

decided to administer conservative treatment due to the patient's poor prognosis. A genetic study of lissencephaly and congenital disorders of glycosylation yielded negative results. At 7 months of life, the patient was brought to hospital due to fever and decompensation of the underlying liver disease; despite medical treatment, she died due to cardiorespiratory arrest. A post-mortem liver biopsy confirmed the diagnosis of EBA. This is the first case of LCH associated with EBA to be reported in the literature. To date, no study has described mutations linking LCH and EBA.

Funding

This study has received no funding of any kind.

References

1. Barkovich AJ, Guerrini R, Kuzniecky RI, Jackson GD, Dobyns WB. A developmental and genetic classification for malformations of cortical development: update 2012. *Brain*. 2012;135:1348–69.
2. Kato M. Genotype-phenotype correlation in neuronal migration disorders and cortical dysplasias. *Front Neurosci*. 2015;9:181.
3. Gerald MD. In: Martinez MA, editor. *Pediatric Neurology: A signs and symptoms approach*. Ed Elsevier España S. L; 2010.
4. Kumar RA, Pilz DT, Babatz DT, Cushion TD, Harvey K, Topf M, et al. TUBA1A mutations cause wide spectrum lissencephaly (smooth brain) and suggest that multiple neuronal migration pathways converge on alpha tubulins. *Hum Mol Genet*. 2010;19:2817–27.
5. Forman MS, Squier W, Dobyns WB, Golden JA. Genotypically defined lissencephalies show distinct pathologies. *J Neuropathol Exp Neurol*. 2005;64:847–57.
6. Spalice A, Parisi P, Nicita F, Pizzardi G, Del Balzo F, Iannetti P. Neuronal migration disorders: clinical, neuroradiologic and genetics aspects. *Acta Paediatr*. 2009;98:421–33.
7. Schwartz KB, Harber BH, Rosenthal P, Mack CL, Moore J, Bove KE, et al. Extra-hepatic anomalies in infants with biliary atresia: results of a large prospective North American multi-center study. *Hepatology*. 2013;58:1724–31.
8. Rasmussen SA, Olney RS, Holmes LB, Lin AE, Keppler-Noreuil KM, Moore CA. National Birth Defects Prevention Study. Guidelines for case classification for the National Birth Defects Prevention Study. *Birth Defects Res (Part A) Clin Mol Teratol*. 2003;67:193–201.

I. del Castillo Velilla*, M.D. Martínez Jiménez,
M. Pascual Martín, M.Á. García Cabezas

Servicio de Pediatría, Hospital General Universitario de Ciudad Real, Ciudad Real, Spain

* Corresponding author.

E-mail address: ndelcastillo6@gmail.com

(I. del Castillo Velilla).

11 December 2018

<https://doi.org/10.1016/j.nrleng.2020.07.001>
2173-5808/

© 2019 Sociedad Española de Neurología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Leriche syndrome as a rare cause of cauda equina syndrome*



Síndrome de Leriche como causa inhabitual del síndrome de la cola de caballo

Dear Editor:

Cauda equina syndrome is caused by lesions to the nerve roots emerging from below the conus medullaris (lumbar, sacral, and coccygeal nerve roots). It generally manifests as low back pain irradiating to the gluteus muscles, weakness of the lower limbs, saddle anaesthesia, and sexual and sphincter dysfunction. Cauda equina syndrome is an infrequent entity but requires urgent aetiological diagnosis and early treatment in order to minimise the potentially severe and irreversible sequelae.

We present the case of a 72-year-old man, a former smoker, with a diagnosis of arterial hypertension and

atrial fibrillation treated with Sintrom® and antihypertensive drugs. The patient presented sudden weakness and hypoaesthesia of the lower limbs, accompanied by severe bilateral pain in the gluteus muscles, irradiating to both legs. He reported no history of intermittent claudication or changes in skin colour or temperature in the distal region of the lower limbs.

Neurological examination showed weakness of the lower limbs (hip flexion 3/5, knee flexion 3/5, knee extension 4/5, dorsiflexion and plantar flexion 0/5) and no weakness in the upper limbs. He presented tactile hypoaesthesia of the dorsal region of the feet and lateral region of the legs (L5), the external face of the thighs (L2–L3), and groin area (L1–L2); apallaeesthesia up to the knees; and abolished positional sensitivity in the toes. The right Achilles and patellar reflexes were abolished, whereas the remaining stretch reflexes were normal. The plantar reflex was abolished bilaterally. The abdominal and cremasteric reflexes were absent on both sides. In conclusion, neurological examination findings were compatible with cauda equina syndrome. Furthermore, the peripheral pulses were not palpable.

Blood analysis revealed mild thrombocytopaenia, an International Normalised Ratio of 1.4, and prothrombin activity of 60%. An emergency lumbosacral spinal CT scan revealed no spinal cord anomalies or signs of haemorrhage. An MRI scan of the thoracolumbosacral spine detected no haemorrhages, spinal signal alterations, or cauda equina

* Please cite this article as: Casas E, Vázquez F, Witek NP. Síndrome de Leriche como causa inhabitual del síndrome de la cola de caballo. *Neurología*. 2020;35:503–504.



Figure 1 Aortic CT angiography. Occlusion of the abdominal aorta and both iliac arteries.

compression. However, the image showed aortic wall irregularities, continuing throughout both common iliac arteries. Diagnosis of Leriche syndrome was confirmed with a CT angiography, which revealed occlusion of the inferior mesenteric artery, infrarenal abdominal aorta, and both common iliac arteries (Fig. 1).

The patient underwent an emergency bilateral aortoiliac thrombectomy and a stent was placed in the left iliac artery. Peripheral pulses became palpable and the patient progressed favourably; anticoagulant treatment was maintained and antiplatelet treatment started.

At 2 months, strength and tactile sensitivity were completely normal. The patient presented distal apallæsthesia (up to the malleoli) and recovered the abdominal and cremasteric reflexes bilaterally, with only abolished Achilles reflexes persisting. A motor nerve conduction study showed sensorimotor anomalies in the affected nerve roots.

The most frequent aetiology of cauda equina syndrome is compression (hernias, tumours, cysts, aneurysms, haemorrhages, etc.),¹ which should be ruled out in the first instance. Ischaemic aetiology is less frequent, with embolisms being the main cause within this group.

This case is of particular interest as Leriche syndrome is a very infrequent cause of cauda equina syndrome. We reviewed the literature and found only one another reported case.² When Leriche syndrome is accompanied by paraparesis, it is typically explained by spinal cord ischaemia, and not by damage to the nerve roots. Furthermore, onset of acute paraparesis represents a diagnostic challenge, as Leriche syndrome generally causes intermittent claudication with a progressive course. In case of paraparesis or sensory symptoms, a vascular origin should be suspected and peripheral pulses should always be examined.³

Finally, onset of paraparesis without anomalies in imaging studies may suggest cauda equina syndrome as the cause. Contrast-enhanced imaging studies assessing vascular damage may be very useful, as this rare but incapacitating disease requires early diagnosis and urgent treatment, with the aim of minimising possible sequelae.

Conflicts of interest

The authors have no conflicts of interest to declare. This study has received no funding of any kind.

References

1. Dias ALN, Araújo FF, Cristante AF, Marcon RM, Barros Filho TEP, Letaif OB. Epidemiology of cauda equina syndrome. What changed until 2015. *Rev Bras Ortop.* 2017;53:107–12.
2. Groth M, Much C, Fiehler J. Manifestation of acute Leriche syndrome as cauda equina syndrome. *Rofo.* 2013;185:375–6.
3. Irizarry L, Wray A, Guishard K. Acute paraplegia as a presentation of aortic saddle embolism. *Case Rep Emerg Med.* 2016, <http://dx.doi.org/10.1155/2016/1250153>.

E. Casas^{a,*}, F. Vázquez^b, N.P. Witek^c

^a *Servicio de Neurología, Hospital Central de la Cruz Roja San José y Santa Adela, Madrid, Spain*

^b *Servicio de Neurología, Hospital Universitario de Burgos, Burgos, Spain*

^c *Parkinson's Disease and Movement Disorders Program, Rush University Medical Center, Chicago, IL, United States*

* Corresponding author.

E-mail address: elena.caspe@gmail.com (E. Casas).

<https://doi.org/10.1016/j.nrleng.2018.12.010>
2173-5808/

© 2019 Sociedad Española de Neurología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).