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Abdominal Pseudohernia After Spinal Cord Injury: Report of Three Cases☆,☆☆

Seudohernia abdominal tras lesión medular. A propósito de 3 casos

An abdominal pseudohernia is a protrusion of the abdominal wall that resembles a hernia, but differs from a true hernia in that there is no real muscle disruption and all muscle layers remain intact.[1] Pseudohernias are rare conditions that are caused by atrophy of the abdominal musculature. They have been described in association with neuropathy sultrasoundndary to medical causes[2,3] or post-lumbotomy.[4] Pseudohernias resulting from medical causes are resolved partially or totally,[2,3] while those sultrasoundndary to a surgical complication do not improve.[4] To date, they have not been described after spinal cord injury. The objective of this paper is to reported the cases of 3 patients who developed abdominal pseudohernias after spinal cord injury. These patients were identified in the outpatient clinic of the spinal cord injury unit from 2009 to 2015. At a follow-up visit, each had reported diffuse symptoms, including unilateral abdominal heaviness, lumbar discomfort or imbalance while sitting, but no clear limitations for activities of daily living. Two of these patients had been referred by their family doctor to the general surgery department with the diagnosis of abdominal hernia.

The patients began reporting symptoms 4–6 months after the spinal cord injury. The 3 patients were males between the ages of 45 and 57. In 2 cases, the spinal cord injury was caused by a trauma injury (D12-L1 fractures), and in the third case the origin of the spinal cord injury was medullary ischemia affecting the lumbosacral spinal cord segments and produced an incomplete medullary lesion. Clinical examination revealed abdominal asymmetry (Fig. 1). The cranial magnetic resonance imaging (MRI) study of the vertebral-medullary trauma causing the spinal cord injury was reviewed, and an abdominal ultrasound, electromyogram of the abdominal wall and measurement of the abdominal wall thickness by ultrasound were requested, as well as a new spinal MRI if there had been surgery after the spinal cord injury (Table 1). In

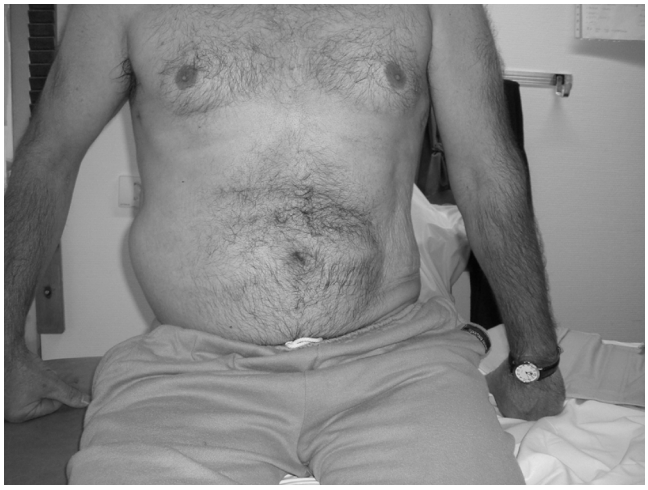

Fig. 1 – Right abdominal pseudohernia after asymmetrical D12 spinal cord injury.

the 3 cases, there was unilateral involvement of the L1 root, which caused unilateral thinning of the abdominal wall. The patients, who have continued in follow-up in the outpatient clinic of the ULM, have continued to present the same clinical symptoms and examination results for 2–8 years. Described by Taylor in 1895,[5] abdominal pseudohernia has usually been described in various studies as a medical complication of a herpes zoster infection[2,6,7] or diabetic radiculopathy.[3,8,9] Likewise, it has also been described after surgery,[4] invasive diagnostic procedures[10] or rib fractures. Unlike the cases described in the literature where the neurological lesion occurred in the peripheral nerve, the cases presented in this study relate the origin of the pseudohernia with a unilateral lesion of the anterior horn of the spinal cord.

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Table 1 – Type of Injury and Complementary Tests.

	Age	Spinal cord diagnosis	Initial MRI	Surgery	EMG, abdominal wall	Ultrasound, abdominal wall	Ultrasound abdomen	Later MRI
Patient 1	60	Incomplete SCI D12, ASIA C	L1 fracture with spinal cord compression	Arthrodesis D11-L2	Major oblique neurogenic atrophy of right abdomen	Left abdominal wall thickness 1.85 cm; right 1.60 cm	Ultrasound with no pathologic findings	Arthrodesis D11-L2, with no alterations in the vertebral canal
Patient 2	45	Complete SCI D12, ASIA A	D12-L1 fracture with spinal compression	Arthrodesis D10-L2	Denervation in left oblique and rectus	Left abdominal wall thickness 1.80 cm; right 1.88 cm	Ultrasound with no pathologic findings	Reduction of canal, correct situation of screws in pedicles
Patient 3	55	Incomplete SCI D12, ASIA C	Distal spinal cord infarction (6 cm)	No surgery	Chronic neurogenic pattern in right D12 root; atrophy of major oblique and rectus	Left abdominal wall thickness 1.98 cm; right 1.85 cm	Ultrasound with no pathologic findings	Lumbar spondylosis

ASIA: American Spinal Injury Association; D: dorsal; SCI: spinal cord injury; MRI: magnetic resonance imaging.

On examination, the 3 patients presented better sensory preservation at D12 (residual sensitivity) on one side; 2 were incomplete spinal cord injuries and another was complete. For the differential diagnosis, abdominal hernia was ruled with abdominal ultrasound, which confirmed the integrity of the abdominal wall and the normal visceral location of its contents. Likewise, in the 3 ultrasounds of the abdominal wall, a reduction in the thickness of the abdominal wall was observed, which in the 3 cases has occurred at the expense of the outermost muscular layer (greater oblique dependent on the D12 root), with greater preservation of the thickness of the minor oblique and the transverse muscle (L1). Posterior MRI of the spinal column ruled out any associated injuries. The authors suggest that the 3 patients presented asymmetric medullary lesions that would affect the anterior root to the union of the first and sultrasoundnd motor neurons on one side of the cord, with preservation of the contralateral side at that level, which would result in atrophy of the musculature of the affected hemiabdomen. Although the cases presented correspond to very rare spinal cord injuries (spinal cord injury at D12 on one side, with preservation on the contralateral side), similar cases must have undoubtedly presented previously on numerous occasions. However, according to the authors' review of the literature, no reports have been published to date. This may possibly be due to the fact that, among other things, this complication scarcely produces remarkable symptomatology within the context of the spinal cord injury itself and does not usually require treatment. Abdominal pseudohernia is a lesion that may appear after asymmetric spinal cord injury at D12. Although the symptoms that it causes are minimal and do not usually require treatment, this pathology should be included in the differential diagnosis of abdominal hernias.

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Acral Ischemia Secondary to Pheochromocytoma☆

Isquemia acral secundaria a feocromocitoma

Catecholamine-producing tumors are uncommon and called pheochromocytomas when their origin is intra-adrenal and paragangliomas when they are extra-adrenal.[1] The symptoms of pheochromocytoma include maintained or paroxysmal hypertension, headaches, palpitations and sweating. However, its symptoms can vary greatly, which is why it has been called "the great simulator".[1] Pheochromocytoma is rarely a cause of peripheral ischemia, but when this occurs it can lead to necrosis or gangrene, due in most cases to extreme vasoconstriction or diffuse arterial vasospasms induced by hypercatecholaminemia.[1–10] This vasospasm induced by catecholamines can easily be overlooked if the patient does not present other symptoms characteristic of pheochromocytoma; some patients may even have a history of intermittent claudication, a situation that can hinder and delay diagnosis.[1–10]

We report the case of a patient with a pheochromocytoma that presented multiple clinical manifestations and severe ischemic involvement of both the upper and lower limbs.

The patient is a 76-year-old woman with a history of hypertension under treatment for many years, ischemic stroke 20 years earlier with recovery and minimal sequelae, and gastric surgery for a bleeding ulcer 10 years ago. In the emergency room, she reported persistent intense headache for the previous 5 days associated with dizziness, nausea and vomiting. At this time, she was diagnosed with a hypertensive crisis, which subsided with medical treatment. She returned 24 h later and reported the same symptoms as before, in addition to epigastralgia radiating toward the thorax and diaphoresis. Upon examination, blood pressure was 185/90 mmHg, electrocardiogram showed a decrease in ST and negative T waves, and lab work-up detected elevated myocardial ischemia enzymes, so the patient was diagnosed with hypertensive crisis and acute coronary syndrome. 36 h after admission, the patient presented neurological deterioration with a Glasgow index of 12 and subsequent acute renal failure and respiratory failure, so she was transferred to the intensive care unit and intubated. Abdominal computed tomography (CT) scan showed a right adrenal mass measuring 8 cm in diameter; urine analysis demonstrated increased catecholamines (metanephrine 857 μg/24 h [Nr: 60–350]; noradrenaline 99 μg/24 h [Nr: 12–86]; adrenaline 45 μg/24 h [Nr: 2–23]). Blood pressure was controlled with iv labetalol during the 24 h of intubation, after which the treatment was changed to phenoxybenzamine and oral antihypertensive drugs. The patient presented an episode of atrial fibrillation that reverted to sinus rhythm with amiodarone. Renal failure and encephalopathy progressively improved.

During the hospital stay, the patient presented progressive acral ischemia of the 4 limbs, with necrosis of the fingers and toes (Fig. 1). Treatment was initiated with acetylsalicylic acid and perfusion of buflomedil. One month after admission to the hospital, right adrenalectomy was performed, initially by laparoscopy but then requiring conversion to subcostal laparotomy due adhesions caused by previous gastric surgery. The patient presented a favorable postoperative period, with progressive remission of the symptoms. The ischemic lesions of the 4 limbs became localized, and 53 days after admission, the patient required bilateral supramalleolar subcondylar amputation and metacarpophalangeal amputations of the fingers of both hands and interphalangeal amputations in the 2 thumbs. The patient recovered adequately. Vasculopathy and peripheral tissue ischemia are exceptional manifestations of pheochromocytoma and are due to the intense vasoconstriction of the cutaneous vessels in response to sympathetic overstimulation.[1,2] Early diagnosis of these cases is important to avoid vasoconstrictive medication, which may worsen the distal ischemia of both the upper and lower extremities.[3–8]

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