

## **Thoracic spine arachnoiditis and arachnoid cysts following aneurysmal subarachnoid haemorrhage: A Case Report**

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**Abstract:** Neurological syndromes following arachnoid cyst is a rare complication of aneurysmal subarachnoid haemorrhage which is less than 1%. The best treatment option is complete removal of these bubbles. Posterior cysts are accessible to complete surgical resection without spinal cord damage. Anterior cysts are often managed with a cystoperitoneal shunt. However, Patients with associated arachnoiditis usually experience an incomplete recovery. We report a 48-year-old male who complained of gait disturbances and dysuria 16 months after subarachnoid haemorrhage. Clinical history, plain radiographs, MRI study of the patient was collected. A review of the literature was also described. The patient was managed by fenestration of the arachnoid cysts, symptoms gradually resolved in his gait disturbance and muscle strength. However, dysuria was not improved. In our daily practice, Arachnoid cysts due to adhesive arachnoiditis should be recognized as a late complication of subarachnoid haemorrhage.

**Keywords:** Thoracic spine; arachnoiditis; arachnoid cyst; subarachnoid haemorrhage.

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### **INTRODUCTION**

Although symptomatic intradural extramedullary arachnoid cysts (AC) caused by arachnoiditis are rarely seen in clinical practice, enlargement of these bubbles can produce significant neurological deficits which dependent on the extent and location of compression site. More recently, subarachnoid haemorrhage, contrast media, trauma, epidural steroids, and local anaesthesia [1-4] have all been implicated in the cause of AC, however all of them are formed as a result of the inflammatory process of arachnoiditis, in which arachnoid transform from mild thickenings to catastrophic adhesions of subarachnoid space. The time interval between inflammatory onset and clinical symptom of the AC extends from a few hours to 40 years, which is extremely useful in determination of pathogeny.

In this article, we aimed to present a case of spinal AC formation following arachnoiditis caused by aneurysmal subarachnoid haemorrhage. At the same time, similar literature were examined.

### **CASE REPORT**

#### **History**

48-year-old male had a medical history of subarachnoid haemorrhage (SAH) 28 months ago, he had been admitted to local hospital for sudden onset of headache and unconsciousness. Cranial unenhanced computed tomography scans disclosed a diffuse SAH and associated hydrocephalus which was clinically a

Hunt and Hess grade 4. After external ventricular drainage had been established 5 hours later, cerebral angiography was performed the next day, which revealed a 3.3-33.5-33-mm fusiform aneurysm of the distal end of right vertebral artery. The coil embolization was successful and he regained his consciousness; however, hyperpyrexia was observed the next day, his body temperature reaches 42°C and administered with antibiotics. 5 days later, the patient complained of muscle weakness of both lower limbs and pyretotyposis. The manual muscle test in the lower extremities demonstrated 3/5 antigravity strength. The pyretotyposis subsided 1 month later and the patient could walk unaided after 3 month of rehabilitation. He was living independently and was fully ambulatory.

10 months after SAH, involuntary spasm was discovered at his right toe, the symptom went upward gradually and accompanied by muscle weakness. 6 months later, the patient had gait disturbances and dysuria. He received a MRI examination and revealed that intradural cystic lesions located at T7 and T11, which producing spinal cord compression. Arachnoiditis was observed extending from T7 to T11 (Fig. 1). Surgery was suggested, however, the patient preferred conservative treatment. 28 months after SAH, The patient was admitted in our hospital at the age of 48, with flasco-spasmodic paraparesis and urinary retention for 12 months.

### Examinations

Neurologic examination revealed hypesthesia below navel. Hyperactivity of deep tendons was noted. Babinski sign was present bilaterally. Muscle tonus at the lower extremities was highly increased. He was extremely hyper-reflexic in the lower extremities with no voluntary muscle contraction. Proprioception and sensation sensory was not appreciated below groin. He had no upper extremity involvement. MRI revealed an intradural extramedullary cysts compressing the spinal cord at T7 and T11 respectively, and defused arachnoiditis lesion between T7-11. The cysts had the same signal intensity as cerebrospinal fluid on T1- and T2- weighted images, and no contrast enhancement was found which was similar with 12 months ago.

### Surgery

The T7 and T11 level cysts were considered to be responsible for to motor dysfunction. These levels were marked using fluoroscopy; a linear incision was made over these levels separately. Standard laminectomies of T7-8 and T11-12 were performed with a lateral extension to the pedicles, thus enabling the surgeons to reach the ventral component of the lesion. At T7-8 level, with the aid of the operating microscope, the dura was opened. Immediately obvious was the extent of arachnoid scarring in this area—a sign consistent with arachnoiditis. The cyst extended ventrally from the dorsal aspect of the cord, causing an obvious mass effect on the spinal cord. The dentate ligaments were divided and the spinal cord was mobilized. Dense

fibrous adhesions prevented total excision, but fenestration of the arachnoid cyst in several areas was possible. Shunt placement was not performed because of the consideration of surgical site infection and poor outcome according to pervious reports [5]. The dura was reapproximated in a watertight fashion with Tisseal and Dura repair, and the wound was closed in the usual multilayered fashion. Surgical procedure was the same at T11-12 level, except internal fixation which was employed to prevent local kyphosis.

### Postoperative course

3 days after surgery his muscle tonus was deceased and muscle strength was improved. The manual muscle test demonstrated 2/5 strength on the left and 1/5 on the right. After 2 month of rehabilitation, the patient can stand up mainly depended on left lower extremity. After an uncomplicated hospital course, he was discharged home 3 months later. Postoperative imaging demonstrated decompression of the AC (Fig. 1). Seven months postoperatively, the patient demonstrates significant improvement in his gait disturbance and muscle strength; however, dysuria was not improved.

The design and performance of this study conformed to ethical standards of Helsinki Declaration and our national legislation. It was approved by Medical Ethical Committee of our institution. Before enrolment, patient was enquired whether or not willing to take part in a scientific research and informed consent forms were signed by himself.



**Fig-1: Pre and post-operative MRI scans. a> There are 2 arachnoid cysts located at T7 and T11 levels respectively; b> Post-operation, the cysts was successfully managed by fenestration.**

## DISCUSSION

Acquired AC arises in the environment of arachnoiditis, which produces small adhesive spaces and imbalance of subarachnoid pressure around the adhesive point. The pressure ensures CSF running into these small pockets. Incidentally, the unilateral valve mechanism, with gradual inflow and no egress of CSF from these spaces guarantees the increase of the size, which followed by their mass effect and neurologic symptoms. The inflammatory reaction of arachnoid could be commonly seen in trauma, infection, surgery, SAH and even myelography [6], however, spinal AC with spinal cord and nerve root compression could be found in no more than 1% of cases [7,8].

Spinal arachnoiditis can be characterized by acute, sub-acute, or chronic disease progression. Acute spinal arachnoiditis is mostly found in patients with severe systemic infections, such as sepsis and bacterial meningitis. It is often accompanied with hyperthermia, fever, acute phase symptoms. In the most severe cases it is complicated by hemorheological disorders, multiorgan failure, etc. Neurologic symptoms develop quickly over only a few days or even hours; in particular, acute progression of severe conductive symptoms emerges. Sub-acute spinal arachnoiditis also accompanies infections, but in a more latent form, developing over months; it is characterized by normal or subfebrile temperature. In the neurologic status, radicular symptoms come to forefront, while conductive symptoms emerge later. In patients with chronic spinal arachnoiditis, it is often impossible to determine an exact infectious agent; disease progresses very slowly; spastic and afferent paresis, as well as pelvic disorders, is prevalent.

In our case, hyperpyrexia was observed 2 days after ventricular drainage, which was followed by muscle weakness of both lower limbs 3 days later. The patient was diagnosed as acute spinal arachnoiditis which was believed to be a complication of bacterial meningitis. The symptoms were developed by compression of spinal cord, which was induced by accumulation of inflammatory substance intra-dural. Muscle weakness subsided 1 month later along with pyretosis subsidence suggested that the inflammatory substances had been eliminated by antibiotic therapy; however, the second state of spinal cord compression was produced by AC as a complication of SAH 10 months later. In fact, the incidence of this complication was lower than 1% [9].

The irruption of blood in the subarachnoid space could have produced a leptomeningeal inflammatory reaction. It is thought that these cysts were formed from discrete pockets of CSF that had been loculated within the arachnoid membranes. Locations may include intradural, extradural, perineural, intracranial, or intraspinal areas [10]. Cyst enlargement results from the continuous production of CSF within these loculated

arachnoid membranes [11]. This may result in the displacement of surrounding structures, thereby producing symptoms.

To date, in fact, the literature remains sparse regarding this disease entity in patients in whom SAH has occurred, with only 25 case presentations in the literature [12]; however, once the spinal cord is compressed, the consequence is devastating. Despite the continually growing number of patients successfully treated every year for aneurysmal SAH, both surgically and endovascularly, there has not been a parallel rise in the number of SAH induced spinal AC or arachnoiditis.

Patients with massive and posterior fossa SAH seemed to be at risk of developing this complication, probably because of contamination of the spinal subarachnoid space. The preferential site was the thoracic canal from T3 to T8. Prolonged decubitus in patients with severe SAH might have favored stagnation of blood in the thoracic kyphosis and development of the cyst in this location.

The surgical management of spinal arachnoid cysts depended on the location of the lesion [13,14]. Posterior cysts were usually accessible to complete surgical resection without spinal cord damage. Anterior cysts were often managed with a cystoperitoneal shunt. Only patients without associated arachnoiditis have experienced a good (but often incomplete) recovery after surgery. However, A.A. KASHCHEEV reported that Flexible Endoscopy in Surgical Treatment of Spinal Adhesive Arachnoiditis and AC could achieve a satisfied clinical outcome. Neurological improvement was seen in 87% of patients operated on [15].

## CONCLUSIONS

Arachnoid cysts caused by adhesive arachnoiditis are a rare and late complication of subarachnoid haemorrhage. Although patients with arachnoiditis usually have experienced an incomplete recovery, surgical treatment is still the first choice. The treatment should focus on the lesion which considered to be the source of neurological syndromes on the background of massive arachnoiditis. More recently, Flexible endoscopy has been proved to be an efficient and safe method, which could be widely used for treatment of spinal adhesive arachnoiditis, and arachnoid cysts.

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