The following stages were described by Burton in 1978:

First stage: Radiculitis: the spinal nerve roots are inflamed and the adjacent blood vessels distended (hyperaemia). The subarachnoid space is encroached upon by the swollen nerve roots and practically disappears. Deposition of collagen fibrils (scar tissue) begins.

Second stage: Arachnoiditis: the scar tissue increases, and the nerves become adherent to each other and the dura.

Third stage: Adhesive arachnoiditis: involves complete encapsulation of the nerve roots. The subsequent compression causes them to atrophy.

The scarring prevents contact with the spinal fluid in that area. Severe adhesive arachnoiditis may be obliterative, causing completely impeded CSF flow within the affected area (and hence loss of dural pulse).

There may be cysts containing CSF or oil-based myelogram dye. There may also be calcification or ossification.

Aldrete suggests ([i]) that radiculitis and acute arachnoiditis constitute the inflammatory phase which can be prolonged and progressive if the individual's immune system over-reacts or if there are repeated insults.

In other individuals, this phase may gradually subside, especially if anti-inflammatory measures are taken.

After several months, (4-8) the onset of the proliferative phase may occur, in which the adhesions begin to form.

Long ([ii]), a Johns Hopkins neurosurgeon, described the following features of arachnoiditis:

Partial or complete block or narrowing of the subarachnoid space, thickening of the nerve roots, obliteration of nerve root sleeves, irregular distribution of contrast medium with loculation of retained iophendylate, formation of cysts and spinal cord atrophy.

Essentially, the nerve roots resemble over-cooked strands of spaghetti, which are entwined and distorted. In the later stages, the nerve roots adhere to each other and pull out to the sides of the spinal canal to adhere to the dura.

In 1983, Hoffman, ([iii]) working with dogs, found pre-mortem multilevel blockage of the subarachnoid space, leptomeningeal inflammation, fibrosis, adhesions, cysts and nerve roots embedded in thick bundles of collagen.

Hoffman remarked:

"it would seem that the literature on this subject deals with only the most severe clinical examples and that arachnoiditis producing symptoms in the absence of sensory-motor abnormalities is unrecognised."

He proposed the following grades of arachnoiditis:

Grade 1: inflammatory infiltration of the arachnoid and pia mater, no adhesions.

Grade 2: inflammatory infiltrations, mild adhesions without stenosis of the subarachnoid space, partial minor calcification

Grade 3: inflammation of the leptomeninx with severe adhesions, massive calcification

Grade 4: inflammation of the leptomeninx and nervous tissue, complete obstruction of the subarachnoid space by scar tissue.

Bourne ([iv]) suggested that arachnoiditis involves inflammation with proliferation of fibrous tissue that strangles and destroys nerve cells and fibres and engorgement of veins with blood when standing or walking causing increased pressure on the enclosed nerves may explain worsening pain during those activities.

Burton ([v]) noted that adhesions may restrict nerve root mobility, leading to increased incidence of lateral herniated disc symptoms with increased tension, decreased blood flow, or additional trauma.

Iophendylate effects:

Dujovny et al ([vi]) in their dog study used a scanning electron microscope (SEM) to detect the effects of contrast media on the arachnoid membrane.

They found that the normal fenestrations of the membrane became closed by a fibrin-like structure after the dye had been used, and there were macrophages present, indicating inflammation.

Iophendylate produced the greatest number of macrophages within the fenestrations.

In some cases, the scar tissue calcifies and may form bony plaques: Arachnoiditis Ossificans (AO), a term introduced by Puusepp in 1931([vii]). In most cases, the thoracic cord is affected.

Shiraishi, Crock and Reynolds ([viii]) have written about spinal arachnoiditis ossificans.

They state that it is a "rare pathological entity" which is precipitated by peridural bleeding and meningeal irritation secondary to surgical intervention.

Whilst small plaques of calcified material may be found on the spinal meninges at autopsy in around 75% of specimens, most of these are regarded as benign and are likely to have been asymptomatic.

The 1971 proposition by Kaufman and Dunsmore ([ix]) reserves the term arachnoiditis ossificans for the clinically significant entity that can cause progressive neurological deficit due to compression of nerve roots and spinal cord.

Slavin et al. ([x]) presented a case of thoracic myelopathy caused by extensive ossification of the arachnoid membrane with an associated intramedullary syrinx.

The patient presented with a 9 year history of progressive spastic paraparesis of the lower limbs and bilateral sensory loss to a level of T8.

There were no obvious precipitating causes. At operation, on opening the dura, the spinal cord was covered "with a light gray hard shell": calcified arachnoid membrane, "circumferentially encasing the spinal cord like an armor" but not follow exiting nerve roots.

There was a smooth outer surface facing the dura and a rough irregular surface facing the spinal cord.

One piece was thicker and when removed, left a deep groove on the surface of the spinal cord.

A vascular structure resembling a venous malformation was also found.

It is unclear whether this malformation was cause (perhaps due to a small haemorrhage resulting in inflammation) or effect (post-compression varicose dilation).

Similar lesions have been noted in other cases.

The authors divided cases of arachnoiditis Ossificans into 3 groups:

Type 1: consisting of minute calcifications and ossifications frequently encountered at surgical procedures in asymptomatic individuals. It may be quite extensive causing the arachnoid to appear chalky white. It is frequently associated with adhesive arachnoiditis.

Type 2: a small segment of ossified arachnoid occurs, either an isolated plaque or a thicker piece of met plastic bone; this may compress the underlying spinal cord or nerve roots. This occurs rarely and has causes such as trauma, haemorrhage and arachnoiditis.

Type 3: the rarest, involves circumferential ossification of the arachnoid around the spinal cord or cauda equina. This almost invariably causes progressive neurological deficit.

The authors proposed 3 different theories of aetiology:

1. Ossification occurs as a result of chronic inflammation: supported by the frequent association with adhesive arachnoiditis. Histological studies have shown formation of bone and signs of chronic inflammation, whilst CSF inflammatory changes have also been noted. The authors noted the history of infective or chemical insult in some cases, and their known link in causing arachnoiditis. In particular, we should note Carta et al. ([xi]) who reported on calcification after Depo-Medrol; Van Paesschen et al. ([xii]) reporting on Ossificans and arachnoid cyst after cranial tuberculous meningitis; Tanaka et al. ([xiii]) AO after repeated myelography and spinal surgery.

2. Formation of bone in the presence of arachnoidal granulations, which calcify and ossify over time. Cell clusters were seen in studies early in the Twentieth century. This theory is supported by the presence of arachnoiditis Ossificans in the absence of ongoing inflammation. This includes cases such as that reported by Nagpal et al. ([xiv]) of ossification with unrelated syrinx. In addition, it is worth noting that meningiomas and arachnoid around them may involve ossification/calcification. ([xv]) For this reason, the authors suggested that the term Ossificans is misleading and should be "arachnoid ossification" to avoid suggesting inflammation is present.

3. The third theory suggests that ossification is analogous to other cases of heterotopic ossification such as that seen in cases of spinal cord injury, ([xvi]) where ossification involves soft tissue around major joints. The precise cause remains unknown, although a number of associated factors have been suggested: micro trauma, chronic infection, genetic factors, and disturbance of calcium metabolism. Ossification has also been reported in patients with post-traumatic paraparesis or paraplegia without spinal cord injury.

The authors also postulated possible pathogeneses for the association between AO and syrinx formation.

These include, as suggested by other authors, focal disturbances of regional blood circulation,

which could cause ischaemia in the dura, leading to gliotic changes and eventually intramedullary cavitation.

Other possible causes include intramedullary haemorrhage, and other factors for tissue defects.

As Errea et al. suggested ([xvii]), impaired CSF circulation might be the cause, and Slavin et al. found absent CSF flow on cineMR imaging flow studies on their patients.

This concurred with the pathogenesis proposed by Milhorat et al. (91)
Kahler et al. ([xviii]) reported another case of syringomyelia and arachnoiditis Ossificans.

MRI revealed the syrinx but not the extensive arachnoiditis, which was noted in insertion of a syringopleural shunt.

Post-operative CT clearly showed the extent of the lesion.

Faure et al ([xix]) reported a case of AO of the cauda equina, which they suggested was unusual as AO tends to affect the thoracic and lumbar regions.

They suggested that although pathogenesis has been regarded as involving clusters of arachnoidal cells, that in fact the environment induced by arachnoiditis and the consequent disturbance of CSF flow is implicated.

The authors reviewed cases of AO in the literature and noted that

"mechanical dural sac lesions with probable dural breaching during repeated surgery or

following SCI were involved in all but two cases."

They proposed that opening the dura could be an important causative factor.

They also suggested vascular abnormalities, subarachnoid haemorrhage, spinal cord injury, meningitis, repeated mechanical insult and spinal anaesthesia as predisposing factors.

Ossified arachnoid membrane contains well-formed osteoid tissue, suggesting that the ossification process is active and progressive.

Faure et al. remarked:

"The association of ossified arachnoid with contiguous and often extensive dense fibrosis of the arachnoid suggest that ossification is the end point of arachnoiditis." (Citing Whittle et al. [xx])

Manabe et al. ([xxi]) described a patient with venous insufficiency and a fluctuating monoplegia due to compressive Arachnoiditis Ossificans at T11-12. Symptoms appeared monthly lasting a few weeks each time.

Brazilian authors, Mello et al. ([xxii]) described 3 cases of thoracic arachnoid ossification, associated with sensory and motor symptoms, sphincter dysfunction and inferior limb pain.

They noted that calcium deposits tend to occur mainly in the middle and lower thoracic spine

"where the majority of trabeculated arachnoid cells are located".

The ossification is progressive over time, causing ongoing deterioration in the patient.

They suggested that spinal cavitation can be due to spinal cord tethering, stretching, and central cord oedema formation, with cerebrospinal fluid blockage and pulse pressure changes.

They also noted intramedullary cavitation associated with the ossified lesions and suggested that this was due to a lowered CSF pressure distal to the thoracic blockage caused by the lesions, which allowed

"development of comparatively higher centrifugally directed intramedullary pressure gradients."

Two cases involved the use of iophendylate dye before the first operation (and one patient had a thoracic meningioma resected) and findings of extensive adhesions at re-operation some years later.

However, in the first case, there was no obvious cause for the ossified plaques found at the first operative intervention.

In the third case, it appears that meningitis and 4 lumbar punctures were the precipitating factors.

The authors therefore remarked that the cyst formation they observed was due to spinal cord deformity resulting from meningioma, arachnoiditis and ossification.

Mello and his colleagues noted that their three cases involved similar presenting features as those reported in the literature, i.e. in women, persistent thoracolumbar and leg pain, crural paraparesis and urinary incontinence.

In private correspondence, Dr. Mello recently told me of a fourth case he had come across, which involved a multiloculated syringomyelia associated with arachnoiditis.

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