An unusual case of acute wandering paralysis

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ABSTRACT

The authors reported on a 63-year-old diabetic male who developed a strange wandering hemiparesis affecting first left side and then right side, not consistent with a right parasagittal meningioma nor with ischemic stroke. The subsequent rapid worsening of clinical picture, with occurrence of paraparesis, urinary incontinence and midthoracic sensory level, together with the evidence of leucocytosis, led to the diagnosis of a T6-T7 spondylodiscitis with spinal cord compression. The authors underlined the difficulties in diagnosing this condition in the setting of general hospital practice and the importance of clinical and neurological examination.

1. Introduction

Mismatch between clinical signs and symptoms, known medical or surgical pathologies and radiological evidence, is a common and hazardous issue in daily medical practice, especially in the setting of emergency room (ER). Moreover, even in case of complex and confounding clinical pictures, physicians generally prefer to look for a single diagnosis that can explain all, or most of, patients’ complaints. However, this process often results in neglecting some key findings that could lead to choose alternative diagnoses and perhaps to find more effective treatments. In the present work, the authors aim to illustrate these clinical difficulties by means of a challenging case of wandering paralysis.

2. Case report

A 63-year-old man suffering from type II diabetes came to the ER because of paralysis in his left arm and pain at the left shoulder since waking up. On neurological examination, there was complete left arm paralysis and left leg paresis without sensory changes. Serum glucose was 340 mg/dL, glycosylated hemoglobin was 9.3% and white cell count was 11.95 × 10⁹/L. An unenhanced brain CT scan revealed a small parasagittal meningioma adjacent to right motor cortex (Figure 1A). On electroencephalography, few spikes on right hemisphere were recorded. During the following 5 days, the patient completely recovered from motor deficit and was discharged with anticonvulsant therapy and surgical removal of meningioma was scheduled.

Fifteen days later, the patient came back to ER complaining for severe pain in the lumbar region and in right leg. Neurological examination disclosed Lasègue sign at 30°/C14, absence of achilles reflex, mild weakness in foot plantar flexion-all in right side, without changes in sensation. CT scan of the lumbosacral spine showed degenerative changes with multiple bulging disks, which could not explain the symptoms. Non-steroid anti-inflammatory medications were started with partial relief of radicular pain and persistence of back pain.

Two days later, the patient developed a paraparesis; pyramidal signs and sensation deficits were absent; upper extremities were fine. Body temperature was normal. Laboratory examinations confirmed moderate leucocytosis (12.43 × 10⁹/L, neutrophils 77.8%) with an increased alkaline phosphatase (409 IU/L); serum glucose was 140 mg/dL. A brain MRI was undertaken that confirmed the right parasagittal meningioma without bleeding, edema or signs of venous obstruction (Figure 1B). In the following hours, back pain increased and extended to midthoracic region (patient could not lie down). Moreover, urinary retention developed, together with a midthoracic sensory level.
Therefore, emergent spinal MRI was carried out that showed severe spinal cord compression sustained by an epidural mass centered on T6-T7 vertebral bodies and a casting of pathological tissue dropped down in the vertebral canal towards the cauda (Figure 1C). A CT scan confirmed the extensive destruction of T6-T7 endplates and vertebral bodies, suggesting infectious spondylodiscitis (Figure 1D).

Emergent decompressive laminectomy was performed and an extradural abscess was drained. Microbiological examination revealed *Staphylococcus aureus* while blood and urinary cultures were negative. Biopptic material was also negative for cancer cells. The patient underwent prolonged specific antibiotic treatment (teicoplanine, levoflaxacine and meropenem for 3 months) and bed rehabilitation, with marked improvement of motor and sensory deficits (ASIA, grade D); white cell count dropped to normal. A second surgery was then performed for spine stabilization. Removal of intracranial meningioma was delayed.

### 3. Discussion

Two key points can be identified from this case report: the synchronous occurrence of different diseases and the insidious onset of spondylodiscitis.

Coexistence of different diseases is generally overlooked by physicians, who aim to find a single cause for all symptoms. Even though this latter condition is far more frequent, when clinical findings are too divergent and radiological examinations do not lead to a definite diagnosis, physicians should be sufficiently open-minded and not rule over the possibility of concurrent diseases. Using this method, it is possible to avoid delays in diagnosis of serious illnesses and therefore to start the right therapy as soon as possible. In our case, both the previous occurrence of meningioma-related seizures and the known diagnosis of diabetes mellitus masked the subacute onset of a spondylodiscitis that was causing progressive paraparesis.

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**Table 1**  
*ASIA classification.*

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Complete: no motor or sensory function preserved</td>
</tr>
<tr>
<td>B</td>
<td>Incomplete: sensory but not motor function preserved below the neurological level (includes sacral segments S4-S5)</td>
</tr>
<tr>
<td>C</td>
<td>Incomplete: motor function preserved below the neurological level (more than half of key muscles below the neurological level have a muscle strength grade &lt; 3)</td>
</tr>
<tr>
<td>D</td>
<td>Incomplete: motor function preserved below the neurological level (more than half of key muscles below the neurological level have a muscle strength grade &gt; 3)</td>
</tr>
<tr>
<td>E</td>
<td>Normal: sensory and motor function normal</td>
</tr>
</tbody>
</table>
Spondylodiscitis is always to be considered as a diagnosis when facing with symptoms like fever, back pain and leucocytosis, especially if they appear insidiously\cite{2-7}. In fact, the disease can thereafter evolve tumultuously towards flaccid paraplegia, anestesia and sphincter disturbances. Risk factors for spondylodiscitis are previous spinal procedures or distal polyneuropathy from a variety of causes, including diabetes. MRI of the whole spinal cord is warranted in case of neurological deficits that are not fully consistent with other pathologies (intracranial mass, stroke, lumbar disk herniation)\cite{8}. The severity of the clinical picture can be described using ASIA classification\cite{1}, which was originally designed for spinal trauma and carries prognostic implications. In detail, the probability to achieve a neurological improvement in the first year from a spinal injury, whatever the treatment strategy, in patients starting from class A is 20\%, with reversely 80\% chance to remain stable\cite{9}. However in spondylodiscitis, even in case of a severe spinal cord injury, like the one described in this work, clinical recovery can be highly satisfactory with surgical decompression, when needed, and antibiotic therapy\cite{10}.

**Conflict of interest statement**

The authors report no conflict of interest.

**References**


