ABSTRACT

The association between motor tics and cervical myelopathy is rare and not well understood. Only a few papers in the literature reported this disorder until the present date. This is a case report of a cervical myelopathy case secondary to a motor tic disorder. A 23-year-old male with a 10-year history of motor tic disorder, involving sudden forced extension of the head and cervical spine. Disturbed tactile sensation and kinetic posturing that progressed to the Lhermitte sign every time he made the movement were detected over the last six months. Magnetic resonance imaging (MRI) showed hyperintense intramedullary lesion at C2-C3, degeneration at C3-C4, and no signs of spinal cord compression. On sagittal view, functional MRI with head extension showed anterior compression with protrusion of the intervertebral disc and posterior compression of the yellow ligaments causing spinal cord stenosis. Anterior discectomy and fixation of C3-C4 were performed. There were no complications. The patient showed improvement and the motor tics were controlled by haloperidol. The patient remains symptom-free after 2 years of follow-up. Uncontrolled motor tics can compromise spinal cord function. Functional MRI can reproduce the abnormal movements and clarify the physiopathology.

Keywords: Tic disorders; Spinal cord injuries; Tics; Spinal cord compression.

RESUMO


Descritores: Transtornos de tiques; Traumatismos da medula espinhal; Tiques; Compressão da medula espinhal.

RESUMEN

La asociación entre tics motores y mielopatía cervical es rara y no es bien comprendida. Pocos estudios, que analizan este disturbio, han sido encontrados en la literatura hasta el momento. Este es el relato de un caso de mielopatía cervical secundaria a tics motores. Paciente masculino de 23 años con historial de trastorno de tic motor desde hace diez años, involucrando extensión forzada de la cabeza y columna cervical. En los últimos seis meses, se diagnosticaron deficiencias de sensaciones táctiles y postura cinética que progresaron hacia la señal de Lhermitte, cada vez que el paciente se movía. La Resonancia Magnética (RM) reveló lesión intramedular hiperintensa al nivel de C2-C3, degeneración al nivel de C3-C4, y ausencia de señales de compresión medular. En la visión sagital, la RM funcional con extensión de la cabeza reveló compresión anterior con protrusión del disco intervertebral y compresión posterior de los ligamentos amarillos, causando estenosis medular. Se realizó discectomía anterior y fijación de C3-C4. No hubo complicaciones. El paciente presentó mejora y los tics motores fueron controlados por haloperidol. Luego de 2 años de acompañamiento, permaneció libre de los síntomas y tics motores. Tics motores no controlados pueden comprometer a la médula espinal. La resonancia magnética funcional puede reproducir los movimientos anormales y aclarar la patofisiología.

Descritos: Transtornos de tics; Traumatismos da medula espinal; Tiques; Compressão da medula espinal.

1. Médico do Departamento de Neurocirurgia do Hospital Maior de Vento, Porto Alegre, Brasil.
2. Médico do Departamento de Neurocirurgia do Hospital Municipal de Novo Hamburgo, Novo Hamburgo, Brasil.
Correspondência: Asdrubal Falavigna. Rua: General Arcy da Rocha Nóbrega, 401 / 602 - Caxias do Sul – RS – Brasil – CEP: 95040-290. E-mail: asdrubalmd@gmail.com
INTRODUCTION

Cervical myelopathy is a pathological entity that may develop as a result of movement disorders such as motor tics\(^1\). Tics are sudden, involuntary, stereotypic, repetitive, but non-rhythmic movements or vocalizations. Their prevalence ranges between 5 to 100 cases per 10,000\(^2\). Only a few papers reporting the association between motor tics and cervical myelopathy have been found in the literature\(^1,3-10\). We report a case of a cervical motor tic that caused a traumatic cervical myelopathy.

CASE REPORT

A 23-year-old male patient had a 10-year history of motor tic disorder, involving rapid and abrupt movements of forced extension of the head and cervical region (DSM-IV F95.1). Movements were recurrent, arrhythmic, stereotyped, uncontrollable, and would disappear for variable periods of time.

The patient presented with numbness in the lower limbs that rapidly progressed to the upper limbs six months prior to neurological consultation. His symptoms worsened in the week before consultation, with the onset of more intense and uncontrollable tics associated with the sensation of shocks throughout his body (Lhermitte’s sign). The patient was investigated by MRI that showed a hyperintense, nonexpansive, intramedullary lesion at C2 with an extension at the C2-C3 level. The intervertebral disk at C3-C4 showed signal loss and mild posterior protrusion (Figure 1). He underwent a functional MRI with cervical extension, which showed posterior spinal cord compression at C3-C4 caused by a cervical stenosis at the yellow ligament.

An anterior cervical approach with C3-C4 discectomy was performed. An iliac bone graft was placed at the intervertebral space with a C3-C4 anterior fixation (Figure 2). At the 2-year follow-up, the patient was neurologically asymptomatic without myelopathy. The motor tic disorder was controlled by haloperidol.

DISCUSSION

Tics are defined as stereotyped and repetitive movements or vocalizations\(^11\). They are classified into four categories: Tourette’s syndrome (TS), defined as the presence of multiple motor tics and one or more vocal tics occurring over the span of at least 12 months; chronic tic disorder (CTD), defined as either motor or vocal tics (but not both) lasting at least 12 months; transient tic disorder, when tics are present for at least 4 weeks but not 12 months; and tic disorder not otherwise specified (NOS), diagnosed when tics are present but do not meet criteria for another tic disorder\(^12\). Studies suggested that cervical myelopathy caused by motor tics are caused by violent and frequent neck flexion or twisting and extension movements\(^6,7\). This association was usually present in youngsters with the mean interval between the onset of the tic and myelopathy 11.1 years\(^8\).

The literature contains only a few reports correlating a motor tic disorder with traumatic cervical myelopathy\(^1,3-10\) (Table 1). Nomura et al.\(^7\) reported a case of cervical myelopathy with no spondylosis or disk herniation and suggested that the lesion was due to the direct trauma caused by a neck flexion tic. Some studies found tics to be associated with cervical disk herniation and cervical spondylosis\(^5\). Fen et al.\(^13\) on the other hand, found no alteration at the neurological exam of 58 patients with Tourette’s disorder. This patient underwent a functional MRI with cervical extension in order to investigate spinal cord compression, because routine exams did not reveal it.

Cervical myelopathy due to motor tics can be treated with surgery\(^3,4,6,8\), pharmacological therapy\(^1,5\), psychotherapy\(^6\), or with botulinum toxin (BTX)\(^9,10\). In three previously reported cases, patients underwent surgical decompression but their symptoms worsened postoperatively despite treatment, and the tic remained constant in all cases\(^4,6,10\). These results probably indicate that segment stabilization is not enough to make myelopathy disappear and that treatment is necessary to control the tics. If involuntary movements persist after surgery, the risk recurrence increases; thus, pharmacologic treatment is as important as neurosurgical intervention\(^8\).
CONCLUSION

The association between motor tic disorder and myelopathy of the cervical spine is extremely rare and difficult to diagnose. We report a patient who underwent C3-C4 discectomy and anterior fixation and who remained symptom free at 2-year follow up with the administration of haloperidol. When there are no evident signs of compression, especially in the presence of discopathy, functional MRI is an effective method to clarify the physiopathology of the medullar lesion. Treatments must be individualized in order to achieve the best results for each patient.

REFERENCES


Table 1. Literature review of motor tic and cervical myelopathy.

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Age</th>
<th>Sex</th>
<th>Neuro Image</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goetz, 1980</td>
<td>16</td>
<td>F</td>
<td>Not described</td>
<td>Pharmacological</td>
<td>Positive</td>
</tr>
<tr>
<td></td>
<td>41</td>
<td>M</td>
<td>Not described</td>
<td>Psychotherapy</td>
<td>Positive</td>
</tr>
<tr>
<td>Brill, 1981</td>
<td>27</td>
<td>M</td>
<td>Disc herniation</td>
<td>Anterior cervical discectomy</td>
<td>Positive</td>
</tr>
<tr>
<td>Krauss, 1996</td>
<td>21</td>
<td>M</td>
<td>Disc herniation and spinal cord canal stenosis</td>
<td>Pharmacological</td>
<td>Unchanged</td>
</tr>
<tr>
<td></td>
<td>23</td>
<td>M</td>
<td>Spinal cord canal stenosis and Klippel-Feil malformation</td>
<td>Cervical decompression by C3-C5 laminectomies</td>
<td>Relapse</td>
</tr>
<tr>
<td>Adler, 1996</td>
<td>38</td>
<td>M</td>
<td>Disc herniation</td>
<td>C6-C7 anterior fusion, C3-T2 laminectomies, C7-T1 foraminotomy on the left followed by a C7-T1 posterior cervical fusion and Botulinum toxin type A</td>
<td>Relapse</td>
</tr>
<tr>
<td>Muroi, 2002</td>
<td>15</td>
<td>M</td>
<td>Spinal cord canal stenosis and high intensity T2-weighted MRI</td>
<td>C3 to C7 laminoplasty</td>
<td>Positive</td>
</tr>
<tr>
<td>Dobbs, 2003</td>
<td>25</td>
<td>M</td>
<td>Disc herniation</td>
<td>Anterior cervical discectomy</td>
<td>Relapse</td>
</tr>
<tr>
<td>Lin, 2007</td>
<td>20</td>
<td>M</td>
<td>Spinal cord stenosis of the intervertebral space and traumatic herniation of the intervertebral disc</td>
<td>Pharmacological</td>
<td>Positive</td>
</tr>
<tr>
<td>Aguirregomozcorta, 2008</td>
<td>42</td>
<td>M</td>
<td>Cervical spinal stenosis and spondylosis</td>
<td>Botulinum toxin type A</td>
<td>Positive</td>
</tr>
<tr>
<td>Present paper, 2010</td>
<td>23</td>
<td>M</td>
<td>High intensity medullary signs and functional MRI showed posterior compression by yellow ligament.</td>
<td>Anterior cervical discectomy</td>
<td>Positive</td>
</tr>
</tbody>
</table>