

# Ascites as first presentation of ventriculo-peritoneal shunt infection in a neonate

Walid I. El-Naggar, ABP, Ali Y. Mersal, MBBS, FRCP(C), Irfan A. Mamoun, MD.

---

## ABSTRACT

Infection is a common complication of ventriculoperitoneal shunts, and ascites is one of the rare manifestations of shunt infection. We report a neonate in whom shunt infection is presented only by ascites. The causative organism, coagulase negative staphylococci, was detected only in the cerebrospinal fluid although peritoneal fluid analysis was consistent with infection. Our patient shows the importance of considering shunt infection when unexplained ascites is the first and only manifestation in neonates with ventriculoperitoneal shunts.

Neurosciences 2005; Vol. 10 (1): 99-100

---

Infection is a common and serious complication of ventriculoperitoneal (VP) shunt in children. The usual manifestations of infection include fever, shunt malfunction, signs of meningitis and abdominal pain.<sup>1</sup> However, ascites is rare, especially when it becomes the first presentation for infection. We report a case to emphasize that ascites is a rare and unusual manifestation of neonatal VP shunt infection.

**Case Report.** A preterm baby boy, 33 weeks gestational age, a product of in-vitro fertilization, was referred to our neonatal intensive care unit at the age of 6 weeks due to post-hemorrhagic hydrocephalus and cholestatic jaundice. After stabilization of his condition and initial investigations, a VP shunt was placed. The cerebrospinal fluid (CSF) sample that was taken during the operation and sent for analysis and culture, showed no evidence of infection. The shunt functioned well with consequent reduction of the head circumference. Twelve days post-operatively, the baby was noticed to have progressive abdominal distention. His general condition was good, and he was tolerating feeds without gastrointestinal

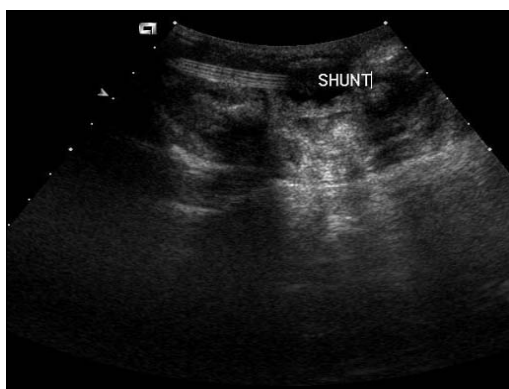
symptoms. Vital signs were stable, and the head circumference was not increasing. The abdomen was soft and lax, however, bowel sounds were diminished and ascites was suspected. Abdominal ultrasound confirmed the presence of ascites (Figures 1 & 2). Hepatic and renal functions were normal. The peritoneal fluid was turbid with a white blood cell (WBC) count of 15900/mm<sup>3</sup> (77% polymorphs), red blood cell (RBC) count of 1383/mm<sup>3</sup>, glucose concentration of 1.7mmol/L and protein content of 25g/L. The gram stain was negative as well as the culture. A CSF analysis revealed WBC count of 33/mm<sup>3</sup> (33% polymorphs), RBC count of 110/mm<sup>3</sup>, glucose concentration of 0.7 mmol/L (<50% of blood sugar) and protein of 1.3 g/L. The culture showed growth of coagulase negative staphylococci. The patient was started on vancomycin. The VP shunt was removed and external ventricular drainage was created. The patient continued on vancomycin for 14 days. After complete clearance of infection and regression of ascites, a new ventriculoatrial shunt was inserted. The new shunt functioned well and the patient was discharged home in good condition.

---

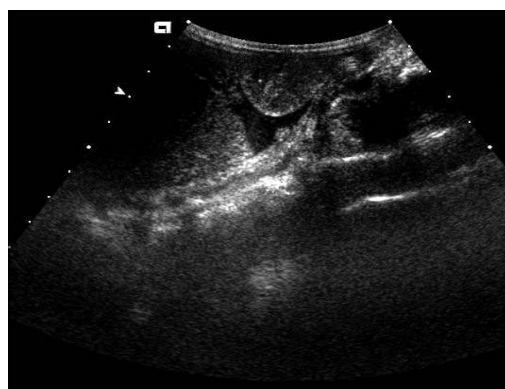
From the Department of Pediatrics, Division of Neonatology (El-Naggar, Mersal) and the Department of Medical Imaging (Mamoun), King Faisal Specialist Hospital and Research Centre, Jeddah, Kingdom of Saudi Arabia.

Received 21st March 2004. Accepted for publication in final form 5th June 2004.

Address correspondence and reprint request to: Dr. Ali Y. Mersal, Department of Pediatrics, Division of Neonatology, King Faisal Specialist Hospital and Research Centre, PO Box 40047, MBC J-58, Jeddah 21499, Kingdom of Saudi Arabia. Tel. +966 55548439. Fax. +966 (2) 667777 Ext. 3508. E-mail: alimersal@dr.com



**Figure 1** - Abdominal ultrasound showing the ventriculoperitoneal shunt located anteriorly in the peritoneal cavity.



**Figure 2** - Abdominal ultrasound showing minimal ascites in the lower abdomen.

**Discussion.** Infection of a VP shunt is estimated to occur in 2-21% of cases.<sup>2</sup> It has been reported to occur within 6 months following implantation of the shunt in 86% of all patients.<sup>1</sup> The most common and important clinical manifestations associated with shunt infection in children are headache, nausea and vomiting due to shunt insufficiency and increased intra-cranial pressure. Abdominal manifestations such as abdominal discomfort, progressive distension, and feeding intolerance are also noted. In neonates, however, temperature instability, increased head circumference, lethargy and vomiting are usually the presenting signs. On the other hand, ascites is a rare complication. It is either due to increased CSF production as in choroid plexus papilloma<sup>3</sup> or inability of the peritoneum to absorb CSF secondary to low grade peritoneal inflammation.<sup>4,5</sup> Peritoneal inflammation interferes with lymphatic drainage, and the resultant protein accumulation increases peritoneal colloid osmotic pressure leading to further fluid collection.<sup>6</sup> Cerebrospinal fluid ascites without a predisposing infection was postulated to be due to the high protein content,<sup>7</sup> but many children with elevated CSF protein have had shunts placed without any untoward effect. In the absence of infection, no definite explanation has been offered for the inability of the peritoneum to absorb the CSF.<sup>8</sup> Goodman and Gourley<sup>6</sup> reported an 11-year-old girl with VP shunt, *Escherichia coli* urinary tract infection and ascites. She presented also with progressive abdominal enlargement only. Her shunt infection however, was secondary to the peritonitis. In our patient, who was a neonate with shunt infection, none of the common manifestations were noted. The patient rather had progressive abdominal distension due to ascites. Although the analysis of CSF and peritoneal fluid was consistent with infection in our patient, only the CSF showed positive culture of coagulase negative staphylococci, which is the common pathogen of VP shunt infection. This would favor that the development of ascites was secondary to shunt

infection instead of a primary peritoneal infection. These findings differentiate our case from the previous report of Gaskill and Marlin<sup>9</sup> who described 7 patients with VP shunts and spontaneous bacterial peritonitis caused by enteric pathogens.

In conclusion, although it is rare, ascites could be the earliest manifestation of VP shunt infection in neonates. We suggest to consider shunt infection if unexplained ascites develops in a neonate with a VP shunt. Early abdominal ultrasound and diagnostic paracentesis should be performed to confirm the diagnosis. Establishment of an alternative route for CSF diversion is also warranted.

## References

1. Kontny U, Hofling B, Gutjahr P, Voth D, Schwarz M, Schmitt HJ. CSF shunt infections in children. *Infection* 1993; 21: 89-92.
2. Odio L, McCracken GH, Nelson JD. CSF shunt infection in pediatrics: A seven year experience. *Am J Dis Child* 1984; 138: 1103-1108.
3. Ray P, Peck F Jr. Papilloma of the choroid plexus of the lateral ventricles causing hydrocephalus in an infant. *J Neurosurg* 1956; 13: 405-410.
4. Rosenthal JD, Golden GT, Shaw CA, Jane JA. Intractable ascites: a complication of ventriculoperitoneal shunting with a silastic catheter. *Am J Surg* 1974; 127: 613-614.
5. Adegbite AB, Khan M. Role of protein content in CSF ascites following ventriculoperitoneal shunting. *J Neurosurg* 1982; 35: 474-476.
6. Goodman GM, Gourley GR. Ascites complicating ventriculoperitoneal shunts. *J Pediatr Gastroentrol Nutr* 1988; 7: 780-782.
7. Tang TT, Whelan HT, Meyer GA, Strother DR, Blank EL, Camitta BM, et al. Optic chiasm glioma associated with inappropriate secretion of antidiuretic hormone, cerebral ischemia, nonobstructive hydrocephalus and chronic ascites following ventriculoperitoneal shunting. *Childs Nerv Syst* 1991; 7: 458-461.
8. Chidambaram B, Balasubramaniam V. CSF ascites: a rare complication of ventriculoperitoneal shunt surgery. *Neurol India* 2000; 48: 378-380.
9. Gaskill SJ, Marlin AE. Spontaneous bacterial peritonitis in patients with ventriculoperitoneal shunts. *Pediatr Neurosurg* 1997; 26: 115-117.