dementia. At the age of 63 years, one year and a half after the onset of impairment, she scored 9/30 on the MMSE. We ruled out more aggressive immunomodulatory treatments.

The clinical spectrum of anti-GAD syndrome is still being reviewed.[2,3] Very few publications have analysed the presence of cognitive impairment in patients with anti-GAD antibodies, although the current evidence seems to support an association between both entities.[4,5] An isolated case report of cognitive impairment associated with anti-GAD antibodies describes a similar clinical profile to that observed in our patient, with no response to IVIG treatment.[6] The decision to start treatment with aggressive immunomodulatory treatments in these patients should be made with caution, taking into account each patient's personal situation and the variable response of other anti-GAD—associated syndromes such as cerebellar ataxia. Furthermore, the sequential onset of cerebellar ataxia, SPS, and rapidly progressive cognitive impairment in the same patient demonstrates symptom overlap, with different symptoms coexisting and manifesting asynchronously. Our study lacks data from monitoring of the antibody titres throughout disease progression. However, future studies may provide evidence on whether variations in antibody titres may be related to the onset of new symptoms.

With this publication, we aim to contribute to the understanding of the clinical spectrum of anti-GAD—associated syndromes. Cognitive impairment may be part of the spectrum of associated clinical manifestations, and a low level of suspicion may delay diagnosis and the chance to start or intensify treatment in early stages of impairment.

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Spinal arachnoid cysts: a delayed manifestation of post hemorrhagic arachnoiditys☆

Quistes aracnoideos espinales: una manifestación tardía de la aracnoiditis poshemorrágica

Dear Editor:

Subarachnoid haemorrhage can cause a chronic leptomeningeal inflammatory response, manifesting with alterations in the anatomical architecture of the arachnoid membrane, known as arachnoiditis.[1] These changes can range from mild thickening to severe adhesions in the subarachnoid space, including the formation of cysts that can cause spinal cord compression[1]; while these are rare, they usually follow certain patterns of regional localisation and time of onset. The case described in this article presents several differences from the other cases reported to date.

Clinical case

The patient is a 52-year-old man with history of subarachnoid haemorrhage secondary to rupture of an aneurysm in the left posterior inferior cerebellar artery, treated with embolisation, and ventricular-peritoneal CSF shunt due to hydrocephalus; the valve was replaced on 3 occasions due to obstruction caused by blood remnants in the system. Eight years later, he developed progressive spastic tetraplegia secondary to extrinsic spinal cord compression, which MRI revealed to be due to arachnoid cysts between C2 and C6, predominantly anterior to the spinal cord; these findings were associated with thinning of the spinal cord and caused syringomyelia between T4 and the conus medullaris.

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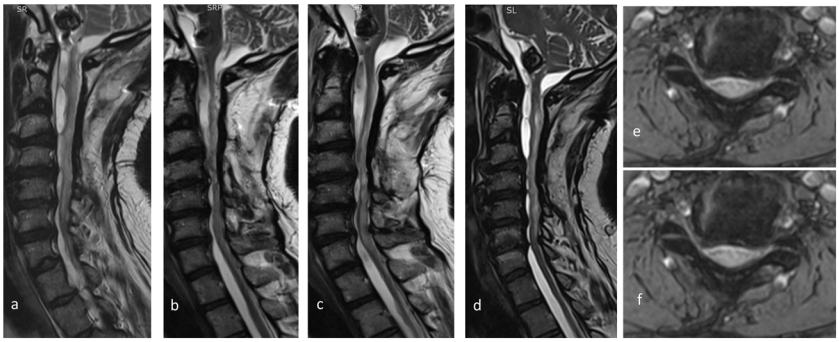

Figure 1 The MRI study showed cervical spondylotic myelopathy with disc degeneration in at least 5 intervertebral spaces, with hypertrophy and calcification of the posterior longitudinal ligament. These lesions caused stenosis of the cervical spinal canal. T2-weighted axial and sagittal sequences show the progression of the multiple arachnoid cysts and trabeculae from the craniocervical junction to the C7 level, with severe compression and atrophy of the spinal cord. Postoperative images obtained in a) 2014, b) 2015, c) 2016, d) 2018, e) 2014, and f) 2018. Gutiérrez et al.

The patient underwent surgery with posterior C2-C4 laminoplasty and removal of all the cysts identified in this region. The patient's gait temporarily improved after the procedure, but deteriorated again at 6 months despite rehabilitation treatment; he also developed proximal weakness in the upper limbs. We opted to perform a second surgical intervention, performing arachnoidolysis. Progression after this procedure was unfavourable, with reappearance of the arachnoid cysts in follow-up radiology studies and progressive loss of strength; the patient has only recovered partial motor function in the left arm.

Fig. 1 shows the patient's radiological progression.

Discussion

Intraspinal arachnoid cysts are a rare entity and remain undiagnosed in many cases.[2,3] They generally affect the thoracic spine,[3—5] with only 15% of cases affecting cervical segments.[6—8] The posterior intradural segment is clearly the most frequently affected, to the extent that anterior localisation has been described as exceptional.[5,9] Such specific localisation is probably related to long periods of rest in the supine position, which would favour blood stagnation in patients with thoracic kyphosis.[10]

These formations may be congenital, acquired, or idiopathic.[11] It has been suggested that the main aetiological mechanisms are such inflammatory processes as meningoencephalitis and post-haemorrhagic inflammation,[11] including cases of spontaneous haemorrhage of vascular (rupture of cavernomas and dural fistulae), post-traumatic, and postoperative origin.[2]

Although its aetiopathogenesis is not well understood, arachnoiditis is generally believed to cause fibrosis and thickening of the leptomeninges and local membrane adhesions, leading to mechanical and secretion mechanisms that contribute to the maintenance and growth of the cystic cavities[3,4,12]; when they reach a determined size, the compressive effect causes neurological symptoms.

The disease is diagnosed by spinal cord MRI. Differential diagnosis includes neurocysticercosis, neurenteric cysts, and epidural abscesses.[11]

In most case reports, as in our own, the treatment of choice is laminectomy and resection of the cysts.[6] Multiple resources are available for treating the cysts, including puncture and fenestration,[3] marsupialisation, atrial or peritoneal shunt, and a watchful waiting approach with clinical and radiological follow-up.

Prognosis is influenced by the degree of neurological involvement prior to surgery, progression time, cyst size,[13] and how early the procedure is performed. We must not rule out conservative treatment in these cases,[14] although the watchful waiting strategy is difficult to sustain in the long term,[15] particularly in cases of progressive impairment.

Conclusion

Post-haemorrhagic arachnoid cysts, like cysts of infectious origin, are a very rare complication; therefore, they should be suspected in patients presenting delayed neurological impairment after the underlying disease due to the risk of irreversible spinal cord lesions. Management almost always requires early surgical treatment, although given the low prevalence of the condition, the truly difficult decision is the moment and type of intervention.

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Lemierre syndrome with brain abscesses located in watershed regions. Clinical report☆

Síndrome de Lemierre con abscesos cerebrales distribuidos en territorios frontera vasculares. Caso clínico

Dear Editor:

Lemierre syndrome progresses with septic thrombophlebitis of the internal jugular vein with distant septic thromboemboli. It was first described by A. Lemierre nearly a century ago in patients with sepsis due to *Fusobacterium necrophorum*, mainly associated with pharyngeal infections.[1] The literature includes cases with a range of aetiological agents, foci of infection, and thrombus locations.[2,3] Brain abscesses caused by anaerobic bacteria generally occur as a result of disruption of anatomical barriers, with bacterial invasion from adjacent or distant tissues. In the case of non-clostridial bacteria, the barriers in question tend to be mucosal membranes, where they are present as saprophytic flora. The single most important factor facilitating the growth of anaerobic bacteria is low oxygen tension, usually secondary to reduced tissue perfusion, although other factors may also influence this process.[4]

Clinical case

We describe the case of a 79-year-old man with personal history of arterial hypertension, atrial fibrillation, aortic valve bioprosthesis implantation due to severe aortic insufficiency, and left parotid carcinoma treated with surgery and adjuvant radiotherapy at 68 years of age.

He presented left otalgia of 5 days' progression, associated with fever, nausea, and general discomfort. Outpatient treatment with amoxicillin and clavulanic acid was prescribed due to suspicion of acute otitis media. He was subsequently referred to the emergency department due to subacute onset of left hemiparesis and persistence of fever.

Laboratory tests showed leukocytosis (17 500 $\times$ 10^3 cells/μL; 94% neutrophils), C-reactive protein level of 176 mg/L (reference range, 0-5), and procalcitonin level of 1.1 ng/mL (reference range, 0-0.5). Emergency head and neck CT revealed signs of acute left otomastoiditis with destruction of the osseous septa, enlargement of pre- and retrostyloid soft tissue, and destruction of the bone of the occipital condyle and inferomedial region of the petrous bone. Contrast-enhanced imaging revealed lack of flow in the left jugular vein and in the internal carotid artery (ICA) after its bifurcation. Several intraparenchymal ring-enhancing lesions were observed in the left temporal

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