A "cystic and adhesive" arachnoiditis was described by Benini and Blanco ([i]), in which the cysts are collections of spinal fluid walled off by the meningeal adhesions.

Arachnoid cysts are particularly seen after Pantopaque myelography, where the dye has become encysted rather than as in other cases, persisting as a diffuse thin film.

They may also be associated with epidural anaesthesia. Lee and Cho ([ii]) suggest that

"Intradural spinal arachnoid cysts appear to result from an alteration of the arachnoid trabeculae; some such cysts are ascribed anecdotally to previous trauma or arachnoiditis, whereas the majority are idiopathic and congenital."

Cysts may be spinal, or intracranial (most common locations are the middle fossa (near the temporal lobe), the suprasellar region (near the third ventricle) and the posterior fossa, which contains the cerebellum, pons, and medulla oblongata.), where they are associated with headache and seizures, with focal neurologic signs occurring less frequently.

Pathologically, arachnoid cyst walls are formed from a splitting of the arachnoid membrane, with an inner and outer leaflet surrounding the cyst cavity, the cyst wall consisting of fibrous connective tissue slightly denser than normal arachnoid tissue.

Sato et al ([iii]) suggest that spinal intradural cysts are "uncommon and rarely cause neural compression".

They note that the lining of the cyst may or may not be arachnoidal tissue.

A number of terms are used in the medical literature so that the term arachnoidal cyst may be regarded as synonymous with: extradural arachnoid cyst, sacral meningocele, arachnoid pouch, arachnoid diverticula and meningeal cyst.

Nabors et al. ([iv]) classified spinal meningeal cysts as follows:

Type I: extradural cysts without nerve root fibres IA: extradural meningeal (arachnoid) cyst; IB sacral meningocele

Type II: extradural cyst with nerve root fibres Tarlov perineural cyst; spinal nerve root diverticulum

Type III: intradural cysts: intradural arachnoid cyst

The authors suggested that spinal meningeal cysts account for 1-3% of all spinal tumours and occur most frequently in the thoracic spine (65%), then lumbar/Lumbosacral (13%), thoracolumbar (12%), sacral (6.6%) and cervical spine (3.3%). Most of the lesions occur posteriorly in the spinal canal.

Thoracic cysts occur more in adolescents whereas sacral cysts are found more often in adults.

Josephson et al. ([v]) postulated a hypothesis explaining how spinal cord cysts form secondary to obstructions of the spinal canal such as caused by arachnoiditis. Using rats, they used ligation to achieve thecal sac constriction, which caused oedema either side of the ligation within 3 weeks and later cysts developed after 8 to 13 weeks.

The authors found that

"induced intramedullary pressure gradients originating from cerebrospinal fluid pulse pressure may underlie cyst formation in the vicinity of spinal canal obstructions and that cysts are preceded by edema."

Shah et al. ([vi]) noted that

"Acquired arachnoid cyst formation can occur with arachnoiditis of various aetiologies."

Santamarta et al. ([vii]) looked at the pathophysiology of arachnoid cysts. Cine-mode MRI showed 2 patterns of CSF flow within the cavity; some were harmonic with a patent flow entry zone; these patients tended to have non-progressive and non-localising symptoms and did not require surgical intervention.

Those with a more chaotic pattern of CSF flow with swirls throughout the cardiac cycle had a "more disabling clinical picture".

Endoscopy revealed that arachnoid cysts "always and variably" communicate with the subarachnoid space, CSF entering the cyst through a patent entry zone or via minute perforations within the arachnoid network, acting as a flexible mesh to modify the area of flowing CSF.

Poorly channelled slipstreams of CSF within the cyst can cause damage to the surrounding tissue.

Kumar et al. ([viii]) recently described 2 cases of symptomatic spinal arachnoid cysts.

These were of the noncommunicating intradural extramedullary type, which are rarer than communicating intradural extramedullary cysts.

They are a very rare cause of spinal cord compression and can rarely present with bizarre symptoms, such as angina.

Both cases involved spinal cord compression, which was relieved by surgical intervention.

They noted that arachnoid cysts are typically located in the midthoracic region dorsal to the spinal cord.

As the thoracic spinal canal is relatively small in diameter, cysts here tend to become symptomatic.

Usually intradural arachnoid cysts present in adolescence or early adulthood.

The authors divided intradural arachnoid cysts in adults into 5 categories:

1) Congenital

2) Arachnoid adhesions secondary to inflammatory process caused by infective agents (virus/bacteria/spirochaete)

3) Arachnoiditis secondary to subarachnoid haemorrhage, contrast media, spinal anaesthetics, meningitis, fibrin glue and bone dust

4) Trauma, lumbar puncture, intradural spinal surgery

5) Idiopathic (unknown cause)

They also discussed the various postulated theories as to the mechanism for enlargement of the cysts:

1. secretions of cells within the cyst wall

2. unidirectional valves

3. pathological distribution of arachnoid trabeculae leading to a diverticulum.

Agnoli et al. ([ix]) have hypothesised that these trabecular cells degenerate which causes an increased osmotic pressure within the cyst and thus transudation of fluid into the cyst.

Kumar et al. found arachnoid trabeculation and septation in both their cases.

Jean et al. ([x]) described 3 cases in which cervical arachnoid cysts caused spinal cord compression after repeated surgical decompression for Chiari II malformations.

All three children were treated for neural tube defect and later developed anteriorly situated arachnoid cysts compressing the brain stem and/or cervical cord.

The authors noted that an association between spinal arachnoid cysts and neural tube defect had previously been reported.

However, these were cases of previously undetected cysts, which seemed to develop after craniocervical decompression.

The authors suggested that the CSF dynamics were altered by the surgery, causing alternating compression and dilation of the anterior subarachnoid space.

They also noted that arachnoiditis might cause the CSF to become loculated and act as a mass.

Lee and Cho ([xi]) described 3 children with symptomatic intradural arachnoid cysts.

One was at T12-L1, compressing the conus medullaris, presenting with neurogenic bladder and cauda equina syndrome; the second was at C5-T1, causing spastic gait and neurogenic bladder; the third was at T2-3, presenting suddenly after playing skipping rope.

The authors remark that spinal arachnoid cysts are "relatively uncommon" and specifically the intradural type are "even less common".

They are usually asymptomatic but can cause symptoms that come on suddenly or gradually.

The MRI scans of the cases demonstrated the intradural arachnoid cysts with slightly lower CSF signal intensity on the gradient echo images and slightly higher signal intensity on T1-weighted images.

The authors suggested that intradural spinal arachnoid cysts "appear to result from an alteration of the arachnoid trabeculae".

They noted that some are associated with a history of trauma or with arachnoiditis.

Most are located posterior and in the thoracic region.

Lumbosacral arachnoid cysts can cause cauda equina syndrome. Ziv et al. ([xii]) looked at 2 children with cauda equina compression due to spinal arachnoid cysts.

One had neurogenic bladder dysfunction causing recurrent urinary tract infection, sensory loss in the lower limbs and abnormal tendon reflexes; the second had an unstable gait due to weakness and reduced sensation in the legs. MRI demonstrated cysts in both cases.

The authors concluded that lumbosacral arachnoid cysts are a rare cause of cauda equina syndrome in children.

Perineural Tarlov cysts, especially if multiple, can also be a cause of cauda equina syndrome, as first described by Tarlov himself.

Nicpon et al. ([xiii]) discussed a case of an 80 year old man presenting with cauda equina features, which were found to be due to a number of Tarlov cysts in the lumbosacral region.

Zarski and Leo reported that Tarlov cysts cause 7.3% of pain syndrome cases.

Paulsen et al. ([xiv]) showed that MRI revealed Tarlov cysts in 4.6% of patients although only 1% were symptomatic as a result.

The authors stated: "Lumbosacral perineurial cysts are common lesions that are usually asymptomatic but may cause pressure symptoms".

They suggested that whilst cyst puncture can alleviate the pain, the cysts repressurise causing return of the symptoms in most cases.

Voyadzis et al. ([xv]) looked at 10 cases of Tarlov cysts.

They noted that these cysts are found most often in the nerve roots in the sacral region. 7 of the patients, who were symptomatic, had cysts larger than 1.5cm in diameter, causing radicular pain or bladder/bowel dysfunction. 3 had smaller cysts associated with non-radicular pain.

Histopathological examination of specimens from 8 patients demonstrated nerve fibres in 75% of cases, ganglion cells in 25% of cases, and evidence of old haemorrhage in half.

The authors suggested:

"Tarlov cysts may result from increased hydrostatic pressure and trauma."

Nadler et al., ([xvi]) reporting on a case of a Tarlov cyst presenting as S1 radiculopathy, noted that the patient had had a normal MRI report, although review showed Tarlov cysts within the sacral canal at level S2 with compression of the adjacent nerve root.

This accounted for the patient's presenting symptom of posterior thigh pain.

Shaw et al. ([xvii]) reported a case of cauda equina syndrome with multiple lumbar arachnoid cysts in a patient with ankylosing spondylitis. The authors noted that early intervention is necessary before irreversible damage is done to the cauda equina.

Shih et al. ([xviii]) presented a case of a 9 year old girl who developed paraparesis and cauda equina syndrome as a result of an anteriorly located intradural arachnoid cyst.

Tsumoto et al. ([xix]) reported a case of thoracic intradural arachnoid cyst presenting as Brown-Sequard syndrome.

Interestingly, myelography and CT myelography failed to show the cyst wall. The authors noted that all 7 previous cases in the literature showing incomplete features of Brown-Sequard syndrome, all were at mid-thoracic level and 4 were in the midline.

They suggested that the laterality of the lesion and the asymmetrical circulation in the watershed area were important factors.

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