

Tarlov Cysts of the Spine: A Comprehensive Review

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Abstract

Tarlov cysts, also known as sacral perineural cysts, are cerebrospinal fluid-filled dilatations that arise between the endoneurium and perineurium of the posterior nerve root sheath, most commonly at the S2 and S3 levels. First described by Isadore Tarlov in 1938 as incidental autopsy findings, they are now recognised on magnetic resonance imaging (MRI) in roughly 4 - 9% of the population undergoing lumbosacral imaging, although only about 1% of those harbouring cysts become symptomatic. When symptomatic, Tarlov cysts may present with sacrococcygeal or radicular pain, sensory disturbance, bladder, bowel, or sexual dysfunction and, rarely, acute cauda equina syndrome following cyst haemorrhage or rupture. Their pathogenesis remains incompletely understood, but a one-way valve mechanism permitting cerebrospinal fluid ingress without egress, congenital dural wall weakness, and an association with heritable connective tissue disorders such as Ehlers-Danlos and Marfan syndromes have all been implicated. MRI is the diagnostic investigation of choice, with CT myelography reserved for confirming communication with the thecal sac before intervention. Management is graded according to symptom severity: asymptomatic and mildly symptomatic cysts are observed; moderately symptomatic cysts may respond to CT-guided percutaneous aspiration with fibrin sealant injection; and cysts causing progressive neurological deficit or refractory pain are considered for microsurgical treatment, most often cyst fenestration with imbrication of the redundant nerve root sheath and fibrin glue reinforcement. This review synthesises the anatomy, classification, epidemiology, clinical features, imaging characteristics, differential diagnosis, and current conservative, percutaneous, and surgical management strategies for spinal Tarlov cysts, with attention to outcomes and complications reported in the contemporary literature.

Keywords: *Tarlov Cyst; Perineural Cyst; Sacral Meningeal Cyst; Spinal Arachnoid Cyst; Cauda Equina; Cyst Fenestration; Fibrin Glue*

Introduction

Cystic lesions of the spinal meninges are an uncommon but increasingly recognised cause of low back pain, radiculopathy, and pelvic floor dysfunction. Among these, the perineural (Tarlov) cyst is the most frequently encountered subtype and the one most often confused, both clinically and radiologically, with other sacral and presacral masses. Named after the American neurosurgeon Isadore Tarlov, who first described these lesions as incidental autopsy findings in 1938, Tarlov cysts occupy a distinctive position in spinal pathology: they are common as incidental radiological findings yet remain a diagnosis of exclusion when symptomatic, and their management continues to generate debate almost nine decades after their initial description [1-4]. This review summarises the current understanding of the anatomy, pathophysiology, classification, epidemiology, clinical presentation, imaging, differential diagnosis, and treatment of spinal Tarlov cysts, with an emphasis on the evidence underpinning conservative, percutaneous, and surgical management.

Anatomy and pathophysiology

Tarlov cysts arise at the junction of the posterior spinal nerve root and its dorsal root ganglion, typically within the sacral canal, where the nerve roots are enclosed within a relatively unsupported dural sleeve and are subjected to the highest hydrostatic cerebrospinal fluid (CSF) pressure in the spinal canal because of upright posture. The cyst wall is composed of arachnoid and, in places, dural tissue, and the cyst cavity lies between the endoneurium and perineurium of the nerve root, distinguishing it histologically from a simple arachnoid diverticulum. Nerve fibres of the affected root are frequently found coursing through or along the cyst wall, which is the defining histological feature separating a true perineural cyst from other extradural meningeal cysts [5]. The precise mechanism of cyst formation remains debated. Tarlov's original hypothesis proposed that haemorrhage into the nerve root sheath, followed by venous or lymphatic obstruction, produced progressive cystic degeneration [1,3,4]. A more widely accepted contemporary model holds that a one-way, ball-valve communication develops between the cyst and the subarachnoid space, allowing CSF to enter the cyst during normal pulsatile pressure fluctuations but preventing its egress; this progressively distends the cyst, thins the surrounding bone, and can produce nerve root traction, compression, or venous congestion of the ganglion itself, which is thought to be the principal source of neuropathic pain rather than mechanical compression alone [5-7]. Cysts may enlarge over years and erode the surrounding sacral lamina and neural foramina, producing the smooth, scalloped bony remodelling that is characteristic on cross-sectional imaging.

Classification

The classification proposed by Nabors and colleagues in 1988 remains the standard framework for spinal meningeal cysts and is essential for distinguishing Tarlov cysts from other cystic lesions that may mimic them clinically and radiologically. Type I cysts are extradural meningeal cysts without spinal nerve root fibres in their walls and are further subdivided into Type IA (extradural arachnoid cysts) and Type IB (sacral meningoceles). Type II cysts are extradural meningeal cysts that contain nerve root fibres in their walls; the Tarlov (perineural) cyst is the prototype of this category. Type III cysts are intradural meningeal (arachnoid) cysts [5]. This distinction has direct clinical relevance because Type I and Type III cysts more often communicate freely with the subarachnoid space and behave differently on CT myelography, whereas the valve-like communication of Type II cysts explains their tendency to enlarge progressively and their more variable response to simple decompression.

Epidemiology and associated conditions

Incidental perineural cysts are identified on 4 - 9% of lumbosacral MRI studies performed for unrelated indications, and pooled meta-analytic data suggest a global prevalence of approximately 4 - 5% of the imaged population, with a consistent female predominance of roughly 2:1 [8-10]. Most of these cysts are small, asymptomatic, and require no treatment; only an estimated 1% of patients with a Tarlov cyst develop symptoms attributable to it, and symptomatic disease is most often diagnosed in the fourth to sixth decades of life [6,9,11]. An association between Tarlov cysts and heritable disorders of connective tissue, particularly Ehlers-Danlos syndrome and Marfan syndrome, has been repeatedly reported in case series and is thought to reflect underlying weakness of the dura and perineurium that predisposes to cyst formation and dural ectasia more broadly, although robust epidemiological confirmation of this association is still lacking [12-14].

Clinical presentation

Symptomatic Tarlov cysts most often present with chronic dull or aching low back, sacral, or coccygeal pain that is characteristically aggravated by activities that raise intraspinal CSF pressure, such as prolonged standing, sitting, the Valsalva manoeuvre, coughing, or straining, and is sometimes relieved by recumbency. Radicular pain in an S1-S3 distribution, perineal or buttock pain, and paraesthesia along the posterior thigh and leg are common. Because the sacral roots also carry autonomic and pelvic floor innervation, larger or multiple cysts can produce bladder dysfunction, including urgency, frequency, incomplete emptying, or retention; bowel dysfunction, including constipation or faecal incontinence; and sexual dysfunction, including, uncommonly, persistent genital arousal-type symptoms

[6,9,11,15]. Motor weakness is less common and, when present, typically reflects compression of the ventral motor rootlets by a large or multiloculated cyst. Rarely, spontaneous haemorrhage into a cyst or acute cyst rupture can precipitate abrupt cauda equina syndrome, which constitutes a surgical emergency [16]. Given the non-specific nature of these symptoms, which overlap considerably with degenerative lumbosacral disease, pelvic floor dysfunction, and gynaecological or urological pathology, a high index of suspicion, combined with correlation between the size, location, and laterality of the cyst and the patient's symptoms, is essential before symptoms are attributed to an incidentally discovered cyst.

Diagnostic imaging

MRI of the lumbosacral spine is the investigation of choice and typically demonstrates a well-circumscribed, thin-walled lesion that follows CSF signal on all sequences, being hypointense on T1-weighted images and hyperintense on T2-weighted images. The lesion most often arises from the S2 or S3 nerve root sleeve, with associated smooth scalloping or erosion of the adjacent sacral lamina in longstanding cases [17,18]. Contrast administration is useful for excluding an enhancing solid or complex cystic mass. Computed tomography (CT) is complementary and demonstrates the characteristic bony remodelling of the sacral canal to good effect, while CT myelography remains the reference standard for confirming free or delayed communication between the cyst and the subarachnoid space. This information is important for pre-operative planning and for differentiating a Type II perineural cyst from a Type I or Type III meningeal cyst [5,8,18]. Electrodiagnostic studies, including electromyography and nerve conduction studies, may be used to corroborate the clinical relevance of a radiologically identified cyst when the diagnosis remains uncertain.

Differential diagnosis

Because the sacrum is a common site for both meningeal cysts and a wide range of other pathologies, Tarlov cysts must be differentiated from sacral meningoceles (Nabors Type IB), intradural arachnoid cysts (Type III), spinal or presacral schwannomas and other nerve sheath tumours, dermoid and epidermoid cysts, chordoma and other primary sacral bone tumours, synovial or juxta-articular cysts, and, in female patients, adnexal or ovarian cystic masses, with which pelvic Tarlov cysts have been repeatedly misdiagnosed on transvaginal or abdominal ultrasound [18]. The presence of a lesion in continuity with a neural foramen, its CSF signal characteristics, and demonstration of communication with the thecal sac on CT myelography are the key features that establish the correct diagnosis.

Management

There is no universally accepted treatment algorithm for Tarlov cysts, and management should be individualised according to cyst size, the severity and trajectory of symptoms, and the degree of correlation between the cyst and the patient's clinical picture. In general, a graded approach progressing from observation through minimally invasive percutaneous techniques to microsurgical treatment is favoured [9,19].

Conservative management

The majority of Tarlov cysts are asymptomatic or minimally symptomatic and are managed with reassurance, serial clinical follow-up, and symptomatic measures such as analgesia, activity modification, and pelvic floor physiotherapy. Because natural history data suggest that most cysts remain stable over years, observation is appropriate whenever symptoms are mild or when the correlation between the cyst and the presenting complaint is uncertain [6,9].

Percutaneous techniques

For patients with moderate, persistent symptoms who have failed conservative measures, CT-guided percutaneous aspiration of cyst fluid, with or without instillation of a fibrin sealant, offers a minimally invasive alternative to open surgery. Early reports of simple needle aspiration demonstrated only transient relief, prompting the addition of fibrin glue to promote fibrosis of the cyst wall and reduce recurrence

[17,20]. Subsequent single- and two-needle aspiration-injection protocols have reported satisfactory symptomatic improvement in most treated patients with a low complication rate, although recurrence requiring repeat intervention is not infrequent [21-24]. The largest published series, comprising 213 patients treated with a two-needle CT-guided aspiration and fibrin sealant technique, reported that approximately 82% of patients were satisfied with their outcome at one year, with sustained benefit in around three-quarters of patients at three to six years and no clinically significant complications [24]. A subsequent 15-year, 220-patient retrospective institutional experience similarly supported percutaneous aspiration and fibrin glue injection as the predominant interventional approach among the 95 patients who underwent intervention, reserving open surgery for cysts that recur or fail to respond [25]. Diagnostic aspiration, in which transient reproduction or relief of the patient's typical symptoms is used to confirm the clinical significance of the cyst, has also been advocated as a useful adjunct before committing a patient to surgery.

Surgical management

Surgery is generally reserved for large, refractory, or rapidly progressive symptomatic cysts, particularly those associated with objective neurological deficit, bladder or bowel dysfunction, or a positive response to diagnostic aspiration. A variety of microsurgical techniques have been described via a posterior sacral laminectomy or laminotomy approach, including simple cyst fenestration and drainage, cyst wall resection down to the nerve root, imbrication (plication) of the redundant, attenuated nerve root sheath to obliterate the cyst cavity while preserving functioning nerve fibres, obliteration of the CSF fistula connecting the cyst to the subarachnoid space, and reinforcement of the repair with fibrin glue or a muscle or fat graft. Cystoperitoneal shunting and, for very large or multiloculated cysts, more extensive sacral reconstruction have also been reported for refractory cases [6,7,11,15,19,26]. Comparative single-institution series have found broadly similar rates of clinical improvement with fenestration and nerve root imbrication, in the region of 80%, although the choice of technique is often dictated by the cyst's size, wall thickness, and the density of nerve fibres traversing it [15]. A systematic review and meta-analysis of surgical series reported clinically meaningful symptomatic improvement in a majority of operated patients, most consistently for neurological deficits and bladder or bowel symptoms, while chronic pain proved somewhat less reliably responsive to surgery than sensory or motor deficits [10,27]. Reported surgical complication rates range from approximately 20% to 40% and include CSF leak, pseudomeningocele, wound infection, meningitis, and, less commonly, worsening of pre-existing neurological deficit. Recurrence following fenestration alone has been reported in a substantial proportion of patients in some series, which has driven a preference for imbrication or fenestration combined with fibrin sealant reinforcement in more recent practice [11,15,19].

Outcomes and prognosis

Across both percutaneous and surgical series, neurological deficits and pelvic autonomic symptoms tend to show more consistent and durable improvement than chronic pain, which may persist even after technically successful cyst obliteration, presumably reflecting established neuropathic sensitisation. Recurrence is the principal long-term concern with both percutaneous and surgical treatment and underscores the rationale for long-term clinical and, where indicated, radiological follow-up. Given the relatively small size of most published series, the absence of randomised comparative data, and considerable heterogeneity in patient selection and outcome measures, current recommendations for the choice of intervention rest on a low level of evidence, and further prospective comparative studies are needed to define an optimal, evidence-based treatment algorithm [26-30].

Conclusion

Tarlov cysts are a common incidental finding on lumbosacral MRI but an uncommon, and frequently under-recognised, cause of chronic sacral pain, radiculopathy, and pelvic autonomic dysfunction. A sound understanding of their anatomy, the Nabors classification, and their characteristic imaging features allows Tarlov cysts to be reliably distinguished from other, more consequential, sacral pathologies. Management should be graded according to symptom severity, beginning with observation for asymptomatic or mildly symptomatic disease, progressing to CT-guided percutaneous aspiration and fibrin sealant injection for moderate, refractory symptoms, and reserving

microsurgical fenestration or nerve root imbrication for patients with progressive neurological deficit or symptoms refractory to less invasive measures. Continued accumulation of comparative outcome data will be required to refine patient selection and establish a more definitive, evidence-based treatment algorithm for this enigmatic condition.

Conflict of Interest

The authors declare no conflict of interest.

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