Role of protein content in CSF ascites following ventriculoperitoneal shunting

Case report

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Since the use of the peritoneal cavity for the shunting of cerebrospinal fluid (CSF) was first reported late in the 19th century by Ferguson,8 reports of complications attendant on its use have grown in number.1,2,9,15 One of these complications, CSF ascites, has been reported only 12 times in the literature.2,4,6,9,10,12–16,18,20 The etiology has been attributed to various factors, but little or no mention has been made of an elevated CSF protein level being a predisposing factor. We are reporting an additional case of ascites, which was associated with a ventriculoperitoneal (VP) shunt placed for treatment of hydrocephalus and elevated CSF protein levels. The CSF ascites did not recur after repeat VP shunting when the protein in the CSF had returned to normal levels following resection of the craniopharyngioma.

Case Report

This 6-year-old girl was first admitted to the neurosurgical unit because of headache, vomiting, and ataxia in April, 1975. Her left eye was blind, and she had a temporal field defect on the right side. Investigations revealed a suprasellar mass lesion, which was partly resected through a left frontal craniotomy and was found to be a craniopharyngioma. She subsequently received a course of radiotherapy. She then developed pituitary insufficiency and required maintenance therapy. This child remained well until September, 1980, when she began to suffer from recurrent headaches, nausea and vomiting, ataxia, urinary incontinence, and increasing lethargy. Computerized tomography (CT) scans revealed moderate hydrocephalus and evidence of a recurrent craniopharyngioma (Fig. 1). A VP shunt was placed, and her symptoms rapidly disappeared. Ventricular CSF protein content was 400 mg%. About 4 weeks after the shunting procedure, the patient's abdomen became progressively distended but her neurological status remained unchanged. Physical examination revealed abdominal distention, shifting dullness, and a palpable fluid wave. Her abdominal girth was 83 cm. She was afebrile. Plain abdominal radiographs suggested free peritoneal fluid and no demonstrable mass. Abdominal ultrasonic studies indicated the presence of a large volume.
of intraperitoneal fluid which moved freely (Fig. 2). A repeat CT scan showed resolution of the hydrocephalus.

Her abdominal girth increased to 87.5 cm within 3 days after admission, and fell to 77.5 cm after 3500 ml of clear fluid was removed from the peritoneal cavity. The ascitic fluid had a specific gravity of 1.011, a sodium content of 150 mEq/liter, potassium 3.7 mEq/liter, and chloride 118 mEq/liter. Levels in the serum measured on the same day were 150 mEq/liter for sodium, 3.9 mEq/liter for potassium, and 106 mEq/liter for chloride. There were a few lymphocytes but no abnormal cells or cholesterol crystals in the ascitic fluid. The glucose and protein levels in the ascitic fluid were 90 mg% and 1.2 gm%, respectively, and in the ventricular CSF were 97 mg% and 620 mg%, respectively. Cytological examination of the CSF revealed 136 red cells/cu mm and 16 white blood cells/cu mm, with a mononuclear cell predominance. Cultures of both CSF and ascitic fluid were negative.

The VP shunt was replaced by a ventriculoatrial shunt. One week later, the patient again suffered from headache, nausea, and vomiting, and the vision in her right eye had deteriorated rapidly. Repeat CT scanning showed further enlargement of the ventricles, and shunt patency studies indicated obstruction at the atrial end of the system. She underwent a second craniotomy for gross total removal of the craniopharyngioma. Her symptoms subsided, and vision in the right eye returned to its previous state. She was discharged 10 days after surgery.

The patient remained well for 2 months but then developed severe headaches and within a few days became stuporous, with hyperactive deep-tendon reflexes and bilateral extensor plantar responses. A third series of CT scans showed hydrocephalus. The CSF was again removed from the shunt reservoir, producing mild temporary clinical improvement. The CSF protein ranged from 18 to 55 mg%. The ventriculooatrial shunt was replaced by a VP shunt, and symptoms resolved within 24 hours. The patient was discharged 1 week after surgery when her abdominal girth was 75 cm. She remains well at 12 months follow-up examination, with a patent VP shunt, resolution of hydrocephalus, and absence of ascites.

**Discussion**

Cerebrospinal fluid ascites as a complication of VP shunting has been reported in a variety of cases, the clinical details of which suggest a multiplicity of causative or predisposing factors. It has been postulated that the development of CSF ascites might be related to a decrease in the absorptive function of the peritoneum or overproduction of CSF beyond the normal absorptive capacity of the peritoneum. Functional immaturity of the membrane during the first few months of life, leading to peritoneal malabsorption and development of ascites, has been proposed as a cause in infants, but CSF ascites was not reported in the series by Grosfeld, et al., in which a relatively high intraabdominal complication rate of 24% was recorded in 185 infants. A history of shunt revision or previous abdominal surgery has been present in a few patients who developed CSF ascites or local accumulation of CSF in the peritoneal cavity. It is not unlikely that peritoneal absorption was impaired by such procedures. The same may be said of peritoneal infections that might conceivably result in blockage of the draining lymphatic system by cellular debris. Immune reaction secondary to a routine diphtheria-pertussis-tetanus (DPT) injection has also been mentioned as a predisposing factor.

In our patient, the CSF protein was considerably elevated prior to removal of the recurrent craniopha-
CSF protein in ascites after VP shunting

ryngioma. However, after excision of the tumor, with subsequent reduction in CSF protein to normal levels, ascites did not recur when a VP shunt was reintroduced. It appears, therefore, that the absorption of CSF was impaired by its high protein content. It has been established that absorption of protein from the peritoneal cavity in experimental animals is achieved mainly through the lymphatic ducts under the diaphragm. The presence of stomata between peritoneal cells in those animals has been demonstrated. We believe that the absorptive capacity of the exit pathways for protein molecules could conceivably be overwhelmed by a massive increase in the number and/or in the size of the protein molecules. The increased protein in ascitic fluid could result either from increased CSF protein or from peritoneal inflammation. In the latter situation, obstruction of the lymphatic system by cellular debris would likely contribute to ascitic fluid formation. Other factors are, however, likely to be operative in cases where ascitic fluid protein is relatively low, for example, in the case reported by Rosenthal, et al.

Previous reports of abdominal ascites following VP shunting frequently refer to the protein content of the ascitic fluid, but Weidmann also made specific reference to a high CSF protein content. The protein level in the ascitic fluid in our patient was considerably higher than the CSF protein content. This might be due to the addition of protein from the peritoneal tissues in the presence of impairment of protein absorption and/or to the reabsorption of protein-free fluid.

In conclusion, it would seem reasonable to suggest that in any case of CSF ascites, the CSF protein level should be determined, since it may help in elucidating the pathogenesis of the ascites and in guiding further management in certain cases.

References


Manuscript received January 21, 1982. Accepted in final form April 14, 1982.

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