A case of migration of abdominal catheter of ventriculoperitoneal shunt into scrotum is presented. To explain this complication, different mechanisms have been suggested. Several authors pointed out that the dissection of the catheter was due to tight ligature at the connector site or to bowel contractions and repeated tractions of the peritoneal catheter.

Key words: Hydrocephalus, scrotum, migration, ventriculoperitoneal catheter

CASE REPORT

A five-month old male infant, operated on because of a nasal encephalocele when he was two days old, developed hydrocephalus a week after the operation. A ventriculoperitoneal shunt was inserted. He was discharged without problems ten days after the ventriculoperitoneal shunt insertion, but was readmitted at 5 months because of an enlarged right scrotum. On physical examination an enlarged right scrotum and the distal part of peritoneal catheter in this region was determined. Radiographic examination showed the shunt tip within the enlarged right scrotum. At surgery, the previous incision scar was reincised. The distal

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catheter tip was found, shortened and repositioned into the abdominal cavity. After the surgery the scrotal swelling disappeared and the patient was discharged without any problem.

**DISCUSSION**

Hydrocele associated with slippage of the shunt tip into the scrotum have been mentioned in the literature. In addition to the frequent abdominal complications such as peritonitis, infections, visceral perforations, cyst formation, etc, there are also complications such as upward or downward migration of the catheter (6).

Grosfeld and Cooney (8) found that the average interval from placement of the ventriculoperitoneal shunt to the development of hernia or hydrocele was 6.8 months, while this interval was about 5 months in our patient. In their series of four cases, however, the range that Oktem et al. (14) found was 24 hours-6 months.

To explain this complication, different mechanisms have been suggested. Fuwa et al. (6) pointed out that the dissection of the catheter was due to either too tight ligature at the connector or the bowel contractions and repeated tractions of the peritoneal catheter.

Murtag and Lehman (13) reported that peristaltic intestinal and omental activity pushed any foreign body present in the peritoneal cavity towards the umbilicus or inguinal canal. Since the catheter is a forcing body, both omental and intestinal activity contributes to the migration of the catheter.

Grosfeld and Cooney (8) found that children with ventriculostriatal shunts had a much lower incidence of inguinal hernia than those with ventriculoperitoneal shunts. These results suggest that the increased frequency of inguinal hernias and hydroceles in children with ventriculoperitoneal shunts may seem to be related to increased intraabdominal pressure and/or fluid caused by the presence of excess fluid in the peritoneal cavity, at a time when there is patency of the processus vaginalis. The higher incidence of inguinal hernia in conditions of increased intraabdominal pressure due to the various obstructive lesions support this mechanism (17).

Increased intraabdominal pressure may cause hydrocele, without or with inguinal hernias, in two ways. The presence of raised intraabdominal pressure and/or fluid may either prevent natural closure of the processus vaginalis or convert a patent processus vaginalis to a clinical processus vaginalis. The latter seems to be much less likely to happen. Rowe et al. (17) also found that patients with evident bilateral inguinal hernias had higher incidence of raised intraabdominal pressure than those with unilateral evident inguinal hernias and a patent processus vaginalis on the contralateral side, but the incidence of the raised intraabdominal pressure in children with unilateral inguinal hernias was the same as those with or without the patent processus vaginalis on the contralateral side. These results suggest that raised intraabdominal pressure may cause the increased development of bilateral inguinal hernias, but not continued patency of the processus vaginalis.

As for the period of time of natural closure of the processus vaginalis, there is no common agreement among authors. There are two types of studies that can provide information about the natural closure history of the processus vaginalis. Although the first group of the studies are based on autopsies, the other group of the studies are based on the patency of the contralateral processus vaginalis in inguinal hernial repair. In one study based on autopsies, the processus vaginalis was found to be partly or completely open in 80% of cases at birth (11). The results found in another study also suggest that there may be some degree of patency of at least one processus vaginalis in over 50% of cases thro-
throughout infancy (15). Several studies based on the incidence of patency of contralateral processus vaginalis at various age, during bilateral inguinal exploration in cases of clinically unilateral hernias, suggest that the patency rate of processus vaginalis is about 70% to 80% at birth, gradually decreasing to about 30% to 40% by 3 to 4 years of age (7,17). In one study that was undertaken in 430 children between 1985 and 1995, Clarrett et al (3) proposed that the development of an inguinal hernia or hydrocele after the insertion of a ventriculoperitoneal shunt could be used as an indirect marker of patency of the processus vaginalis at the time of insertion of the ventriculoperitoneal shunt. In addition, they claim that the processus vaginalis remains patent in at least 30% of children in the first few months of life, after which time the patency rate appears to fall off quite sharply. This study also demonstrates that the incidence of subsequent development of hydrocele, with or without inguinal hernia, is closely related to the age of insertion of the ventriculoperitoneal shunt.

In conclusion, we believe that the factors mentioned above contribute concomitantly to the development of hydrocele. In a hydrocephalic infant, whose processus vaginalis are open, peristaltic intestinal and omental movements may drive the catheter into the inguinal canal and subsequently in to the scrotum. If, in infants with a relatively small abdominal cavity, the intraabdominal pressure is too much raised due to large volume of cerebrospinal fluid which exceeds the ability of the peritoneal lining to absorb, this theory may be especially true.

If an infant with a ventriculoperitoneal shunt develops hydrocele, this complication should be immediately recognised and repaired surgically. Using a shunt including a high pressure valve and a shorter catheter may perhaps decrease the incidence of this complication.

REFERENCES

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