Arachnoiditis affecting the brain is usually related to infections (meningitis), trauma, tumour, intracranial haemorrhage and chemical insult (myelogram dyes).

It was reported as early as 1924 by Horrax who reported symptoms suggestive of a tumour, but which turned out to be arachnoiditis in the cisterns.

The commonest cause of cerebral arachnoiditis (CA) is infection, whether local (within the CNS) or in other parts of the body, particularly the sinuses and the middle ear.

Tuberculous CA has been shown to arise in the absence of pulmonary infection. Cystercicosis is a parasite, which can cause this type of arachnoiditis.

A study in the late 1980s looked at 92 patients with hydrocephalus secondary to cysticercotic meningitis.

The mortality rate was 50%, with most patients dying within the first 2 years after cerebrospinal fluid (CSF) shunting, with spontaneous remission of the cysticercotic arachnoiditis, as shown by the CSF findings, occurring in only 18%.

The authors noted,

"In most patients, arachnoiditis and positive immune reactions persisted unchanged even after several years."
White (iii) noted:

"Subarachnoid cysticercosis is associated with arachnoiditis. The arachnoiditis may result in meningitis, vasculitis with stroke, or hydrocephalus."

In 1978, a paper (iv) on parasite infections in Thailand noted,

"Cysticercus cellulosae, caused by Taenia solium*, commonly results in epilepsy, and sometimes increased intracranial pressure from intraventricular obstruction or from basal arachnoiditis."

The author went on to remark that spinal cord and cauda equina involvement tend to occur much less frequently and to recommend Cysticercus complement fixation tests on the CSF and computerised axial tomography (CAT scan) as helpful in establishing diagnosis.

* Taenia solium is the tapeworm

Mexican doctors (vi) reported a case of the 32 year old man who presented with a subarachnoid haemorrhage and was found to have a cerebral aneurysm that was surrounded by "an area of severe arachnoiditis around a cysticercus" (cysticercus being a focus of infection with cysticercosis).

Subarachnoid infection of this type may thus present in unexpected fashion.

This echoed similar findings by their colleagues (vi), who discussed findings relating to 65
patients with stroke associated with neurocysticercosis.

They described a "high frequency of subarachnoidal cysts" adjacent to the ischaemic area. 80% of patients with focal cysticercosis presented with a vascular event such as stroke, compared with 20% of those with diffuse cysticercosis.

In diffuse cysticercosis, 80% had hydrocephalus, 64% multiple cerebral infarcts and 43% mental disorders.

The authors concluded:

"Based on the distribution of cysticercal disease and the severity of concomitant chronic arachnoiditis, it is possible to identify a wide spectrum of cerebrovascular involvement caused by neurocysticercosis."

Russian investigators ([vii]) have found that immune indices are affected in cases of labile or progressive cerebral arachnoiditis: aggravation of clinical symptoms is accompanied by a decrease of a number of T-lymphocytes, an increase of the levels of immunoglobulins A and G as well as of P-proteins and of a general activity of serum interferons.

Immune compromised individuals may develop cerebral arachnoiditis in association with unusual infectious diseases such as aspergillosis, as recently reported by a European group ([viii]): a previously healthy man was found, after some fourteen months of chronic meningitis, ventriculitis, choroid plexitis, and lumbar arachnoiditis, complicated by acute hydrocephalus, to have an Aspergillus organism in the cerebrospinal fluid.

Eosinophilic aseptic arachnoiditis has been seen in HIV patients ([ix]).
Cerebral arachnoiditis has also been noted in cases of sarcoidosis.

Bahr et al. ([i]) noted:

“Communicating hydrocephalus with sarcoid arachnoiditis is the most common finding” in sarcoidosis of the central nervous system.


