Movement of hemostatic clips from the ventricles through the aqueduct to the lumbar spinal canal

Case report

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The authors report a patient in whom rebleeding after operation for a left temporal arteriovenous malformation resulted in the dislocation of multiple hemostatic clips. Several clips, including a Yaşargil aneurysm clip, were detected incidentally in the lumbar spinal canal. No clinical signs or symptoms were noted. Retrospectively, passage of the aneurysm clip through the aqueduct could be detected on computerized tomography scans performed to evaluate a series of epileptic seizures.

KEY WORDS • arteriovenous malformation • clip • foreign body migration

FOREIGN bodies have been reported to migrate within the brain and within the ventricle system as well as from a cranial location to the spinal canal. The earliest report by Vilvandré and Morgan was published in 1916. Iatrogenic implantation of foreign bodies bears some risk of dislocation. Microsurgical treatment of intracranial aneurysms by clip application has been complicated by clip failure. Oyesiku and Jones reported the migration of an infratentorially applied Heftetz aneurysm clip to the cauda equina, causing radicular symptoms. We report a case in which multiple hemostatic clips, including a Yaşargil clip, migrated from an initial supratentorial location to the lumbosacral spinal canal.

Case Report

This 55-year-old man experienced acute occipital headache and neck pain following unusually heavy physical work on May 8, 1985. Two days later, vertigo and headache increased and he suffered two attacks of transient aphasia within an interval of 3 days. He could remember a similar event 4 years before. He had binaural hypoacusis and impaired vision in the left eye related to previous trauma.

One week after the onset of symptoms, an atypical left temporal intracerebral hemorrhage was diagnosed with the aid of computerized tomography (CT) and the patient was hospitalized. At that time, he was alert and showed only discrete dysphasia and slight anisocoria with dilation of the right pupil, which resolved the next day. Cerebral angiography revealed a large arteriovenous angionoma in the left temporal region with two major feeding vessels from the middle cerebral artery and high shunt volume (Fig. 1). The anterior

Fig. 1. Postoperative left carotid angiogram, lateral view, showing an angionoma filling through the middle cerebral artery and early venous outflow.
cerebral artery was not filled, presumably as a steal phenomenon.

Operation. After hematomat resorption and stabilization of his clinical condition, the patient underwent surgery on June 18, 1985. In a 20-hour procedure the angiomata was totally removed. Hemoclips were applied to the two major feeding arteries and some smaller vessels (Fig. 2). The angiomata was attached to the ventricular plexus in the region of the trigone, and a Yaşargil aneurysm clip was applied to the plexus vessels contributing blood to the angiomata. The bone flap was not replaced because of slight brain swelling.

Postoperative Course. Two days after the operation, rebleeding occurred with tamponade of the left ventricle and obstructive hydrocephalus; this condition was treated by external ventricular drainage for 7 days. The patient required respirator ventilation until June 26 and thereafter gradually improved; he was moved to a rehabilitation center 5 weeks later. At this time, he was again alert. Neurological examination disclosed a mild organic psychiatric syndrome and residual signs of minor sensory dysphasia.

After 6 weeks of rehabilitative training the patient was discharged home and returned to our clinic for cranioplasty on October 18, 1985. Skull x-ray films obtained before cranioplasty demonstrated migration of the aneurysm clip which was now lying free in the lateral ventricle opposite to the operated side. Angiography confirmed complete extirpation of the angiomata. The patient made an uneventful recovery after this operation.

On January 14, 1986, the patient noted transient hemihypesthesia and hemiparesis of the right side, after which he suffered a grand mal seizure. A CT scan showed no signs of bleeding. Laboratory parameters, including the serum phenytoin level, were all within normal range. On February 12 and 20, similar events occurred. On March 7, the patient returned to the neurological department with complaints of prolonged but transient dysphasia and right-sided hemiparesis and hypesthesia; these phenomena had occurred repeatedly during the previous few weeks and had lasted for up to 1 hour. A repeat CT scan showed no signs of recurrent bleeding or cerebrospinal fluid (CSF) circulation disorder. The phenytoin serum level was in the lower therapeutic range, so the dose was increased to 450 mg/day, and the patient was discharged.

Later the seizures decreased in frequency (in 1987 the patient suffered only two minor events). In November, 1987, he returned to the hospital with abdominal pain due to constipation. To rule out a colonic neoplasm, an air-contrast barium enema was performed with normal results except for the presence of nine hemoclips in the lumbar and sacral spinal canal (Fig. 3). The patient reported that he felt perfectly well and had never experienced lumbago or sciatica. A very discrete dysphasia was noted, otherwise his neurological
Migration of hemostatic clips

Fig. 4. Retrospective examination of the postoperative computerized tomography scans documents migration of the Yaşargil clip. Left: Scan obtained on October 31, 1985. The clip is visible in the ventricle trigone of the side opposite to the surgery (arrow). Center: Scan obtained on January 15, 1986. The clip (arrow) now lies in the entrance of the aqueduct. A lack of clinical information to the radiologist led to misinterpretation of this area as pineal calcification. Right: Scan obtained on March 11, 1986, showing an area extending from the foramen magnum to the upper vertical region. The clip is no longer visible.

state was normal. It should be emphasized that no signs of radicular irritation could be detected. Retrospectively, the passage of the Yaşargil clip through the aqueduct could be identified on CT scans. It had obviously initially been misinterpreted as plexus and pineal body calcification (Fig. 4).

Discussion

Implantation of foreign bodies always carries a small risk of dislocation. Earlier reports have described migrating bullets after war injuries or civilian gunshot wounds. These movements have mostly occurred in either supratentorial or infratentorial regions or from one level of the spinal canal to another (usually lower) level. Gravitational forces are the possible cause of intracerebral movements (with the tendency of metallic bodies to sink), and vessels or other tissue inhomogeneities may be responsible for rotational movements. Intraventricular bodies have the potential for free movement unless they are attached to the plexus. Physiological stenosis is provided by the foramen of Monro, the aqueduct, and the foramina of Luschka and Magendie. Lang reported a patient in whom a bullet caused hydrocephalus by occluding the entrance of the aqueduct. In a case reported by Liebeskind, et al., a bullet must have passed the tentorial level migrating within the brain-stem parenchyma. In a patient described by Kellhammer, a grain of shot migrated from the region of the foramen of Monro to the sacral spinal canal within 12 days.

In our case, rebleeding occurred 2 days after the operation and antedated gross clip dislocation. Since complete extirpation of the angioma could be documented (Fig. 2), a hyperemic complication (indicated by the tendency to intraoperative brain swelling) or failure of one of the multiple clips is a possible cause for this rebleeding. Hematoma formation and shearing forces during rebleeding along with softening of adjacent brain tissue possibly helped to produce the dislocation of nine clips to the lumbar and sacral spinal canal, including the Yaşargil aneurysm clip. Migration of an aneurysm clip through the aqueduct was not initially recognized, but the close temporal relationship of the epileptic seizures and passage of the clips through the aqueduct suggests a causal relationship, possibly due to intermittent partial obstruction of CSF passage.

To our knowledge this is the first case describing the passage of an aneurysm clip through the foramen of Monro, the aqueduct, and the fourth ventricle outlets to the very end of the spinal canal.

Radicular symptoms are possible sequelae of foreign bodies in the lumbar spinal canal and may necessitate surgery, however, up to now the actual risk of such bodies causing symptoms is not well defined. As long as primary sterile foreign bodies remain asymptomatic, we see no indication for prophylactic surgery, especially in the present case, where multilevel distribution renders complete surgical sanitation impossible.

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References


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