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Case Report

Diaphragm perforation, a rare complication of V-P shunt surgery

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Abstract

Ventriculoperitoneal shunt surgery is the most widely used procedure in the treatment of hydrocephalus. However, this invasive procedure has been associated with several delayed complications. A 64-year-old male had unusual pneumonia caused by diaphragm perforation due to a ventriculoperitoneal (V-P) shunt catheter 3 years after V-P shunting for secondary normal pressure hydrocephalus after thalamic hemorrhage. Aspiration of hydrothorax of cerebrospinal fluid and removal of the catheter is the only treatment option for patients to alleviate such a lethal pneumonia. Probable causes of this rare complication are discussed, and attention is drawn to the possibility of its appearance and early recognition of respiratory distress.

Key words: normal pressure hydrocephalus, V-P shunt, perforation, diaphragm

Introduction

Spontaneous bowel perforation is a rare complication of V-P shunt surgery, occurring in only 0.01% to 0.07% of patients (Oshio et al., 1991; Rush et al., 1982). However, due to its high mortality rate of 15% (Snow et al., 1986), it is important to be able to recognize its occurrence regardless of its varied symptomatology. Most cases involve the lower abdomen, colon, rectum, vagina, and so on. Only 5 cases of supradiaphragmatic or transdiaphragmatic migration of the V-P shunt catheter have been reported. We also report a case of a very rare complication, diaphragm perforation manifesting cerebrospinal fluid hydrothorax. Migration of the peritoneal end of a ventriculoperitoneal shunt catheter into the chest accounts for the majority of thoracic complications. Diaphragm perforation caused by a V-P shunt catheter is one possible complication for long-term-bedridden patients and/or those with postoperative pleural adhesion.

Case Presentation

The patient had hypertensive thalamic hemorrhage, and ventricular dilatation with typical periventricular lucency gradually appeared, manifesting secondary normal pressure hydrocephalus. V-P shunt operation was enforced for this normal pressure hydrocephalus (NPH) secondary to thalamic hemorrhage. However, ventricular enlargement did not improve even after this, and V-P shunt operation was enforced again via opposite ventriculostomy. However, unfortunately, even though a collect shunt function was confirmed by shuntgraphy, the ventricular enlargement hardly improved (Fig. 1). In this patient, the abdominal end of the catheter gradually migrated to the upper abdominal cavity even after inserting it into the lower abdominal cavity. The migration of the catheter might have been due to peritoneal adhesion from two major open surgeries of the abdomen; one involved removal of a renal stone and the other removal of a gallstone by open abdominal surgery. In order to avoid abdominal

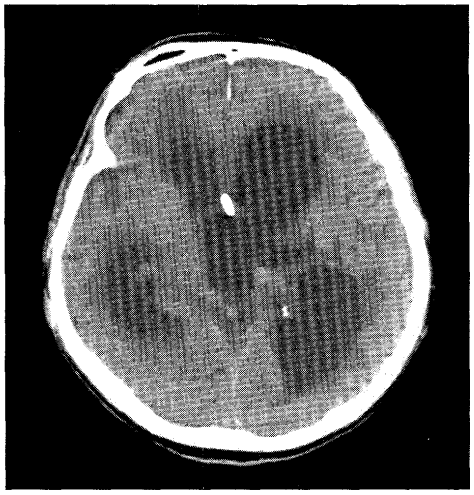

Fig. 1 CT demonstrating hydrocephalus. Normalization of the ventricle size couldn't be achieved even after V-P shunt revision. The patient was admitted in a stuporous condition to our hospital after V-P shunt surgery secondary to left thalamic hemorrhage. The ventricle size was very large with peri ventricular lucency; however, it did not show any change even after V-P shunt revision

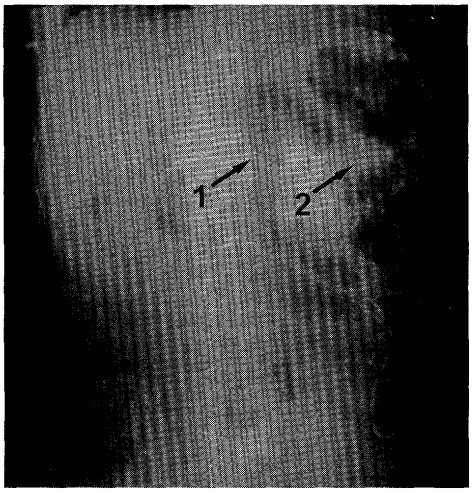

Fig. 2 (A) Radiograph of the abdomen before perforation demonstrating each end of the catheter migrating to the upper part of the abdominal cavity, perhaps due to peritoneal adhesion from the repeated open abdominal surgery

complications, ventriculoatrial (V-A) shunt revision should be planned as an alternative shunting root for further shunt revision. Three years after the V-P shunt revision, unfortunately, left cerebrospinal fluid hydrothorax was revealed by a chest roentgenogram (Fig. 2 (A) and 2 (B)) that was enforced in order to search for the origin of fever and respiratory distress. The abdominal-end of the V-P

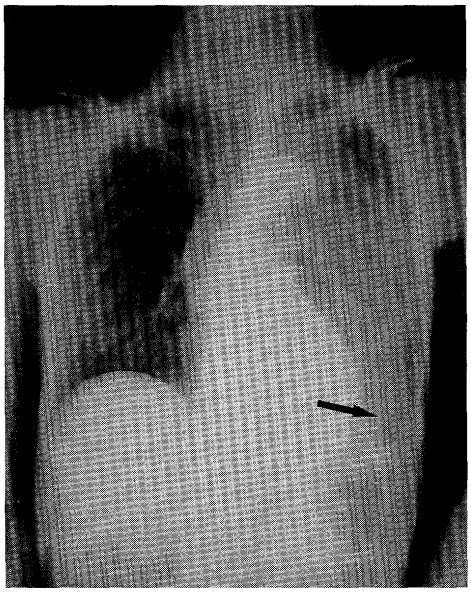

Fig. 2 (B) Radiograph of the chest demonstrating migration of the catheter (arrow head) into the thorax. The left cerebrospinal fluid hydrothorax is revealed

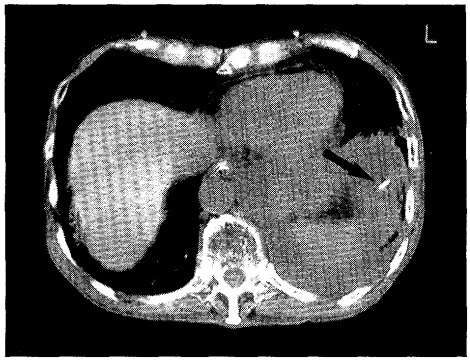

Fig. 3 CT scan of the chest demonstrating diaphragm perforation (arrow head)

shunt catheter had perforated the diaphragm, and it was confirmed that the end of the catheter was located in the supradiaphragm and epipleural space by chest rentogenography and chest CT scan (Fig. 3). CSF collection was immediately aspirated via thoracocentesis. The abdominal-end of the catheter was removed promptly by under local anesthesia. The entire appearance of the catheter was found to be normal. Collect function of the shunt system was also confirmed by pumping maneuvers of the flashing device just before removing the catheter.

Discussion

Ventriculoperitoneal shunting is a standard treatment option for hydrocephalus. Perforation is one of the rare late complications of V-P shunt. Diaphragm perforation is very rare in this area. Only six reports were found in the literature including this report. Generally, the end of the abdominal catheter is inserted into the Dougrous fossa. However, the tip of the abdominal catheter may migrate into the upper abdomen, especially in bed-ridden patients with peritoneal adhesion via repeated surgery, and special attention concerning diagram perforation via a V-P shunt catheter might be necessary for such patients. Although thoracic complications of ventriculoperitoneal shunts are rare, they are potentially serious. A mechanism of diaphragm perforation is suspected whererby the tip of the catheter is compressed by the diaphragm with respiratory movements, the diaphragm is degenerated, and finally perforated.

In our case, indeed, the catheter perforated the diaphragm 3 years after V-P shunt operation, manifesting cerebrospinal hydrothorax and pneumonia symptoms. Both Obrador and Villarejo (Obrador et al., 1977) and Dickman et al. (Dickman et al., 1989) hypothesized in their own cases that the shunt had been unwittingly passed into and out of the pleural cavity, probably in the supraclavicular fossa, during the distal tunneling procedure. Negative inspiratory pressure would then have slowly drawn the entire distal shunt catheter into the chest, with the tip entering the chest last. This may have occurred as well in the other two cases of supradiaphragmatic migration (Agha et al., 1983; Cooper et al., 1978). In four other cases and in the case reported here, migration into the chest was transdiaphragmatic (Anegawa et al., 1986; Gaudio, et al., 1988; Lourie et al., 1985; Rao et al., 1977; Taub et al., 1994). In no case could it be determined whether the tip of the shunt traversed a preexisting hiatus in the diaphragm or eroded through it. Congenital hiatuses in the diaphragm may occur posterolaterally, between its costal and lumbar portions (foramen of Bochdalek), or anteromedially, between its sternal and costal portions (foramen of Morgagni); when an actual foramen is not present, these locations are the weak points of the diaphragm (Romanes et al., 1981). Occasional cases of presumably congenital Bochdalek and Morgagni herniae presenting in adulthood and in old age suggest that such a hiatus may be present in some asymptomatic persons, thus providing a pathway for migration of the tip of a ventriculoperitoneal shunt into the chest (Hunter et al., 1959; Kirkland et al., 1959). In two cases of transdiaphragmatic migration, there is a suggestion that local inflammation may have facilitated erosion of the shunt tip through the diaphragm; a shunt infection was present in one case (Lourie et al., 1985) and probable cholecystitis in another.

A ventriculoperitoneal shunt that migrates into the chest by either a supradiaphragmatic or a transdiaphragmatic route generally causes pleural effusion (Kessler et al., 1962; Anegawa et al., 1986; Cooper et al., 1978; Dickman et al., 1989; Gaudio et al., 1988; Lourie et al., 1985; Obrador et al., 1977), presumably because the flow of CSF into the chest exceeds the resorptive capacity of the pleura. Respiratory compromise is the usual presenting symptom. Tension hydrothorax with shock occurred in one case (Dickman et al., 1989). Perforation of a bronchus by the tip of a shunt, previously observed with ventriculopleural (Kessler et al., 1962) and ventriculoatrial (Hunter, 1959; Isamat, 1969) shunts, has now been seen in two cases of ventriculoperitoneal shunt (Kessler et al., 1962; Taub at al., 1994). One of these patients developed pneumonia as a result (Rao et al., 1977). Given the longstanding experience with ventriculopleural shunts (Hoffman et al., 1983), it seems plausible that intrathoracic migration of a ventriculoperitoneal shunt might occasionally be asymptomatic. Such cases have not been reported to date. Respiratory distress after V-P shunt replacement should be considered an unusual but important sentinel symptom in the differential diagnosis of postoperative shunt complications. V-P shunt revision should be carefully planned. As in this case, there are many reported cases in which no any improvement can be obtained by shunting, although the appearance of NPH is typical on CT scan. We also agree with the opinions that V-P shunt revision should be carefully planned in the chronic stage and/or persistent stupor, even after confirming malfunction of the shunt system.

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