CASO CLÍNICO/CASE REPORT

The Spondylotic Man in a Barrel

O Homem no Barril Espondilótico

Marco Almeida [1,*], Fernando Matias [1]

1-Department of Neurology Hospitais da Universidade de Coimbra, ULS Coimbra, Coimbra Portugal

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***Autor Correspondente / Corresponding Author:**
Marco Almeida
Department of Neurology
Hospitais da Universidade de Coimbra,
Praceta Professor Mota Pinto, Celas,
3004-561 Coimbra, Portugal
marcojosealmeida@gmail.com

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Abstract

Man-in-a-barrel syndrome (MBS) is a rare clinical condition in which patients present with bilateral brachial diplegia, with relatively spared strength in the lower limbs. This syndrome is typically attributed to hypoperfusion brain injury rather than spinal pathology. We describe the case of a 75-year-old man who presented with bilateral upper limb paresis. He had had two cardiorespiratory arrests with no documented neurological sequelae a few years before. Imaging tests however showed severe cervical spondylotic myelopathy as the most likely cause. He was not accepted for surgery given the high surgical risk. Despite its rarity, treatable causes of man-in-a barrel syndrome such as cervical compressive myelopathy must be excluded.

Resumo

A síndrome do homem no barril é uma condição clínica rara no qual a apresentação é marcada por diplegia braquial com força muscular relativamente preservada nos membros inferiores. Esta síndrome é tipicamente atribuído a lesões cerebrais por hipoperfusão e menos frequentemente a patologia medular. Descrevemos o caso de um homem de 75 anos com parésia dos membros superiores. Tinha antecedentes de duas paragens cardiorrespiratórias anos antes, sem sequelas neurológicas documentadas. Os exames de imagem revelaram uma mielopatia espondilótica cervical severa como a causa mais provável. Não foi aceite para cirurgia dado o elevado risco cirúrgico. Apesar da sua raridade, causas tratáveis da síndrome do homem no barril tal como a mielopatia compressiva cervical têm de ser excluídas.

Introduction

The man-in-a-barrel syndrome, or "person-in-a-barrel" as it was recently suggested in the literature,[1] is a rare neurological syndrome that translates bilateral weakness of the upper limbs with preservation of lower limbs strength. The syndrome takes its name from the typical posture of a man trapped inside a barrel.

This syndrome can have several etiologies: bilateral hemispheric lesions, brainstem lesions, cervical and brachial bilateral plexus lesions. The most frequent cause is cerebral hypoperfusion resulting in bilateral watershed strokes, namely cardiac arrest. Brainstem lesions affecting pyramidal fibers may also present with this phenotype. Intrinsic or compressive causes of the cervical spine can mimic this syndrome and must be mandatorily excluded. Bilateral brachial plexus injuries are rare and usually have a traumatic cause.[2]

Case Report

We describe the case of a 75-year-old male who consulted the emergency department for chronic upper limb muscle weakness. His medical and surgical history included: mechanical aortic valve replacement together with an ascending aorta complicated by two perioperative respiratory arrests ten years before, severe asthma, arterial hypertension, type 2 diabetes mellitus, atrial flutter and moderate vascular dementia. He was medicated with bisoprolol 5 mg id, warfarin 5 mg id, montelukast 10 mg id, metformine 500 mg id and lisinopril 20 mg id.

He had a fall from a tree two years earlier with direct neck trauma, without an established hyperextension mechanism. He consulted the emergency department at that time and performed brain, cervical and dorsal computed tomography (CT) without traumatic sequelae. Two months later he describes the onset of symmetrical proximal brachial muscle weakness that slowly worsened over time. Despite the aggravation, he did not consult the health services. He was not able to drive anymore and had a lot of difficulties with eating as he was unable to carry food to his mouth. He was also unable to complete tasks requiring fine motor coordination. Symptoms were constant throughout the day and were associated with neck pain and bilateral proximal brachial pain without irradiation. He denied loss of strength in the lower limbs, loss of sphincter control and sensory complaints. He also denied pain radiating to the upper or lower limbs and electric shock-like sensations on cervical flexion.

Upon inspection, he had global muscular atrophy of the upper limbs, especially evident at the deltoids. The scapular muscles were similarly atrophied bilaterally. He had significant atrophy of the interosseous muscles bilaterally (**Fig. 1**). In the lower limbs, there was a slight atrophy of the quadriceps muscles. The neck was short and an accentuation of the cervical lordosis was notorious. Strength was classified according to the Medical Research Council (MRC) Scale and is presented in **Table 1**. The bicipital and styloradial reflexes were abolished bi-

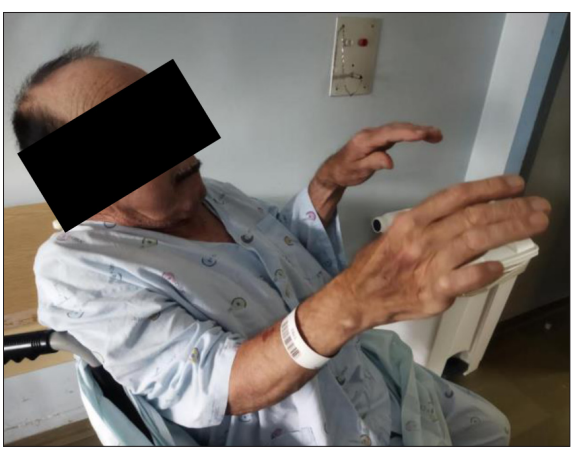


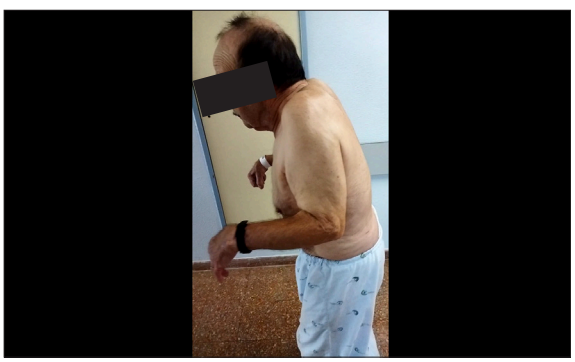
Figure 1. Patient trying to perform shoulder abduction movements with marked difficulty against gravity. Atrophy of the interosseous muscles is notorious.

Table 1. Patient's muscle strength according to the MRC scale by segments.

Movement tested	MRCScale (Right side)	MRCScale (Left side)
Cervical flexion	5	5
Cervical extension	5	5
Shoulder abduction	2	2
Shoulder adduction	2	2
Forearm extension	4	4
Forearm flexion	4	4
Hand flexion	4	4
Hand extension	4	4
Hip flexion	4	4
Hip extension	4	4
Leg flexion	4+	4+
Leg extension	4+	4+
Foot dorsiflexion	4+	4+
Foot plantar flexion	5	5
Hallux extension	5	4+

MRC - Medical Research Council.

laterally. The right triceps reflex was abolished and the left one was 1+. Patellar reflexes were 2+ bilaterally and Achilles reflexes were 1+ bilaterally. Cutaneoplantar reflexes were in extension bilaterally. Upper limb hypotonia was remarkable, as well as lower limb spasticity. The positional sense in the upper limbs was affected bilaterally (more than 3 errors out of 20). We provide a video of the patient walking while being instructed to perform shoulder abduction (**Video 1**).



Video 1. Video of the patient walking while being instructed to perform shoulder abduction. (see the video)

The cranial nerve examination was normal. There were no tongue fasciculations. Spurling's test and Lhermitte's sign were negative. Vibration sense, pinprick sensation, and two-point discrimination could not be reliably tested due to difficulty understanding the test. Light touch did not reveal any abnormalities, nor did positional sense in the lower limbs.

Blood count, ionogram, creatinine, transaminases and CRP were unremarkable. Cerebral CT scan (**Fig. 2**) showed global atrophic enlargement of the CSF pathways, in association with diffuse cortical-subcortical

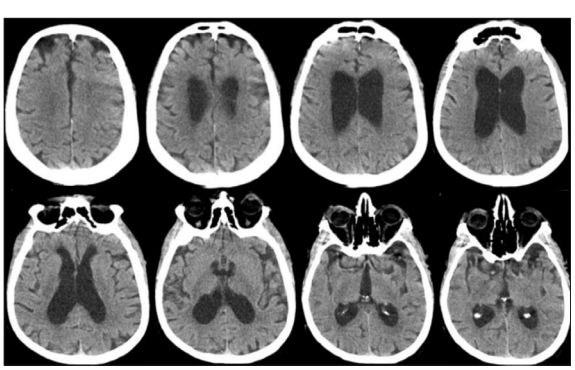


Figure 2. Head CT-Scan, serial axial planes (left to right sequence) - Global cortical atrophy without images suggestive of old cerebral infarcts in watershed areas.

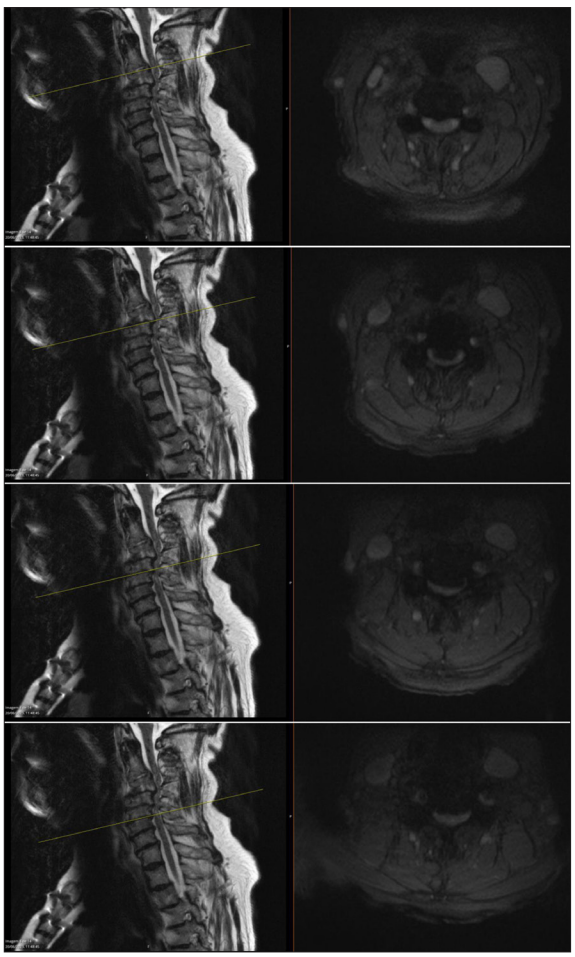


Figure 3. Axial and sagittal planes of cervical MRI, T2 sequence. Compressive spondylotic myelopathy was observed, particularly evident from C3-C6 with a central hypersignal.

volumetric involution. No acute or subacute ischemic lesions were identified. A cervical magnetic resonance imaging (MRI) was performed, which revealed severe compressive spondylotic myelopathy from C3 to C6 (**Fig. 3**). This examination was performed under sedation due to the patient's intolerance. Despite its diagnostic value, given the patient's history of cardiorespiratory arrest, the anesthesiology team deemed it unsafe to prolong sedation to obtain a brain MRI, so we were unable to proceed with the imaging. Gadolinium was not administered due to allergy in the previous MRI. EMG showed signs of bilateral severe and chronic cervical radicular lesion at C5-C7 and signs of moderate chronic radicular lesion at C8 (**Table 2**). Nerve conduction studies performed in the upper and lower limbs were normal. He started an in-hospital physiotherapy program which he carried out with motivation.

Table 2. Electromyography results.

Muscle	Side	Spontaneous activity					Voluntary activity			Recruitment
		IA	FP	PSW	Fasciculation	Polyphasia	MUAP Amp.	MUAP Config.	MUAP duration	
Triceps brachii	R	N	Absent	Absent	Absent	Absent	N	N	+++	Reduced
	L	N	Absent	Absent	Absent	Absent	N	N	+++	Reduced
Deltoideus	R	N	Absent	Absent	Absent	Absent	N	N	+++	Reduced
	L	N	Absent	Absent	Absent	Absent	N	N	+++	Reduced
Biceps brachii	R	N	Absent	Absent	Absent	Absent	N	N	+++	Reduced
	L	N	Absent	Absent	Absent	Absent	N	N	+++	Reduced
1st dorsal interossei	R	N	Absent	Absent	Absent	Absent	N	N	+	Simple
	L	N	Absent	Absent	Absent	Absent	N	N	+	Simple
Extensor indicis proprius	R	N	Absent	Absent	Absent	Absent	N	N	+	Simple
	L	N	Absent	Absent	Absent	Absent	N	N	+	Simple
Tensor fasciae latae	R	N	Absent	Absent	Absent	Absent	N	N	N	N
	L	N	Absent	Absent	Absent	Absent	N	N	N	N
Anterior tibialis	R	N	Absent	Absent	Absent	Absent	N	N	N	N
	L	N	Absent	Absent	Absent	Absent	N	N	N	N
Gastrocnemius (medial head)	R	N	Absent	Absent	Absent	Absent	N	N	N	N
	L	N	Absent	Absent	Absent	Absent	N	N	N	N

Config., configuration; IA, insertional activity; FP, fibrillation potentials; L, Left; MUAP, motor unit action potential; N, normal; PSW, positive Sharp waves; R, Right.

Surgery was considered based on a multidisciplinary assessment involving the Neurology, Neurosurgery, and Anesthesiology teams. The surgical risk was estimated based on the patient's personal history. From a neurological perspective, progression to tetraplegia is a risk given the expected natural progression of the cervical spinal cord. The advanced vascular dementia that the patient presents was also taken into account. Since the patient was classified by the American Society of Anesthesiologists Classification as grade IV, the surgical risk was considered unacceptable. The patient continued to undergo physiotherapy in a hospital environment with muscle strengthening exercises and was discharged to a rehabilitation facility.

Discussion

Our patient presented with man-in-a-barrel syndrome, a rare condition with an atypical cause in this case. While this syndrome is most commonly associated with cerebral hypoperfusion following cardiac arrest, leading to border zone infarctions between the territories of the anterior and middle cerebral arteries, compressive or ischemic causes at the cervical level are rarely reported.[2,3]

The patient had a history of two previous cardiorespiratory arrests. However, these events occurred long before the onset of symptoms, making it difficult to establish a direct correlation with the current clinical presentation. Notably, the patient exhibited signs of lower motor neuron involvement in the upper limbs that would not be explained by this etiology. Furthermore, the anamnesis revealed a traumatic event, followed by the gradual onset of upper limb weakness two months later.

The physical examination findings, including cervical pain, impaired positional sense, and bilateral signs of lower motor neuron involvement, led us to consider cervical spondylotic myeloradiculopathy as the most likely diagnosis. The presence of pyramidal signs in the lower limbs, coupled with questionable sensory testing, also raised the possibility of a motor neuron disease such as amyotrophic lateral sclerosis. However, this was ruled out as the EMG did not reveal acute signs of denervation.

Brain CT and cervical MRI findings confirmed the presence of severe cervical spondylosis, characterized by degenerative changes in the intervertebral discs and cervical spinal elements. Cervical spondylosis often results in instability of the cervical spine and progressive spinal canal stenosis. Trauma can exacerbate this condi-

tion by causing instability of cervical structures, including ligaments, uncovertebral joints, and facet joints.[4,5] In this case, cervical spondylosis primarily manifested as bilateral polyradicular compression and spinal cord compression affecting the cuneiform fascicles. Deformation of the pyramidal tracts was also observed, complicating the assessment of the spinothalamic tracts due to the patient's cognitive limitations.

Conclusion

The approach to a man-in-a-barrel syndrome must be systematic, and a cervical compressive myelopathy must be excluded in the emergency department, as the treatable entity that it is. Our case shows how atypical the presentation of this entity can be, the main etiologies being few in number but important to be known by the neurologist. ■

Contributorship Statement / Declaração de Contribuição

MA: Conception and design. Writing of the manuscript and data interpretation. Final approval.

FM: Conception and design. Critical review of the manuscript; Final approval.

All authors approved the final version to be published.

MA: Conceção e desenho. Redação do manuscrito e interpretação dos dados. Aprovação final.

FM: Conceção e desenho do estudo. Revisão crítica do manuscrito; Aprovação final.

Todos os autores aprovaram a versão final a ser publicada.

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