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https://doi.org/10.1016/j.nrleng.2018.04.006
2173-5808/

Intervertebral disc calcification as a cause of painful torticollis in children☆

Calcificación de discos intervertebrales como causa de tortícolis dolorosa en edad pediátrica

Dear Editor:

Acute torticollis is a frequent reason for consultation with paediatrics departments. The aetiology varies[1]; severe underlying diseases must therefore be ruled out.

Intervertebral disc calcification, an infrequent condition in paediatric patients, has been described as a cause of painful torticollis. We present the cases of 3 patients with torticollis secondary to intervertebral disc calcification attended at our centre, a tertiary hospital, over the past 20 years, and review the literature on this entity.

The patients were aged 5, 6, and 10 years old; the series includes 2 boys and one girl. All 3 patients presented sudden-onset torticollis and deviation of the head (to the right in 2 cases and to the left in 1). Only one of the patients had presented self-limited episodes previously (4 similar episodes in the previous 2 months, lasting 3—8 days). One patient had history of minor trauma. Time from symptom onset to diagnosis ranged from 5 to 10 days.

The physical examination revealed deviation of the head to the side of torticollis, shoulder asymmetry, elevation of the ipsilateral scapula, limited head extension, and mild contracture of the ipsilateral sternocleidomastoid and trapezius muscles. All 3 patients were in good general health, and neurological examination revealed no other pathological findings in any of them.

Cervical and thoracic spine radiography revealed signs of intervertebral disc calcification in all cases. In 2 patients, calcifications were observed in a single intervertebral space (C3-C4 and C2-C3), whereas the remaining patient showed calcifications at multiple locations (C4-C5, C7-T1, T2-T3, and T5-T6) (Fig. 1). Spinal cord involvement was not observed in any patient. Calcification was confirmed with MRI (Fig. 2). Two of the patients underwent a complete blood count; a biochemical study; tests for calcitonin, PTH, hydroxyproline, pyridinoline, and thyroid hormone levels; tests of calcium and phosphorus metabolism; erythrocyte sedimentation rate; renal function studies; and urine tests. All results were normal.

Patients received anti-inflammatory and analgesic treatment (non-steroidal anti-inflammatory drugs), and symptoms resolved after 5 to 35 days. Radiological progression varied. In one patient, calcification disappeared at 3 months, whereas another patient showed no changes at 4 months. The remaining patient underwent no follow-up radiology studies.

Intervertebral disc calcification is rare among the paediatric population. Beluffi et al.[2] conducted a retrospective study of radiographs taken over 26 years from patients aged 0 to 18 years, and identified intervertebral disc calcification in 6 patients; the condition was asymptomatic in one patient. Calcification may affect the intervertebral discs, vertebral bodies, muscles, or ligaments, causing cervical pain, sensorimotor alterations, or torticollis. The aetiology is unknown, but local inflammation of the nucleus pulposus is a possible explanation. Other possible causes include hypervitaminosis D, metabolic disorders, and haemolytic anaemia. On occasion, intervertebral disc calcification is an incidental finding.[2]

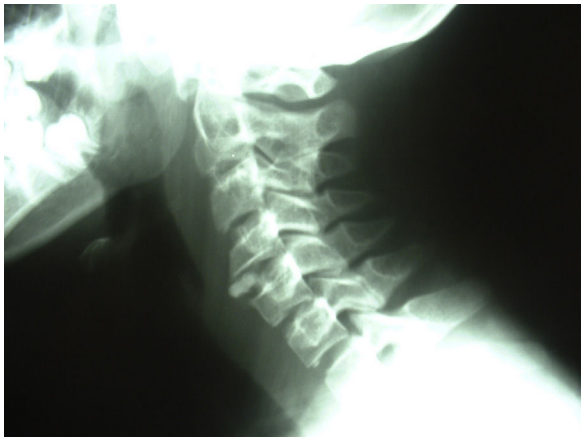

Figure 1 Cervical/thoracic spine radiography, lateral view. Calcifications in the nucleus pulposus of intervertebral discs C4-C5, C7-T1, T2-T3, and T5-T6. Enlarged intervertebral disc space at C4-C5, with greater soft tissue thickness at that level.

☆ Please cite this article as: Fernández Gómez A, Bernal Calmarza R, Monge Galindo L, Luis Peña Segura J. Calcificación de discos intervertebrales como causa de tortícolis dolorosa en edad pediátrica. Neurología. 2020;35:419—421.

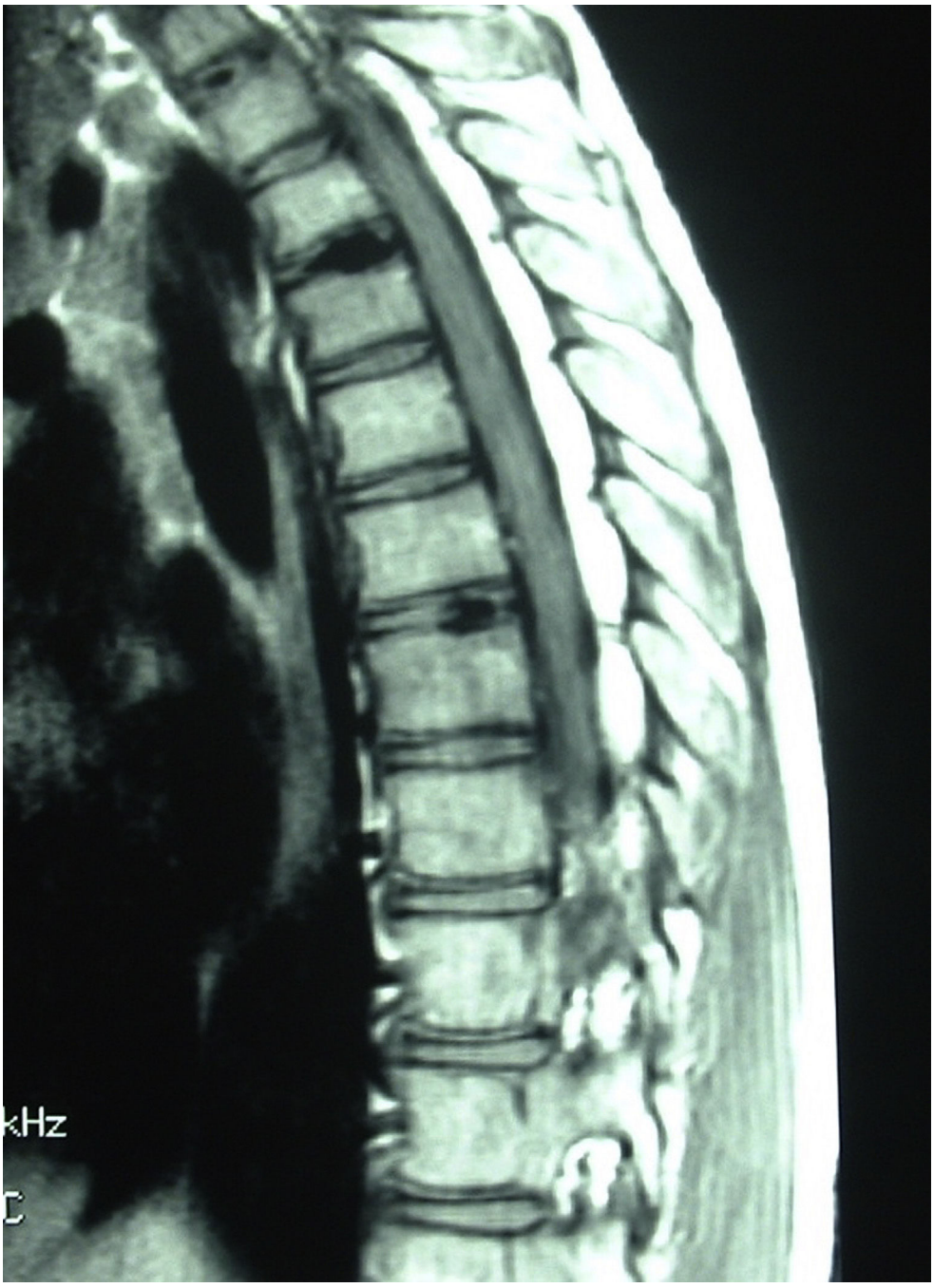

Figure 2 MRI scan of the cervical and thoracic spine, sagittal section. Calcifications in the nucleus pulposus of intervertebral discs C4-C5, T1-T2, T3-T4, and T6-T7.

Over 300 cases of intervertebral disc calcification have been reported since the condition was first described by Baron in 1924.[3] The condition is more frequent among children aged 6 to 10 years, and predominantly affects boys.[4,5] The cervical spine is involved in 70% of cases, and 35% of patients show calcification of more than one intervertebral disc space. The lumbar spine is the least frequently affected location. Cervical involvement is the most symptomatic, causing symptoms in 83% of cases (vs 24% in cases of lumbar involvement). The most frequent symptom is cervical pain, followed by sensorimotor alterations and fever,[6] particularly when calcifications have nearly resolved. The condition causes persistent or recurrent torticollis in 23% of cases.[3]

Diagnosis is established according to cervical radiography, CT, or MRI findings. Alterations in calcium and phosphorus metabolism should be ruled out. Most patients are treated conservatively with non-steroidal anti-inflammatory drugs and rest; a cervical collar may be used if symptoms persist. Surgery (laminectomy) is indicated if the patient presents neurological deficits or symptoms of spinal cord compression.[7,8] The condition usually resolves

spontaneously. In 70% of cases, pain resolves within a month, with calcifications usually disappearing between 28 days and 6 months after symptom onset.[9] Calcifications disappeared spontaneously in all 3 of our patients. Follow-up of these patients helps confirm spontaneous resolution of intervertebral disc calcification, which in turn confirms the diagnosis and rules out other conditions.

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29 January 2018

https://doi.org/10.1016/j.nrleng.2018.04.005
2173-5808/

Tapia syndrome following orotracheal intubation: a case report☆

Síndrome de Tapia tras intubación orotraqueal: a propósito de un caso

Dear Editor:

Tapia syndrome is defined as paralysis of one side of the tongue and the ipsilateral vocal cord, with preserved soft palate motility, secondary to concurrent lesions to the hypoglossal and vagus nerves (the twelfth and tenth cranial nerves, respectively).

Antonio García Tapia, a Spanish otorhinolaryngologist, first described the syndrome in 1904 in a patient with an upper neck injury from a bull's horn (''matador's disease'').[1] However, the eponym has been used in reference to a crossed syndrome of dorsal medullary origin (with involvement of the hypoglossal and ambiguus nuclei and the pyramidal tract),[2,3] and to describe peripheral involvement of the vagus and hypoglossal nerves at the cervical level.[3]

The peripheral form has been observed after manipulation of the airway for orotracheal intubation.

We present the case of a 70-year-old man with history of dilated cardiomyopathy secondary to alcohol abuse, who was admitted to our hospital due to cardiorespiratory arrest.

The cause of the attack was ventricular fibrillation; the patient required emergency life support for 15 minutes, with basic and advanced cardiopulmonary resuscitation. The attack occurred in a public space, and emergency intubation was performed before the patient was transferred to our hospital. Upon arrival at the emergency department, the patient was admitted to the coronary care unit, where an emergency coronography was performed and a hypothermia protocol was initiated (target temperature, 33 °C). The patient was kept under sedation during the first days after arrival at hospital, with no initial neurological assessment being performed at this time. Incidentally, a second orotracheal intubation procedure was required at 72 hours following obstruction of the first intubation tube.

As the patient continued to need mechanical ventilation a week after admission, a tracheostomy was performed to prevent airway lesions associated with orotracheal intubation. At this point, the patient continued to require sedation and parenteral feeding. Ten days after admission to the coronary care unit, the patient began to recover consciousness, spontaneously opening his eyes; brainstem reflexes were preserved, including the pupillary reflex, oculocephalic reflexes, and the cough reflex. Mechanical ventilation was suspended at 2 weeks and a silver tracheostomy tube was placed.

☆ Please cite this article as: Silva-Hernández L, Gil Rojo C, González García N, Porta-Etessam J. Síndrome de Tapia tras intubación orotraqueal: a propósito de un caso. Neurología. 2020;35:421—423.