

LETTERS TO THE EDITOR

Collet–Sicard syndrome caused by metastasis[☆]

Síndrome de Collet–Sicard metastásico

Dear Editor:

It was with great interest that we read the case of Collet–Sicard syndrome (CSS) described by Gutiérrez Ríos et al.¹ and the explanation for the syndrome offered by these researchers. However, we were surprised to note that their literature search only yielded 51 cases of CSS published between 1915 and 2012. We would like to point out that the number of published cases has increased in recent years: a review of post-traumatic cases of CSS published in April 2015 identified 14 cases,² that is, 4 more cases of post-traumatic CSS than those identified by Gutiérrez Ríos et al. The low number of published cases may be explained by several reasons. The first is the rareness of the associated symptoms and the fact that the syndrome may be mistaken for other similar syndromes affecting nearby topographical locations. As Gutiérrez Ríos et al. state, there are many jugular foramen syndromes and they may exhibit gradual progression. This situation may result in different diagnoses in the same patient depending on the stage of disease progression. Another potential explanation for the low number of cases is the difficulty of diagnosing CSS when multiple cranial nerves are affected. In some cases, involvement of one nerve (for example, the vagus nerve) may mask the involvement of another (for example, the glossopharyngeal nerve); this is more likely to occur when patients are not examined by neurologists. And lastly, as in other diseases, many cases identified in our setting may have not been published. We offer the example of a previously unpublished case of CSS in a 90-year-old man who was attended at our hospital a year ago. Our patient had a history of arterial hypertension and type 2 diabetes mellitus, and an mRS score of 0 according to our records. He visited the emergency department on 2 occasions due to progressive dysphonia and dysphagia. He was initially diagnosed with left vocal cord paralysis. An outpatient follow-up study including a CT scan of the neck and chest was scheduled to rule out compression of the left recurrent laryngeal nerve. However, our patient returned to

the emergency department a few days after his first visit due to intense left-sided headache. On that occasion, he was assessed by neurologists who identified dysarthria and tongue deviation to the left side; all other general and neurological findings were normal. A simple cranial CT scan performed at the emergency department revealed no relevant findings and a chest radiography showed thickening of the left parahilar region. The patient was admitted for a more thorough study. During hospitalisation, his symptoms worsened: he presented marked weakness of the left sternocleidomastoid, deviation of the uvula to the right, left palatal paralysis, and abolished left gag reflex with no sympathetic involvement. All these findings pointed to CSS (involvement of the left IX, X, XI, and XII cranial nerves). A cranial MRI scan (Fig. 1A) revealed a lytic lesion with soft tissue mass in the left occipital condyle which suggested metastasis; the lesion was confirmed by a full-body CT scan (Fig. 1B). The CT scan also revealed a spiculated mass in the left infrahilar region measuring 55 mm (Fig. 2A) as well as a solitary pulmonary nodule ipsilateral to the spiculated mass and measuring 20 mm (Fig. 2B). Our patient displayed no symptoms of prostate cancer or apparent bone infiltration in the chest or vertebral column. Given our patient's advanced age and the wishes of his family, we ruled out aggressive treatment and opted for palliative care. The patient died a few days later after developing laryngeal stridor and acute respiratory failure. The physicians who last attended him did not request an autopsy.

In our view, this is a case of CSS caused by a metastatic tumour probably secondary to lung carcinoma; however, we lack anatomical pathology findings to support our hypothesis. The tumours most frequently causing skull-base metastasis are prostate and breast cancers; lung cancers are the fourth most common type (approximately 6% of all cases of skull-base metastasis).³ However, there is only one published case of CSS caused by metastasis of lung cancer (more specifically lung adenocarcinoma).⁴

Lastly, in patients showing involvement of several lower cranial nerves, differential diagnosis should aim to distinguish between carcinomatous meningitis (which is especially likely to affect these nerves in cases of basal arachnoiditis due to their caudal location) and a localised anomaly able to affect multiple nerves since they are very near to one another as they exit the base of the skull. The first diagnostic approach should aim to assess the anatomy of the impaired nerves to determine whether they are affected by a single topographic lesion. Once this step has been completed, we suggest delaying CSF tests until an accurate neuroimaging study of the area has been performed; MRI will be used in most cases of jugular foramen syndromes.³

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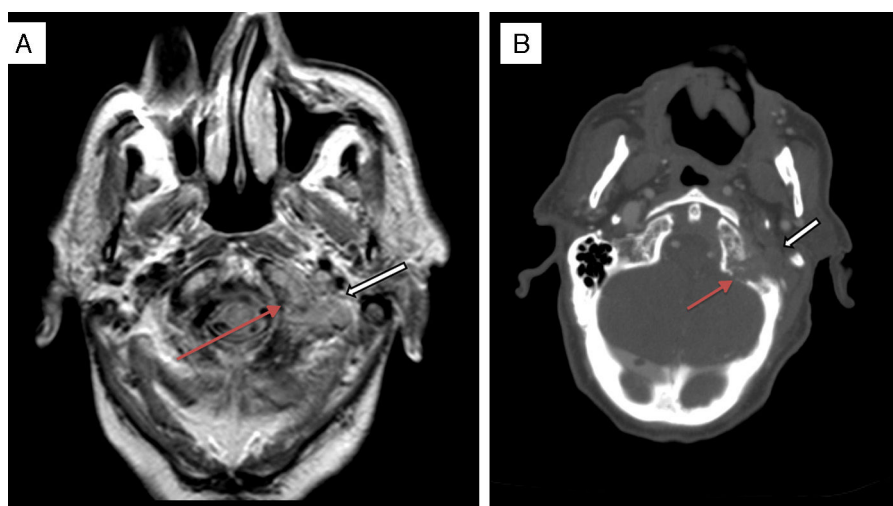


Figure 1 Axial proton density MRI scan (A) and axial cranial CT scan with intravenous contrast (B) showing an osteolytic lesion with bone destruction in the left condyle and left occipital tubercle (thin arrows) and a soft tissue mass (bold arrows) extending to both sides of the bone, invading the foramen magnum, and extending anteriorly to the tip of the odontoid process. The mass also occupies the jugular foramen and hypoglossal canal, and is in contact with the ipsilateral vertebral artery.

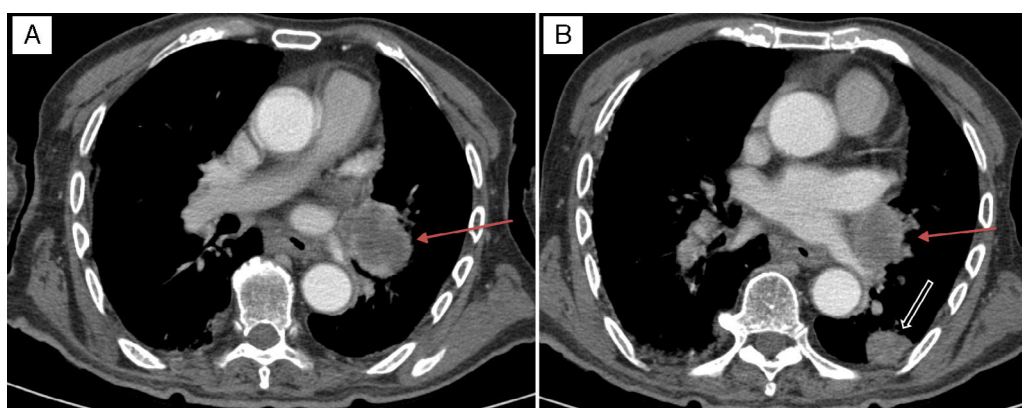


Figure 2 Axial CT scan of the chest with intravenous contrast. (A) Mass in the left infrahilar region with a maximum diameter of approximately 55 mm (arrow), with associated hypodense mediastinal adenopathy. (B) Pulmonary nodule measuring 20 mm located at the edge of the posterolateral segment of the left inferior lobule (thick arrow), probably linked to the satellite metastatic mass of the primary tumour located in the infrahilar region (thin arrow).

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Conflicts of interest

The authors have no conflicts of interest to declare.

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A. Sánchez-Larsen^{a,*}, I. Feria-Vilar^a, R. Collado^b, T. Segura^a

^a Servicio de Neurología, Complejo Hospitalario Universitario de Albacete, Albacete, Spain

^b Servicio de Radiodiagnóstico, Complejo Hospitalario Universitario de Albacete, Albacete, Spain

* Corresponding author.

E-mail address: aa.sanchezlarsen@gmail.com (A. Sánchez-Larsen).

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Orthostatic tremor secondary to recreational use of solvents[☆]



Temblores ortostático secundario al uso recreativo de disolventes

Dear Editor,

Organic solvents are volatile organic compounds used to dissolve raw materials or residual products. Their uses include cleaning agents, glues, paint components, plasticisers, and many other industrial purposes. Since these compounds are widely available, they may be used as psychostimulants; this tendency is more frequent in developing countries.

Our patient is a 30-year old former cocaine user. He had an 8-year history of bilateral optic neuritis with no known aetiology. He visited our emergency department due to frequent falls and gait disturbances which had evolved over 2 years to such an extent that he needed support to walk. Gait disturbances were initially attributed to probable epileptic myoclonic seizures (with normal EEG) and therefore treated with levetiracetam 500 mg/12 h. Treatment failed to control symptoms and provoked side effects which led the patient to stop taking the medication. Considering treatment failure in the context of the patient's age and degree of limitation, we decided to hospitalise him for further study.

Physical examination revealed that the patient was oriented in time and space, with no alterations in speech structures or content. Cranial nerves were unaffected. No motor or sensory alterations were observed in the limbs. He displayed upper limb tremor that was predominantly intention tremor with a postural component; it disappeared at rest and was compatible with tremor of cerebellar origin. Lower limbs showed orthostatic tremor in the proximal segment and a typical helicopter sign, which also provoked gait instability; the patient attempted to compensate with a wider stance. Tremor in the lower limbs was absent with the patient in the supine position or when seated. The patient also showed symmetrical hyperreflexia of the lower limbs.

Electroencephalography, electroneurography, and lumbar puncture procedures all yielded normal results. The electromyogram showed rhythmic muscle activity (tremor) at a frequency of 15 Hz.

Brain MRI revealed FLAIR sequence hyperintensity in both posterior limbs of the internal capsules, especially on the

anterolateral pons. The T2-weighted sequence also showed hyperintense lesions on both pyramidal tracts at the brainstem level and a hypointense lesion at the mesencephalic level in the red nucleus and substantia nigra (Figs. 1 and 2).

A few days after the patient had been admitted, a relative informed us that the patient had been inhaling gasoline fumes for several years (8 approximately) and strongly suspected that he was still doing so without being able to speculate about the frequency. Considering findings from the physical examination and the neuroimaging study, and the patient's history of inhaling gasoline fumes, we established a diagnosis of orthostatic tremor secondary to solvent inhalation (gasoline vapour). Starting clonazepam as symptomatic treatment of orthostatic tremor achieved excellent results. The patient was discharged a few days later. In subsequent follow-up visits, the patient indicated having stopped inhaling solvents but still needed clonazepam treatment to lessen symptoms of tremor.

We believe that the optic neuritis of unknown aetiology may also have been due to recreational use of organic solvents.

Discussion

Literature on central nervous system lesions caused by exposure to volatile organic compounds is scarce. The most widely studied substances are aromatic hydrocarbons (such as toluene) and other hexacarbon solvents, since they are readily available and present in household chemicals.¹

The typical lesions to the nervous system caused by exposure to volatile substances usually include peripheral neuropathy, optic neuritis, and neurosensory hearing loss. Nevertheless, white matter lesions typically follow long-term exposure to hydrocarbons, as shown by the many case studies reporting these findings.²

Lesions caused by long-term toluene exposure are visible in brain MRI, which may reveal white matter hyperintensities in T2-weighted sequences. These findings are more apparent in the posterior limbs of the internal capsules, cerebral peduncle, ventral pons, and middle cerebellar peduncles.³ Basal ganglia may appear hypointense.⁴ Cases of degeneration of the corpus callosum secondary to recreational use of solvents have been described in very recent studies.⁵ Cocaine consumption can also cause white matter lesions that are observable with brain MRI; however, these lesions differ from those secondary to toluene in that they are predominantly located in the frontal lobe, with the brainstem and cerebellum usually remaining intact.⁶

Symptoms of white matter involvement may include orthostatic tremor⁷ (seen in our patient), intention tremor, falling, ataxic gait, dysarthria, wide stance, instability, bal-

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