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# E. coli meningitis as an indicator of intestinal perforation by V-P shunt tube

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**Abstract**

Shunting systems widly used for the treatment of hydrocephalus have been connected with certain risks and potential complications which stem from implanting a non-biological material into the human body. Several complications have been reported to arise from the insertion of ventriculo-peritoneal shunts.

This short report highlights the observation that when anaerobic meningitis together with gram negative E. Coli in the CSF of a V-P shunted patient is found, bowel perforation should be assumed. Four cases developed such a complication among 643 hydrocephalic patients of various etiology over a period of ten years.

**Keywords:** Hydrocephalus, ventriculo-peritoneal shunt, meningitis, E. coli.

## 1 Introduction

The potentially serious complications of the V-P shunt system which may develop during the course of treatment of hydrocephalus include, among others, infection [4, 7, 8, 12 ] blockage [9], pseudocyst formation [14, 17], extrusion [2, 15, 16], intestinal obstruction [6], volvolus [18], peritoneal transhepatic migration [13], CSF hydrocele [3] and perforation of the diaphragm. Several reports have described perforation of the hollow viscosis, particularly the gut [1, 10, 19, 20]. The following is a description of four unusual cases of gut perforations (2 adults and 2 children) encountered among 643 hydrocephalus patients treated at King Fahd Hospital between 1985–1995. In all these cases, the gut was perforated by the peritoneal catheter and the perforation was suspected on the basis of the unusual enteric pathogens found in the CSF. Isolation of gram negative enteric bacilli indicated the possibility of intestinal perforation by the peritoneal catheter. Contrary to the recent reports [1, 5], all of our cases with perforation of the bowel presented with repeated attacks of meningitis. In fact, it was these attacks and the unusual gut pathogens isolated and grown from the CSF which alerted us to the possibility of the bowel perforation. Three of these cases had a functioning shunt system at the time of infection. One of the cases with a demonstrable bowel perforation had an associated clinical peritonitis and unfortunately died.

## 2 Case report

### CASE 1

A 55 year old American women was admitted to the Neurosurgery Service of KFHU with the diagnosis of recurrent attacks of bacterial meningitis. The latest occurred while on a holiday in Pakistan during Christmas of 1987. The woman had been diagnosed as suffering from normal pressure hydrocephalus and a spring coiled shunt was inserted in the USA in October, 1986. She suffered her first attack of meningitis six months after the insertion of the shunt; this was treated elsewhere and she was able to return to work.

In December, 1987, she was struck by another attack of meningitis which required hospitalization. She was later transferred and admitted to our service on the 29th of December, 1987. On admission, she looked quite ill and toxic and had fever of 39.7 °C.

She had neck rigidity and photophobia, and no other source of infection except her CSF which grew Staphylococcus epidermidis and Escherichia coli. The protein level was elevated as well as the number of total white cells, 90 % were polymorphs. She was initially given methicillin and gentamycin which partially controlled her meningitis for one week. However, on the 3rd of January, 1988, the fever relapsed and there was still evidence of infection. A valvogram demonstrated the lower end of the shunt perforating the bowel (Figure 1) which was obviously the source of the ascending infection. The shunt was removed with no evidence of postoperative meningitis. She remained on systemic antibiotic therapy until her symptoms subsided, and two weeks later she had a new system inserted without complications. She remained quite well and had no evidence of infection two years later.

## CASE 2

A 35 year old Saudi female was diagnosed to have a cervical cordoma in January, 1985. The patient had had a cervical laminectomy with a partial excision of the cordoma followed by a course of radiotherapy. Her spastic tetraparesis improved gradually and she was ambulatory, but in October, 1986, she had her first recurrence associated with obstructive hydrocephalus. The tumor had extended into the clivus and caused the obstructive hydrocephalus. The second operation was undertaken with more resection of the tumor and insertion of the V-P shunt for hydrocephalus. She did well for another two years, but in August, 1988, she had her third recurrence. This time, the tumor involved and invaded the arch of C1 and the body of C2 and the lower part of the clivus. Transoral resection of the recurrent tumor was undertaken between August and September, 1989. She had 3 attacks of meningitis which were treated elsewhere. In October, 1989, she presented with a one week history of headache, neck pain, fever and projectile vomiting. On admission, she was irritable, febrile, and had tachycardia. Her neck was extremely stiff from the stabilization loop and she had a residual left hemiparesis and bilateral extensive plantar response.

Meningitis was suspected and CSF culture obtained from the valve grew Escherichia coli, Proteus mirabilis, and Streptococcus group D. The valvogram showed penetration of the shunt into the large bowel (Figure 2). The shunt was removed and she received appropriate antibiotics until her symptoms subsided totally. She did not require the reinsertion of the shunt and she survived after this procedure for 18 months.

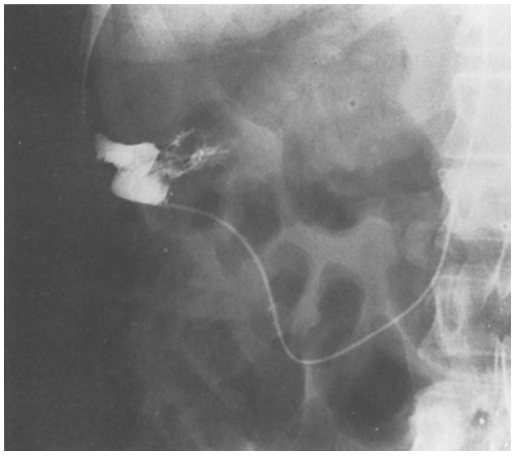

**Figure 1.** Valvogram showing the peritoneal tube (Raimondi spring coiled perforation the hepatic flexure and the radiopaque partially outlining the colon).

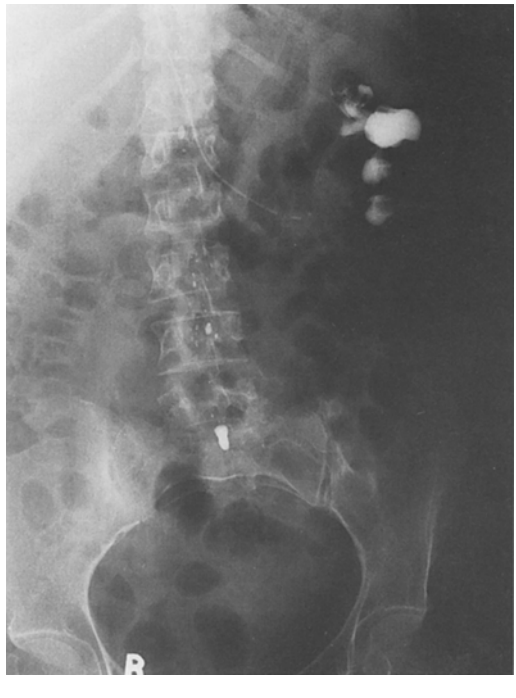

**Figure 2.** Peritoneal tube of V-P shunt penetrating the left side of the transverse colon. Dye outlining the splenic flexure.

### CASE 3

An 8 year old Saudi male had, at the age of 16 months, developed post-meningitic hydrocephalus. The child received the antituberculous treatment for his TBM, and the hydrocephalus was treated by V-P shunt at the age of two years (1986). The sequelae of meningoencephalitis left the child with cerebral palsy, spastic tetraparesis, and epilepsy. In 1988, he presented with repeated attacks of fever, vomiting, and drowsiness. The CSF white count was 12,700/mm$^3$ with protein elevated to 192 mg/ml. CSF culture grew E. coli. The child was started on the appropriate antibiotic, but the meningitis persisted. A week later, Pseudomonas and Streptococcus group D were isolated also from his CSF in addition to the original E. coli. A valvogram was performed on the child which clearly showed the perforation at the lower end of the V-P shunt into his descending colon. The shunt was removed and the antibiotics continued for three weeks, until the CSF analysis returned to normal. Four weeks later, a fresh Cordis Hakim medium pressure valve was inserted into his peritoneum. The child has remained quite well.

### CASE 4

This is a 4 year old Saudi child who had been diagnosed to have hydrocephalus secondary to congenital aqueduct stenosis after birth. The child was treated with Cordis V-P unishunt and did well for three years when he had the first revision. His parents abandoned him at the hospital for almost a year. Ten months after his revision, he developed three attacks of meningitis, 4 to 6 weeks apart. E. coli was isolated in his CSF and shortly after he proved to have a colonic perforation caused by the peritoneal catheter of his V-P shunt. The child developed peritonitis after the removal of the shunt and died shortly after, despite the aggressive antibiotic therapy.

## 3 Discussion

Complications of the V-P shunts are not uncommon. Growthfield [10] et al reported an abdominal complication rate of 24 % in their series requiring shunt revision. The first gut perforation was described by Wilson [21] et al in 1996. Although one of the two cases which he reported had died, retrograde infection of the brain was not suspected in either patient. Hornig [11] (1990) added two more cases to the 40 perforations documented worldwide. All four of my cases developed ascending retrograde meningitis caused by the bacterial invasion of the brain from the gut. All valves were functioning at the time of the diagnosis. Two of the cases were adults, one of them had a Raimondi spring coiled peritoneal tube which has been reported to be implicated in more than 50 % of the documented perforation cases [19, 20]. However, the rest of the cases had ordinary silastic tubes of Holter and Cordis shunts. Out of the four cases, one died signifying the potentially lethal outcome in this particular complication [10]. It is also worth stressing that when a mixed growth of bacteria, particularly gram negative, is obtained from the CSF of a V-P shunted patient, bowel perforation should be assumed until discounted. These four cases presented a common feature of this complication. The mechanism of peritoneal tube perforation remains largely unproven. Adhesions around the tip of the peritoneal catheter which facilitate the catheter perforation is a possibility [1]. However, mechanical rubbing against the gut wall resulting in necrosis which leads to the perforation has been suggested as an alternative mechanism [5]. Ascending infection has been suggested to occur more commonly when perforation is caused by functioning shunts [19] (58 %), while the chances are much less (33 %) if the gut was perforated by the lower end of a non-functioning shunt system. Three out of the four cases, indeed had functioning valves at the time of surgical removal.

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