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REVIEW ARTICLE

Compressive cervicothoracic pseudomeningocele as a rare manifestation of idiopathic intrathecal hypotension after past trauma: a review

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Abstract

Purpose Spontaneous intracranial or intrathecal hypotension (SIH) is an underdiagnosed phenomenon predominantly presenting with low cerebrospinal fluid (CSF) pressure and postural headache in the setting of CSF leak. Extrathecal CSF collections causing compression of the spinal cord or nerve roots present an even rarer subset of this disease. We aim to describe this pathology in a comprehensive manner while illustrating with a case of our own.

Methods We present a literature review on spinal idiopathic pseudomeningoceles and their neurological implications illustrated with a case of an anterior compressive pseudomeningocele between C2 and D7. Further investigations through a myelography and myelo-CT were able to postulate a CSF leak through a discogenic osteophytic microspur at the level C5-C6.

Results Spinal manifestations are uncommon in cases of idiopathic or spontaneous CSF leak, occurring in about 6% of patients, but myelopathy and radiculopathy involving all spinal segments do occur. In contrast to the cranial complaints, the spinal manifestations usually are not positional and are caused by mass effect from an extradural CSF collection.

Conclusion The utility of multiple imaging modalities such as dynamic myelography and the use of epidural blood patches and fibrin glue polymers should be explored, and surgery is an option if the symptoms persist despite other measures.

Keywords Spontaneous intracranial hypotension · Intrathecal hypotension · Idiopathic/traumatic cervical pseudomeningocele · Cervical myeloradiculopathy · Spinal cord compression

Introduction

Spinal pseudomeningoceles are extremely rare and can be congenital, iatrogenic after spinal procedures or post-traumatic. The current knowledge only described a few cases of an anterior cervical compressive cerebrospinal fluid (CSF) collection limited to the spinal canal and causing neurological symptoms through cord or nerve root compromise [1].

The currently known pathophysiological concept describes a CSF leak after a predisposing event [1–3]. The subsequent pseudomeningocele will then develop either acutely when in the setting of an iatrogenic origin, or progressively during years for those idiopathic or spontaneous after a past significant craniocervical trauma. In this last case, symptoms may therefore appear in a more delayed fashion and the correlation between the traumatic event and the collection needs a careful investigation.

Symptoms may also include positional headaches, nausea or vomiting and tinnitus, all four pointing toward a spontaneous intracranial hypotension pattern (SIH) [2]. Rarely, cord and root herniations can develop through these collections leading to myelopathy or meningitis [1–4].

Nevertheless, the fundamentals and management strategy of such disease are still object of discussion and investigation.[4–9]

We aim to present a literature review and to stimulate discussion on the pathophysiology, clinical presentation and possible diagnostic strategy and management of spinal idiopathic pseudomeningoceles. We also correlate these as spinal manifestations of the same etiology also causing SIH, illustrating this article with a case of an idiopathic

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anterior cervical pseudomeningocele causing neck pain and radiculopathy.

Methods

A literature review was conducted through an unrestricted search using the keywords ((‘Spontaneous intracranial hypotension’ OR ‘CSF leak’) AND (‘spinal pseudomeningocele’ OR ‘compressive pseudomeningocele’)) on 15th July 2020 on the following databases: Embase, Cochrane Library, PubMed, Google Scholar and Web of Science resulting in a list of 52 references (Fig. 1).

All references in the list were scanned to identify additional potentially relevant studies ($n=4$). Two reviewers independently screened titles and abstracts of all identified literature, and full-text copies of all relevant articles were acquired. In the case of a discrepancy, a third author would arbitrate until there was a consensus among the authors. After abstract screening, 27 articles were excluded and 29 were fully read in order to assess final eligibility. Full manuscripts were obtained and analyzed as well as some pertinent mentioned references. The Table 1 provides a summary of patient demographics and results for each case.

Results

Case description

A 53-year-old male patient without any known co-morbidity presented to the outpatient spine surgery clinic in December 2017, referred by his family doctor. He complained about bilateral neck and arm pain, predominant on the right side and following a multi-dermatomal distribution since the last three years and progressing.

The neurological examination could elicit a Spurling sign with no signs of clinical myelopathy. There wasn’t any sensory or motor deficit despite some fatigability of both hands.

A first cervical spine magnetic resonance imaging (MRI) showed an anterior epidural collection between C2 to D8, hypointense in T1-weighted and hyperintense in T2-weighted images, initially described as an arachnoid cyst (Fig. 2).

A supplemental myelography and myelo-computer tomography (CT) scan further confirmed the hypothesis of a pseudomeningocele with no identifiable CSF leak and a suspicious C5-C6 posterior osteophytic microspur (Fig. 3).

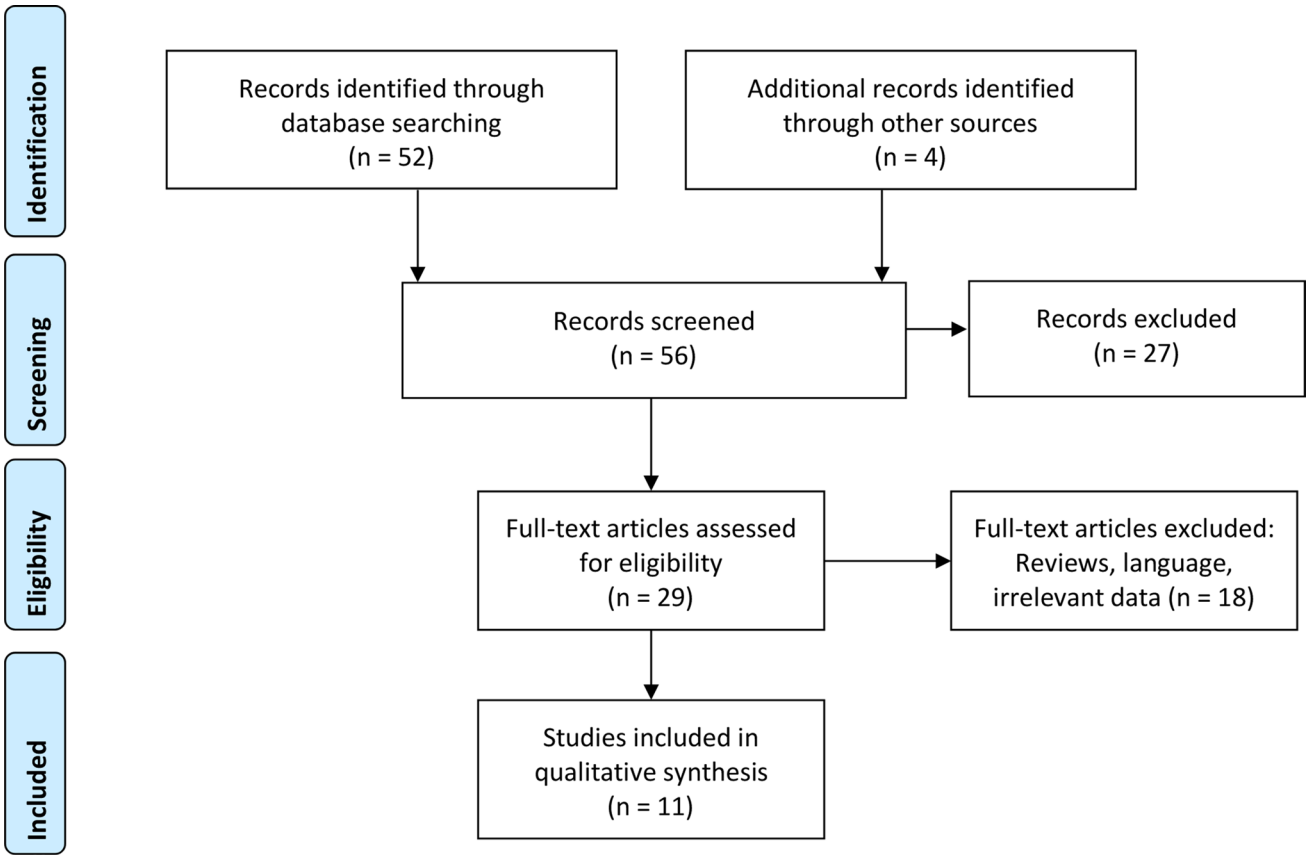

Fig. 1 Literature search flowchart and search results

Table 1 Spinal idiopathic compressive pseudomeningoceles (CSF-cerebrospinal fluid; EBP-epidural blood patch; HA-headache; HNP-herniated nucleus pulposus; LE-lower extremities; op-operation; SIH-spontaneous intracranial hypotension; UE-upper extremities)

Author, year	Age (yrs), sex	Initial symptom	Spinal manifestations	Examination	Brain MRI	Spine MRI/ myelography	Treatment	Outcome
Cervicothoracic myelopathy								
[11]	17, M	Left C8 dysesthesias	Progressive paraplegia	Paraplegic	Normal	Extrathecal CSF collection at C6-T4	Op	Partial recovery
[12]	60, M	Tetraplegia	Tetraplegia	Sensory level at C4	Normal	Extrathecal CSF collection at C4-T3	op	Partial improvement
[14]	39, M	Left UE weakness	Left LE weakness and atrophy	Left LE weakness and atrophy	Normal	Extrathecal CSF collection at C6-C8	op	
17	26, M	Right arm weakness	Progressive right arm paresis	Right arm paresis	Normal	Extrathecal CSF collection at C3-L4	op	Resolution
[18]	37, M	Left arm numbness and tingling	Progressive bilateral hypoesthesia	Right arm paresis	Normal	Extrathecal CSF collection at C5-T7	op	Resolution
[15]	38, M	Orthostatic HA	Bilat UE numbness; Lhermitte sign	Normal	SIH	Extrathecal CSF collection C5-T10	EBP, op	resolution
	46, F	Orthostatic HA	Numbness, paresthesia in trunk and UEs/LEs	Normal	SIH	Extrathecal CSF collection at C3-T12	None	Persistent symptoms
	52, F	Reverse orthostatic HA	Transient quadriplegia	Normal	SIH	Extrathecal CSF collection at C5-T11	EBP	Resolution
	17, M	Orthostatic HA	Lhermitte sign	Pathological UE and LE reflexes	SIH	Extrathecal CSF collection at C2-L2	EBP, op	Resolution
	43, F	Orthostatic HA	Numbness, paresthesia body and extremities	Pathological UE reflexes	SIH	Extrathecal CSF collection at C2-L2	EBP, op	Resolution
	27, F	Orthostatic HA	Numbness, paresthesia UEs/LEs	Normal	SIH	C5-C6 HNP, thoracolumbar meningeal diverticula	EBP	Resolution
	27, M	Orthostatic HA	Ascending numbness	T8 sensory level, ankle clonus	SIH	Extrathecal CSF collection at C5-L1	EBP, op	Resolution
	24, F	Nonpositional HA	Ascending numbness	T4 sensory level	SIH	Extrathecal CSF collection at C1-L5	EBP, glue	Resolution
[15]	41, M	Left hemiparesis	Left hemiparesis	Left hemiparesis	Normal	Extrathecal CSF collection at C3-lumbar spine	op	Resolution

Table 1 (continued)

Author, year	Age (yrs), sex	Initial symptom	Spinal manifestations	Examination	Brain MRI	Spine MRI/ myelography	Treatment	Outcome
[13]	20, M	Right UE hypoesthesia	Right UE weakness and dysesthesias	Rith UE weakness and hypoesthesia	Normal	Extrathecal CSF collection at C6-T1	op	Resolution
	21, M	Right brachial plexus avulsion	Right UE pain and weakness	Diffuse right UE weakness	Normal	Extrathecal CSF collection at C7	None	Resolution
Cervicothoracic radiculopathy								
[17]	35, M	Right hand weakness	Right hand weakness	Right hand weakness	Normal	Extrathecal CSF collection at T1 left nerve root	Op	Partial improvement
[1]	26, F	Orthostatic HA	Bilateral C6-C7 paresthesia	Normal	SIH	Extrathecal CSF collection at C3-T9	EBP, op	Resolution
	60, F	Orthostatic HA	Left C7 pain	Normal	SIH	Thoracic meningeal diverticula	EBP, glue	Resolution
	47, F	Orthostatic HA	Right C7, C8 pain	Normal	SIH	Extrathecal CSF collection at C4-L3	EBP, glue, op	Resolution
	35, F	Orthostatic HA	Left C7 pain/ weakness	Triceps weakness	NL	Extrathecal CSF collection at C6-T10	EBP, op	Resolution
	39, F	Orthostatic HA	Bilateral C7 paresthesia, numbness	Triceps weakness	SIH	Extrathecal CSF collection at C6-T1	EBP, op	Resolution
	56, F	Orthostatic HA	Right T7-T8 pain	Normal	SIH	Extrathecal CSF collection at C7-L1	EBP	Resolution
	34, F	Orthostatic HA	Bilateral T5 pain/paresthesia	Normal	SIH	Extrathecal CSF collection at C3-T12	EBP	Resolution
	25, F	Orthostatic HA	Right T8 pain	Normal	NL	Extrathecal CSF collection at C1-L3	Op	Resolution
[15]	15, F	Left arm paresthesia	Paresthesia left arm	Paresthesia left arm	Normal	Extrathecal CSF collection at C3-lumbar spine	None	Resolution
	23, M	Right arm hypoesthesia	Right arm hypoesthesia	Right arm hypoesthesia	Normal	Extrathecal CSF collection at C3-lumbar spine	None	Resolution
	52, M	Right arm weakness	Right arm weakness	Right arm weakness	Normal	Extrathecal CSF collection at C2-L1	Op	Partial improvement
[16]	12, M	Neck pain	Neck pain	Neck pain	Normal	Extrathecal CSF collection at C2-T1	None	

Table 1 (continued)

Author, year	Age (yrs), sex	Initial symptom	Spinal manifestations	Examination	Brain MRI	Spine MRI/ myelography	Treatment	Outcome
Bilateral amyotrophy								
[1]	22, M	Orthostatic HA	Atrophy and weakness of bilateral UEs	Shoulder atrophy, weakness, fasciculations	NL	Extrathecal CSF collection at C2-L1	EBP, op	No improvement
Lumbosacral radiculopathy								
[1]	16, F	Orthostatic HA	Right S1 pain	Normal	SIH	Lumbosacral dural ectasia	None	Persistent symptoms
	24, F	Orthostatic HA	Right S1 pain	Normal	SIH	Lumbosacral dural ectasia	EBP	Resolution
	58, M	Orthostatic HA	Bilateral L3-L4 paresthesia	Normal	SIH	CSF along lumbar nerve roots	EBP	Resolution
Asymptomatic								
[19]		Atlantoaxial instability	Neck pain	Normal	Normal	Extrathecal CSF collection C0-C2		
[20]	33, M	Asymptomatic	None	Normal	Normal	Extrathecal CSF collection C2-C3 and retropharyngeal collection	None	Resolution
[15]	52, M	Asymptomatic	None	Normal	Normal	Extrathecal CSF collection at C3-lumbar spine	None	Resolution

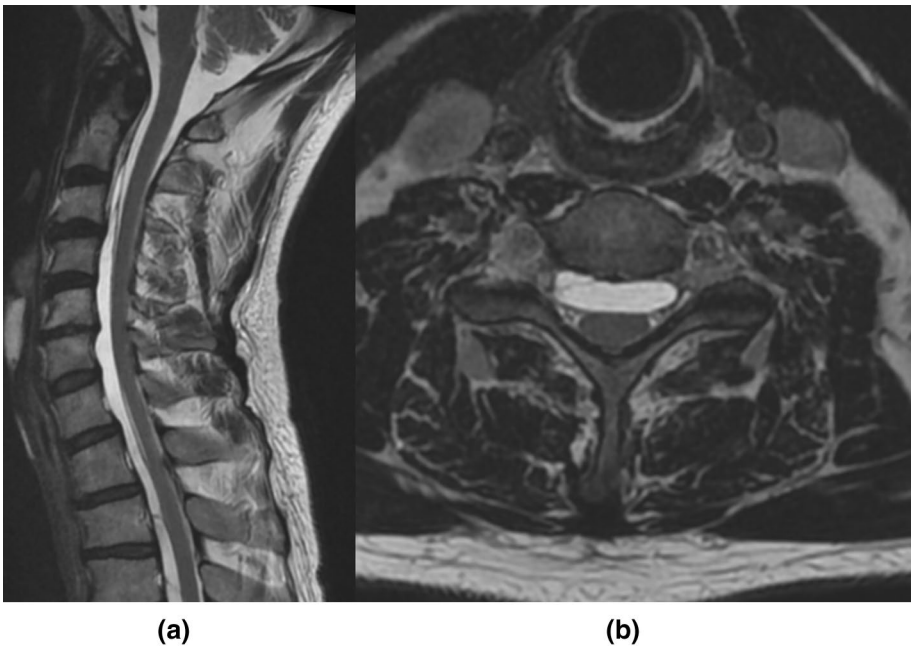

Fig. 2 T2 sagittal (**a**) and axial cuts (**b**) shwing the spontaneous hyperintense anterior extrathecal cervico-thoracic compressive pseudomeningocele

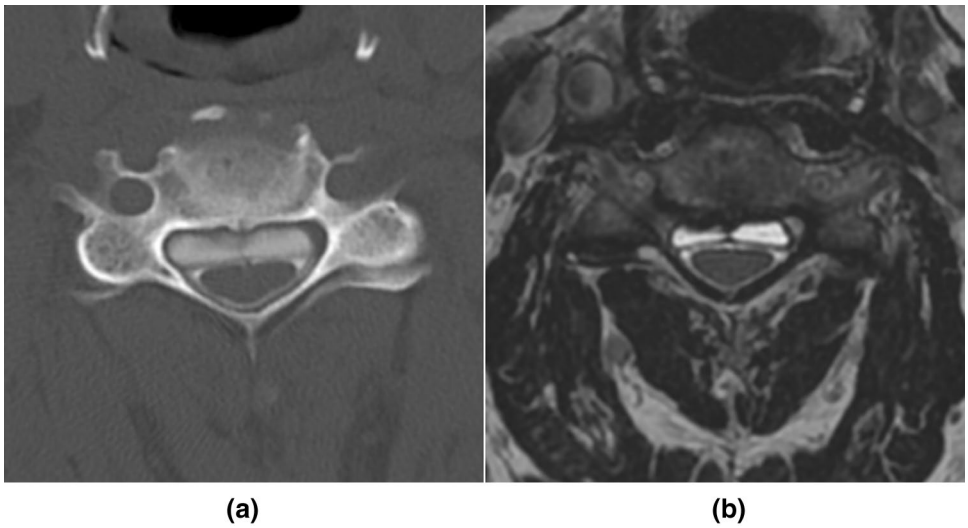

Fig. 3 Myelo-CT (**a**) and MRI T2-weighted (**b**) axial cuts showing the C5-C6 bony microspur and the pseudomeningocele (axial cuts)

This finding motivated further anamnestic investigations with a late discovery that our patient was a former competitive skier with history of multiple head and neck traumas, one of which leaded to a coma lasting for several hours about twenty years ago.

Following clinical worsening with left neck and radicular pain refractory to physiotherapy, an electroneuromyography found signs compatible with a chronic C5 and C6 nerve root irritation with supraspinous and infraspinous muscle atrophy, without any abnormality on somatosensory and motor evoked potentials (SSEPs and MEPs).

Taking into account, the absence of neurological deficits it was then decided to start with a probatory blood patch at the suspected leak level C5-C6. After this procedure, symptoms showed to be almost fully resolved and remained stable since with a slightly decrease in the pseudomeningocele as well (last control in September 2019) (Fig. 4). Our patient was able to regain work and daily activities at 100%.

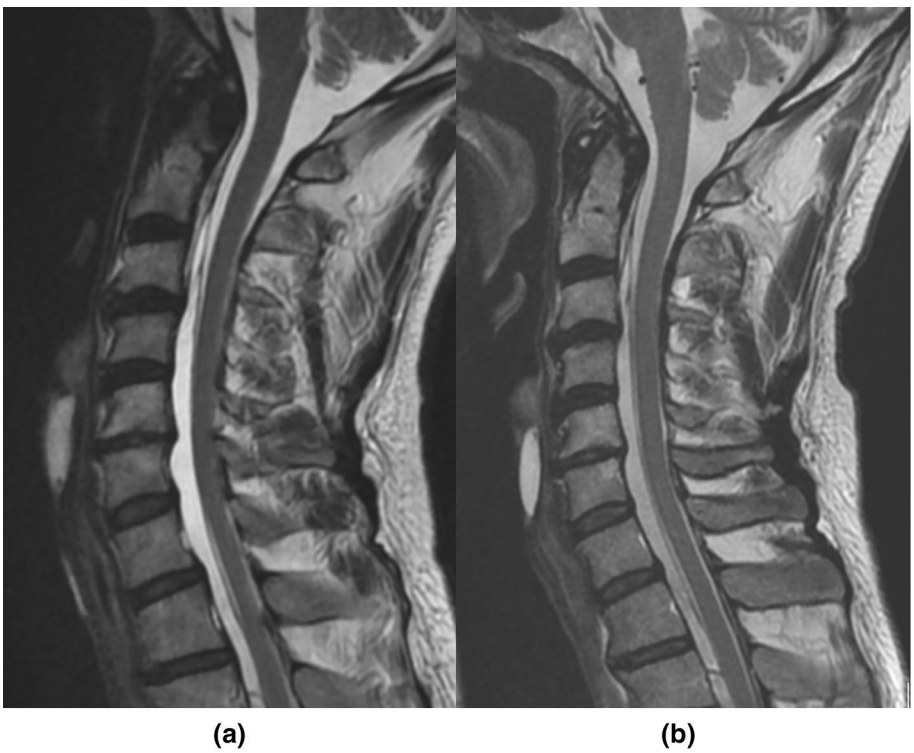

Fig. 4 Partial regression of the pseudomeningocele at the last follow-up (T2-weighted sagittal cuts pre-treatment (**a**) and at last follow-up (**b**))

Discussion and review

This rare case of an anterior cervical compressive pseudomeningocele in an adult male known for multiple spinal traumatic episodes in the context of professional high-level ski practice serves to illustrate this rare group of pathologies causing radiculomyelopathy.

Although there are many case reports of delayed post-traumatic spinal pseudomeningoceles, only thirty-six were epidural[1, 10–20] with most being formed outside the spinal canal in the retropharyngeal, posterior, or posterolateral tissues and after brachial plexus nerve root avulsion. Among the epidural CSF collections, less than ten were purely within the spinal canal[1] and causing cord compression. All became symptomatic in a delayed fashion and developed after severe upper cervical or craniovertebral injuries, some associated with vertebral instability [14, 18]. As described by other colleagues, most of these collections tend to respond to blood patches with a significant clinical improvement [1]. Less than half needed a surgical procedure due to whether osteoligamentous instability or persistent symptoms. Lastly, only four patients presented with acute symptoms after trauma (Table 1) [14, 18–20].

Most likely these CSF collections usually occur iatrogenically after surgery, both immediately or post-operative due to bony spurs or remnants that breach the dura. Their incidence after lumbar surgery ranges from 0.1 to 43%, depending on the type of surgery performed.[1–3]

The development of the remaining cases is therefore not so obvious to understand.

Pathophysiology

In vitro experimentation in order to evaluate spinal post-traumatic pseudomeningocele pathogenesis and formation is limited in humans due to clear ethical limitations. This gave rise to a couple of theories based on scattered data from different fields such as biophysics, radiology, clinical reports and basic sciences. After gathering converging evidences, one can put forth that a major cause of pseudomeningocele is trauma affecting the spine or brachial plexus [1, 3, 10, 14]. The presentation of such lesions can be subtle or dramatic, depending on the compartment that the pseudomeningocele occupies. A small breach in the dura and a subsequent ball-valve mechanism of expansion are thought to begin the process of enlargement (to a point where the collection becomes radiologically and/or clinically important) [1]. Later, discussion from Beretta et al.[14] added a possible anatomical explanation for the fact that this second lumen develops preferentially from and below C2 level. It was postulated that as a result of a very strong ligamentous complex, the dura that extends from C0 to C2 levels appears to be strongly adherent to the thick ligamentous apparatus and is reinforced by multiple layers of connective tissue. Therefore, below the inferior third of the axis the dura is less adherent to adjacent tissues and is only covered by the posterior longitudinal ligament, down to the entire subaxial cervical segment and the rest of the spine. This theory concluded to a possible ‘‘transitional zone’’ of the dura acting as a point of minor resistance between two opponent forces: the elastic force of the subaxial dura and the stiff component of the upper cervical connective tissue, including the dura adherent to the ligamentous complex. These facts point toward a trend between upper cervical traumatic injuries and concomitant pseudomeningoceles [1, 3, 10, 14].

Clinical and radiological features

Concerning the differential diagnosis, among the possible etiologies for radiculomyelopathy symptoms, one must be aware of the most common degenerative cause as stenosis or herniations aside from the normally abrupt, spinal epidural and subdural hematomas and less frequent intra- or extradural tumors or demyelinating diseases. As in any case of compressive lesion of the spine, early detection and prompt treatment are essential to limit neurological injury.

The initial diagnostic workup shall comprise a craniocervical MRI which can lead to the discovery of a spinal epidural collection and SIH indirect signs. This one can be hematic or CSF. Some authors postulate that among these hematomas one can have several CSF collections associated with a small amount of blood products within the cerebrospinal fluid [1, 2, 4, 10]. If a pseudomeningocele is confirmed, the next step shall target the exact location of the contrast passage from the intrathecal to the extrathecal space. The search for the leak also includes the exact anatomic site on the circumferential aspect of the spinal dura. Several protocols are described with some preforming a direct myelography with dynamic (lateral decubitus) images and post-myelography spine CT imaging, including repeated films (4 h later) if initially negative with or without concomitant thin-slice MRI of the spinal axis [1, 4, 5, 10]. Additional electrodiagnostic studies are useful in determining the site of the lesion in brachial plexus injuries. These include measuring parameters such as the compound motor action potential (CMAP) and sensory nerve action potential (SNAP), in addition to conducting needle electromyography (EMG). EMG can aid in distinguishing between preganglionic and postganglionic lesions by evaluating proximal muscles such as the serratus anterior and rhomboid. SSEPs and MEPs can also ensure a better monitoring of a potentially mild sub-clinic myelopathy [1].

Management strategies and outcome

Regarding management, most cases present incidentally or respond well under conservative treatment. Careful observation with serial MRI and SSEP and MEP can be proposed [1]. A spontaneous regression or reduction of the epidural CSF collection can occur, perhaps because the pressure inside the pseudomeningocele reaches a level great enough to overcome the ball-valve effect [20] leading to their resolution. Bed rest, oral and intravenous hydration, pain medication (acetaminophen, ibuprofen, metamizole, morphine), and supportive caffeine constitute the first level of care [1, 2]. Epidural blood patching (EBP) with 20–40 ml of autologous blood can be performed, if known, at the site of the leak and optionally repeated [2, 5–9]. The final diagnostic and therapeutic measure can be microsurgical exploration of the spine at the site of the CSF leak but only for patients were the localization of the CSF leak is known and having at least one failed EBP or progressive neurologic deficits [2, 8]. Our review showed a pair of described techniques to repair these CSF leaks and pseudomeningoceles with better outcomes using the intra(inlay)/extradural(onlay) approach (fascia lata graft, muscle graft or TachoSil (Ethicon Inc, Johnson and Johnson, New Jersey, United States) and fibrin glue) plus dural suture when feasible using autologous fascia lata grafts [8, 14].

Looking at our case from a critical perspective, it was decided not to repeat the myelography or to perform a dynamic exam because of the remarkable improvement after the blood patch. These exams though, shall be organized, together with a new thin-cut MRI in case of future clinical deterioration.

All in all, the discussed causal relationship has not yet been validated in vitro. With only a single illustration from our side, it isn't possible to do so, but together with the learnings from the SIH field that represents a more cranial manifestation of the same leakage problem, one can propose a diagnostic and follow-up protocol paradigm for this specific group of patients.

Conclusions

Spinal manifestations are uncommon in cases of idiopathic/traumatic or spontaneous CSF leak, occurring in about 6% of patients, but myelopathy and radiculopathy involving all spinal segments do occur. Pseudomeningoceles do occur, most being paraspinal [1, 2], but collections limited to the canal are quite rare. In contrast to the cranial complaints, the spinal manifestations usually are not positional and are caused by mass effect from an extradural CSF collection. Magnetic resonance imaging can help differentiating CSF collection from spinal epidural hematomas and dynamic myelography and myelo-CT can guide the treatment strategy helping to point out the CSF leak. The use of epidural blood patches should be explored, and surgery is feasible and successful if symptoms persist despite other measures. Nevertheless, optimal management for large cervical compressive pseudomeningoceles still must be decided on an individual basis.

Author contributions All authors have read and agree to the published version of the manuscript. Conceptualization, R.G. and G.M.; methodology, R.G.; formal analysis, R.G.; investigation, R.G. and I.F.B.; resources, R.G. and I.F.B.; data curation, R.G.; writing—original draft preparation, R.G. and I.F.B.; writing—review and editing, R.G. and D.V.; visualization, R.G.; supervision, G.M.; project administration, R.G.

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Compliance with ethical standards

Conflicts of interest The authors declare that there is no conflict of interest.

Availability of data and material Data can be provided upon request.

Consent for publication Consent for publication obtained from the patient.

Ethics approval As a review paper no ethics approval was needed.

Informed consent Consent to participate obtained from the patient.

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