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Spinal Aracchnoidal Cysts: A Review of Advances in Diagnosis and Treatment

Najmiddin Norkulov

Assistant in Neurosurgery department Samarkand State medical university, Samarkand Uzbekistan

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Spinal arachnoid cysts (SACs) are a heterogeneous group of formations, including SACs with different mechanisms of formation and location [1, 2, 4]. Currently, there are a large number of classifications of SAH depending on morphology, etiology and location.

Acquired SAH can be the consequences of injuries, non-traumatic hemorrhages and purulent-inflammatory lesions. There is no generally accepted treatment for acquired SAH. The vast majority of acquired SAH are localized intradurally , but sometimes they can be fixed in the extradural space. Macroscopically, the walls of the SAH are a compacted, often cloudy arachnoid membrane. Microscopically, the SAH wall is a fibrous tissue with a lining of arachnoendotheliocytes , which in some cases may be absent [3, 5].

Mostly congenital, moreover, multiple SAH are often combined with genetic defects (type I neurofibromatosis , Klippel - Trennon syndrome , against the background of polycystic kidney damage). The reasons for the formation and growth of SAH type 1A are currently not studied. There is an opinion about their formation due to a congenital defect of the dura mater (DM) with further protrusion of the arachnoid through this defect [5-7]. Often the SAH cavity is connected to the intradural arachnoid space through fistula [8-11].

There are 3 theories that interpret the etiology of the gradual progressive increase in SAH [12, 13]:

1. The presence of a valve mechanism for filling the SAH through the fistula with the subarachnoid space. Since the SAH is connected by a leg to the subarachnoid space, with CSF pulsation, the hydrostatic pressure in the SAH increases, and with a decrease in pressure in the subarachnoid space, a higher pressure in the SAH contributes to the closure of the anastomosis valve and blocks the reverse flow of the CSF;

2. There is a theory that the walls of the extradural CAH produce fluid, that is, they have a cellular lining (true cyst). But this theory is not substantiated. The results of histopathological studies do not identify cells capable of producing fluid in the walls of the removed SAH;

3. Filling of the SAH may occur due to the osmotic gradient between the fluid contained in the SAH and the CSF in the subarachnoid space.

Extradural SAH are rare [14]. They are fixed more often in men, at the age of 30-40, often combined with kyphoscoliosis , syringomyelia, spinal dysraphia . These SAH are usually located on the dorsal or dorsolateral surface of the dural sac (DM) and in 65% of cases they are detected in the middle and lower thoracic spine [15, 16]. In 12% of cases, SAH is found in the thoracolumbar junction , in 13% in the lumbar and lumbosacral, in 7% in the sacral. The rarest cervical SAH are found with a frequency of about 3% [17].

The clinical picture includes pain syndrome, manifestations of compression myelopathy and radiculopathy [18, 19]. Pain does not seem to be a mandatory manifestation of the disease. Pain can be fixed in the back at the level of SAH localization, which is presumably associated with irritation of the spinal cord (SM) membranes, and also be radicular in nature when the SAH processes spread into the foraminal openings with compression of these roots. Myelopathic disorders develop gradually over several years. The course of the disease is slowly progressing in 60-70% of cases, and in 30-40% of individuals there is a remitting course, which may be associated with fluctuations in the level of fluid pressure in the SAH [20-24]. Magnetic resonance (MR) research is the basis of modern SAH diagnostics . The MR signal from the SAH content corresponds in intensity to the signal from the CSF [25]. The walls of the SAH do not accumulate a contrast agent. SAH type Ia is visualized as an extradural volumetric formation, in most cases localized dorsally . Type Ia CAA has an elongated cigar shape. MR-examination in the coronary projection, especially in the myelographic mode, can visualize the lateral processes of the SAH, protruding into the lateral recesses and foraminal openings [26].

Primary intradural cysts form from arachnoid diverticula [27]. Possible explanations include congenital weakness of the arachnoid, causing protrusion of the latter in response to CSF pressure drops or, possibly, its hypertrophy and proliferation of arachnoid granulations [28]. Most cysts communicate with the subarachnoid space through a narrow fistula. Congenital pathologies are mainly always localized dorsal to the SM.

In most cases, they are secondary (trauma, infection, hemorrhage, after neurosurgical intervention). The formation of the latter is based on the fragmentation of the subarachnoid space due to the adhesive process (the formation of arachnoiditis). They occur with the same frequency in men and

women, they can manifest at any age, but more often at 30-40 years [29]. The clinical picture is very variable and falls within the limits of SC compression (myelopathy , myeloradiculopathy or radiculopathy) [30, 31].

Methods of surgical treatment:

- 2 or 3-level hemilaminectomy or laminotomy with further resection of the cyst wall;

— endoscopic examination and fenestration of the cyst wall.

Conclusion

Currently, none of the proposed hypotheses explains the mechanism of formation of SM arachnoid cysts. The clinical picture of SM cysts is due to compression of neural structures, liquorodynamic disorders, and destruction of the spinal column. The method of choice for neurosurgical treatment is microsurgical excision of the cyst walls.

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