, a favorable prognosis has been reported in more than 55% of cases [4]. Nevertheless, the effectiveness of these interventions remains highly dependent on the underlying etiology.

However, current literature predominantly focuses on etiology and prenatal management, with limited focus on postnatal stabilization. Importantly, neonates may initially present reassuring clinical parameters, such as favorable Apgar scores, yet remain at risk of rapid clinical deterioration due to impaired pulmonary expansion and fluid imbalance. This underscores the need for a better understanding of early neonatal management strategies. Therefore, this case report aims to describe the initial resuscitation and stabilization of a term neonate with congenital ascites, with particular emphasis on respiratory support and fluid management.

Case Presentation

A male neonate was delivered in the operating room of Dr. Soetomo General Academic Hospital, Surabaya, via cesarean section at 38 weeks of gestation. At birth, the neonate cried immediately and exhibited good muscle tone. The birth weight was 3950 g, with a birth length (BL) of 52 cm, head circumference (HC) of 35 cm, chest circumference (CC) of 36 cm, and abdominal circumference (AC) of 41 cm. The amniotic fluid was clear. The Apgar scores were 8 and 9 in 1 and 5 minutes, respectively.

The mother had received regular antenatal care throughout pregnancy. The mother denied any history of medication use other than prescribed vitamins and reported no complications, including preeclampsia, diabetes mellitus, or infection. Prenatal ultrasonography performed at 37 weeks of gestation demonstrated significant fluid accumulation within the fetal abdominal and thoracic cavities, accompanied by polyhydramnios. The presence of marked fetal ascites and thoracic fluid accumulation raised concern for progressive abdominal distension, impaired fetal respiratory mechanics, and potential hemodynamic compromise. Given these findings, fetal decompression was undertaken to reduce the intra-abdominal pressure and minimize the risk of further fetal deterioration. Fetal paracentesis was performed under ultrasonographic guidance, yielding approximately 950 mL of ascitic fluid. Following the procedure, a reduction in fetal abdominal distension was observed. Because polyhydramnios persisted, amnioreduction was subsequently performed, with approximately 500 mL of amniotic fluid removed. This intervention was intended to decrease uterine overdistension and the associated risks of maternal discomfort, preterm labor, and further fetal compromise. The obtained fetal ascitic fluid was subsequently analyzed. The analysis demonstrated an exudative profile, with a predominant mononuclear cell population (98.7%). The protein concentration was 3.68 g/dL (reference value <3 g/dL), while the glucose concentration was 66.60 mg/dL (reference value <100 mg/dL). The lactate dehydrogenase (LDH) level was 231 U/L. Following the procedures, the pregnancy was monitored with serial ultrasonographic examinations and maternal-fetal clinical assessments. The fetal abdominal distension and amniotic fluid volume were subsequently evaluated, with continued surveillance until delivery. The fetus remained clinically stable during the subsequent monitoring period and was delivered with a favorable condition at birth.

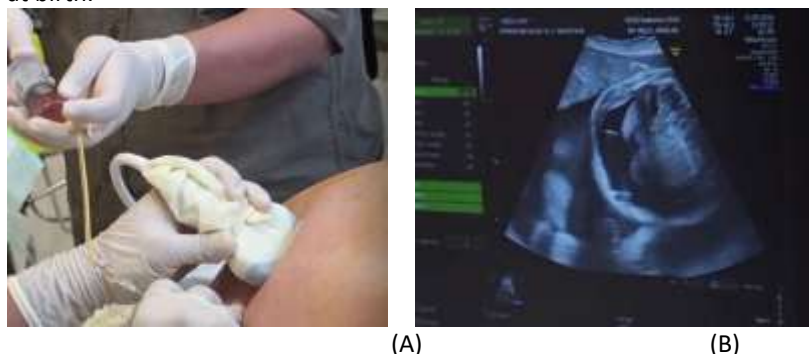


Figure 1. Ultrasound-guided fetal paracentesis during the procedure (A). Ultrasonographic image showing fetal ascites during the procedure (B).

On physical examination after birth, the neonate presented with diffuse abdominal distension. Vital signs were relatively stable, with a respiratory rate of 56 breaths per minute (regular), heart rate of 135 beats per minute (regular), temperature of 36.6°C, and oxygen saturation revealed 97%. Abdominal examination revealed generalized distension, normal bowel sounds, positive shifting dullness, and dullness to percussion, suggestive of ascites. Genital examination showed persistent bilateral scrotal enlargement with positive transillumination; both testes were palpable within the scrotum. Examination of the head, neck, thorax, and extremities was unremarkable.

Following delivery, the neonate received initial management in accordance with neonatal stabilization principles (STABLE), including thermoregulation, airway positioning, secretion clearance, and stimulation. The hemodynamic condition was stable (heart rate >100 beats per minute, strong cry), and no advanced resuscitation was required. The neonate was subsequently admitted to the Neonatal Intensive Care Unit (NICU) for observation, intravenous fluids (10% dextrose at approximately 60 mL/kg/day), and empirical antibiotics (ampicillin and gentamicin). Prophylactic interventions included intramuscular vitamin K, hepatitis B vaccination, and topical antibiotic eye ointment. Close monitoring of vital signs, blood glucose levels, and fluid balance was performed. During the initial NICU management, the neonate was maintained nil per os, and an 8 Fr orogastric tube was inserted, yielding minimal clear aspirate. Supportive care and close monitoring of respiratory and hemodynamic status were maintained, along with family education.



(A) (B)
Figure 2. Clinical neonate's condition at birth (A). Showing massive abdominal distension (AC = 41 cm). (B) Showing bilateral hydrocele.

Comprehensive diagnostic evaluations were conducted to establish the diagnosis and determine the underlying etiology. The complete routine blood test revealed normal hemoglobin (Hb 14.0 g/dL), normal leukocyte (Wbc $11.66 \times 10^3/\mu\text{L}$), normal Platelet ($345 \times 10^3/\mu\text{L}$), relatively stable coagulation (APTT 36.9s, PPT 13.5s), normal electrolyte profiles (sodium 141mmol/L, potassium 5.5 mmol/L, chloride 112.7 mmol/L), a non-significant elevation in transaminase levels (AST 85 U/L, ALT 15 U/L), normal renal function test (BUN 11.3 mg/dL, serum creatinine 0.53 mg/dL) and normal albumin serum (3.5g/dL) Neonatal thyroid-stimulating hormone (TSH) level was 1.77 mIU/L, which was interpreted as within normal limits (negative screening result), and 17-OHP (17-Hydroxyprogesterone) level was 2.1 nmol/L, which was far below the upper limit of the normal range. Chest and abdominal X-ray (Figure 2) showed no significant cardiopulmonary abnormalities but confirmed the presence of ascites. Abdominal ultrasonography (Figure 3) demonstrated ascites and Urogenital ultrasonography (Figure 4) demonstrated bilateral hydrocele. Echocardiographic findings were normal. Blood culture was negative.



Figure 3. X-ray showed ascites

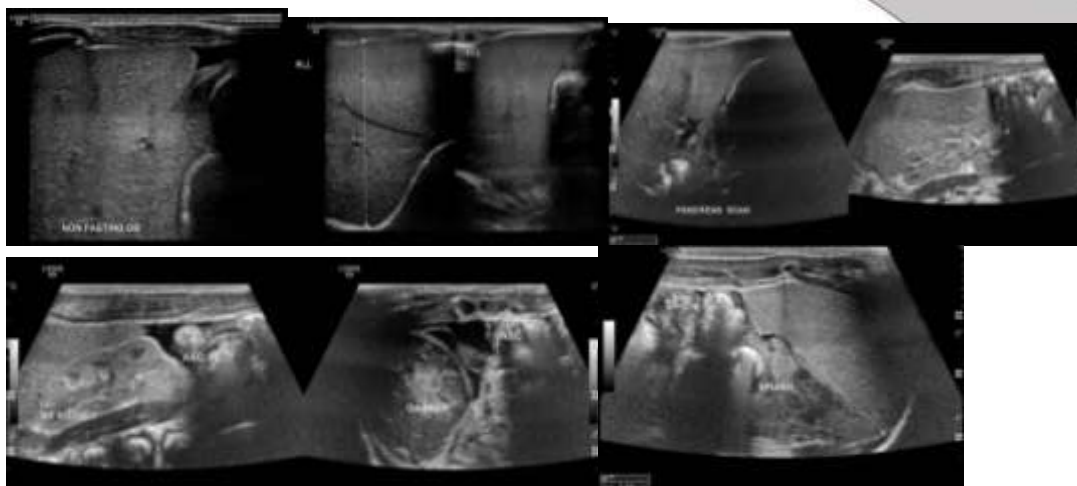


Figure 5. Urogenital ultrasound examination revealed a hydrocele

Based on the clinical and investigative findings, the diagnosis was consistent with neonatal ascites as a continuation of fetal ascites, within the spectrum of non-immune hydrops fetalis (NIHF), accompanied by bilateral hydrocele. The etiology of ascites remained under evaluation, with a multifactorial origin considered, including intrauterine infection, cardiovascular abnormalities, and lymphatic system disorders. Differential diagnoses included inborn errors of metabolism and lymphatic obstruction.

During hospitalization, management was adjusted according to the patient's clinical condition. On the fifth day hospitalization, the patient's clinical presentation and lung ultrasound findings were suggestive pneumonia. Respiratory support was escalated from room air to continuous positive airway pressure (CPAP) followed by noninvasive ventilation (NIV) as needed. Fluid therapy was gradually increased according to the patient's age and clinical condition, along with parenteral nutrition and gradual initiation of minimal enteral feeding with breast milk. Antibiotic therapy was guided by the patient's clinical condition and microbiological culture results.. Diuretic therapy with furosemide was administered to facilitate fluid reduction. A multidisciplinary approach involving neonatology, pediatric cardiology, and pediatric surgeon was implemented to manage complex clinical conditions.

After 13 days of hospitalization, the neonate was deemed clinically stable and suitable for discharge, with vital signs within normal limits. Objective evaluation demonstrated significant clinical improvement, as evidenced by a reduction in abdominal circumference from 41 cm to 37 cm, indicating substantial resolution of ascites. At outpatient follow-up with a pediatric specialist, continued clinical improvement was observed, with only minimal residual ascites. These findings suggest a favorable therapeutic response and a good short-term prognosis.



Figure 6. Significant clinical improvement with Abdominal Circumference (AC) = 37 cm at the post-stabilization follow-up visit.

Discussion

Congenital ascites within the spectrum of non-immune hydrops fetalis (NIHF) remains a diagnostically challenging condition due to its heterogeneous etiologies and frequently inconclusive investigations [2,5] In the

present case, ascitic fluid analysis demonstrated an exudative profile with elevated protein levels (3.68 g/dL) and marked mononuclear predominance (98.7%), indicating a non-transudative process. This pattern is more consistent with lymphatic or chronic inflammatory mechanisms than with cardiac, hepatic, or hypoalbuminemic etiologies, which typically produce low-protein transudates [6]. The coexistence of bilateral hydroceles further supports involvement of the lymphatic system, reflecting impaired drainage across serous compartments [7].

A systematic evaluation enabled the exclusion of several major etiological categories. Normal thyroid function (TSH 1.77 mIU/L) and 17-hydroxyprogesterone (17-OHP) levels effectively ruled out endocrine causes, while normal echocardiographic findings reduced the likelihood of cardiogenic hydrops [8]. Although congenital infections remain an important consideration in NIHF, particularly cytomegalovirus, syphilis, and parvovirus B19, the absence of maternal risk factors and supportive clinical findings reduce the likelihood of congenital infection, acknowledging that subclinical infection cannot be entirely excluded [9]. Abdominal imaging demonstrating isolated ascites with bilateral hydroceles and no evidence of obstruction further narrows the differential diagnosis toward lymphatic abnormalities, including congenital chylous ascites [10,11].

The clinical course provides additional diagnostic insight. The favorable response to diuretic therapy and supportive care suggests a functional and potentially reversible mechanism, consistent with transient lymphatic immaturity. This aligns with the current understanding that a subset of NIHF cases, particularly isolated fetal ascites, may demonstrate spontaneous or treatment-responsive resolution and generally have a more favorable prognosis compared with generalized hydrops [2,4]. Furthermore, the biochemical characteristics of the ascitic fluid in this case closely resemble those described in neonatal chylous ascites, which is typically exudative with lymphocyte predominance, supporting a lymphatic origin [6].

From a management perspective, this case highlights the effectiveness of a structured, physiology-based, non-invasive approach. Respiratory compromise due to elevated intra-abdominal pressure was successfully managed through stepwise escalation from nasal oxygen to continuous positive airway pressure (CPAP) and non-invasive ventilation (NIV), consistent with current neonatal respiratory care recommendations [12]. In parallel, careful fluid management—including fluid restriction, diuretic therapy, and gradual nutritional advancement—facilitated the mobilization of third-space fluid, as reflected by significant weight reduction and clinical improvement. This approach is supported by previous studies demonstrating that conservative management can be effective in selected cases of neonatal ascites, potentially avoiding invasive procedures [13].

The development of pneumonia during hospitalization highlights the vulnerability of neonates with ascites to respiratory complications. Increased intra-abdominal pressure, impaired diaphragmatic excursion, and immature airway protective mechanisms predispose neonates to aspiration and subsequent pulmonary infection. Neonates are particularly susceptible due to their immature immune systems and unstable physiological state, necessitating early recognition and prompt antimicrobial management [14].

These postnatal challenges underscore the importance of antenatal management aimed at reducing the mechanical effects of severe fetal ascites. In the present case, fetal paracentesis and amnioreduction were performed in the third trimester in response to marked fetal ascites accompanied by polyhydramnios. Antenatal drainage may have contributed to a favorable neonatal course by reducing the mechanical consequences of severe intra-abdominal and intra-amniotic fluid accumulation. Fetal paracentesis can decompress the fetal abdomen, thereby reducing intra-abdominal pressure and diaphragmatic compression, while amnioreduction may decrease uterine distension associated with polyhydramnios and facilitate prolongation of pregnancy [13]. A similar management strategy was reported by Sooklin et al., in which a fetus with severe congenital chylous ascites underwent fetal abdominal paracentesis with drainage of 625 mL of ascitic fluid and amnioreduction of 1,600 mL. Although the ascites subsequently recurred and the neonate developed respiratory distress after delivery at 33+2 weeks of gestation, the infant responded to postnatal management, with complete resolution of ascites and normal growth and neurodevelopment at 5 years of follow-up. This favorable long-term outcome following antenatal fluid drainage supports the potential role of fetal paracentesis and amnioreduction in the management of severe fetal ascites, particularly by reducing its mechanical effects and allowing continuation of pregnancy to a more advanced gestational age [13]. In the present case, the favorable neonatal outcome following drainage of 950 mL of fetal ascitic fluid and 500 mL of amniotic fluid is consistent with this previously reported experience. Although antenatal drainage may contribute to favorable neonatal outcomes, its effect should be interpreted alongside other prognostic determinants, particularly the underlying etiology, associated fetal abnormalities, gestational age at delivery, and subsequent postnatal management. In this context, Horgan et al. reported that survival in isolated fetal ascites varies according to etiology, with relatively favorable survival in idiopathic, chylous, gastrointestinal, and genitourinary causes, further supporting the importance of the underlying condition in determining prognosis [15].

Despite a comprehensive evaluation, definitive etiological confirmation was not achieved, reflecting a recognized limitation of NIHF, in which a substantial proportion of cases remain idiopathic [10]. Advanced

diagnostic modalities, including lymphangiography and genetic testing, may provide further clarification but are not always readily available. In such settings, integration of clinical findings, laboratory data, imaging, and therapeutic response remains essential for guiding management.

Overall, this case demonstrates that a stepwise, physiology-driven management strategy, combining antenatal fluid drainage with conservative postnatal management, can achieve effective stabilization and eventual resolution of congenital ascites. These findings reinforce the importance of individualized, physiology-driven care in neonates with NIHF, particularly in cases with uncertain etiology, and contribute to the growing evidence supporting non-invasive management in selected patients.

Conclusion

This case demonstrates that congenital ascites within the spectrum of non-immune hydrops fetalis can be effectively managed using a structured, physiology-based approach combining appropriately selected antenatal interventions with conservative postnatal management. The exudative ascitic fluid profile with marked mononuclear predominance, supported by clinical and imaging findings, suggests a probable lymphatic origin, although the absence of advanced diagnostic modalities, such as magnetic resonance lymphangiography and genetic testing, limited definitive etiological confirmation. Despite limitations related to diagnostic uncertainty and the single-case design, this report provides clinically relevant insights into individualized and pragmatic management of complex neonatal ascites. Antenatal fetal paracentesis and amnioreduction may have contributed to the favorable neonatal course by reducing the mechanical effects of severe fetal ascites and polyhydramnios and facilitating continued fetal maturation before delivery, although their independent contribution to the outcome remains uncertain. Ultimately, a pathophysiology-driven approach, with timely and appropriately selected antenatal and postnatal interventions, may be important in achieving favorable outcomes in selected cases.

References

- [1] Malhotra K, Elayedatt RA, Krishnan V. Fetal Ascites: Its Prenatal Management and Postnatal Outcome. *Journal of Obstetrics and Gynecology of India* 2024. <https://doi.org/10.1007/s13224-023-01913-1>.
- [2] Veluchamy M, Ramasamy K, Liyakat N. Isolated Fetal Ascites: A Rare Cause. *Cureus* 2020. <https://doi.org/10.7759/cureus.8433>.
- [3] Huang Y-Y, CY-J, CL-J, LC-H, CH-N, CJ-Y, CM, & HC-C. Survival of Hydrops Fetalis with and without Fetal Intervention. *Children* 2022;9:530.
- [4] Sablok A, Sharma A, Gupta R, Thakur S, Kaul A. Isolated Fetal Ascites. *J Indian Assoc Pediatr Surg* 2021;26:162–9. https://doi.org/10.4103/jiaps.JIAPS_57_20.
- [5] Swearingen C, Colvin ZA, Leuthner SR. Nonimmune Hydrops Fetalis. *Clin Perinatol* 2020;47. <https://doi.org/10.1016/j.clp.2019.10.001>.
- [6] Bhardwaj R, Vaziri H, Gautam A, Ballesteros E, Karimeddini D, Wu GY. Chylous ascites: A review of pathogenesis, diagnosis and treatment. *J Clin Transl Hepatol* 2018;6. <https://doi.org/10.14218/JCTH.2017.00035>.
- [7] Hoang VT, Van HAT, Hoang TH, Nguyen TTT, Trinh CT. A Review of Classification, Diagnosis, and Management of Hydrocele. *Journal of Ultrasound in Medicine* 2024;43. <https://doi.org/10.1002/jum.16380>.
- [8] Norton ME, Chauhan SP, Dashe JS. Society for maternal-fetal medicine (SMFM) clinical guideline #7: Nonimmune hydrops fetalis. *Am J Obstet Gynecol* 2015;212. <https://doi.org/10.1016/j.ajog.2014.12.018>.
- [9] Dinicu A, Penalosa P, Crosland BA, Steller J. Complete Resolution of Nonimmune Hydrops Fetalis Secondary to Maternal Syphilis Infection. *American Journal of Perinatology Reports* 2023;13:e21–4. <https://doi.org/10.1055/a-2028-7727>.
- [10] Vanaparthi R VEMH. Nonimmune Hydrops Fetalis. Treasure Island (FL): StatPearls Publishing 2024.
- [11] Romańska-Kita J, Borszewska-Kornacka MK, Dobrzańska A, Rudzińska I, Czech-Kowalska J, Wawrzoniak T. Congenital chylous ascites. *Pol J Radiol* 2011;76. <https://doi.org/10.5742/mejfm.2013.111207>.
- [12] Sweet DG, Carnielli V, Greisen G, Hallman M, Ozek E, te Pas A, et al. European Consensus Guidelines on the Management of Respiratory Distress Syndrome – 2019 Update. *Neonatology* 2019;115:432–50. <https://doi.org/10.1159/000499361>.
- [13] Sooklin L, Anand AJ, Rajadurai VS, Chandran S. Management of large congenital chylous ascites in a preterm infant: fetal and neonatal interventions. *BMJ Case Rep* 2020;13:e235849. <https://doi.org/10.1136/bcr-2020-235849>.

- [14] Sedney CJ, Harvill ET. The Neonatal Immune System and Respiratory Pathogens. *Microorganisms* 2023;11. <https://doi.org/10.3390/microorganisms11061597>.
- [15] Horgan R, Youssef JA, Levy AT, Berger SI, Dreux S, Brizot ML, Boutall A, Abuhamad AZ, Angarita AM, Al-Kouatly HB. Etiology and Outcome of Isolated Fetal Ascites: A Systematic Review. *Obstetrics & Gynecology* 2021. <https://doi.org/10.1097/AOG.0000000000004605>.