ACUTE NECROTIC MYELOPATIA AFTER SPIDER BITE

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ABSTRACT: A case of a 56 year-old patient bitten by a spider, species *Loxosceles*, who developed a local bubbly lesion and after six days an ascendent necrotizing myelitis which progressed towards a rapid lethal exit. The authors emphasize the difficulties in establishing the aetiological diagnosis in the myelitis group of undetermined causes and report this present paper trying to relate the venomous accident and the spinal lesion. The mechanisms that could be responsible in the genesis of the spinal lesion and the behaviour of the antigenic complex of the toxin are discussed.

KEYWORDS: Acute myelitis — Spider bite.

INTRODUCTION

Necrotizing myelopathies are characterized by an acute aggression to the spinal cord with extensive necrosis and minimum signs of inflammation (Kissel, P., Schmitt, J. 1972). They are the expression of the action of many aetiopathogenic agents as well as different physiopathological mechanisms. There is a group of unknown causes and some are related to previous venomous accidents.

In *Arachnidae* accidents a neurological onset is rare but, depending on the species, an immediate onset, a temporary one, or a severe onset may occur, due to the toxin itself or to secondary systemic aggressions. (Chok et al., 1979, Dillaha et al, 1964, Gajardo-Tobar, 1966, Jacobs, 1969, Matthews et al, 1973, Pitts, 1962).

The scarcity of necrotizing myelopathy cases associated with venomous accidents, the difficulties in establishing an clinicopathologic correlation and the possible aetiopathogenic mechanisms justify this communication.

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CASE REPORT

A 56 year-old negro man, Brazilian, married, farmer, coming from a region in the hinterland of the São Paulo State, was examined on the 24th of December, 1979, reporting that three days ago, he had been bitten by a spider on the left buttock. Soon after, he started to feel a general physical indisposition, shivering fits and local pain from the bite. Two days before being hospitalized he felt strong headache, nausea and vomiting. One day before, he felt feeble an his left lower limb ached, and in the same day the right leg was also involved. The patient was examined at the Butantan Institute (São Paulo), where by the characteristics of the lesion, the bite was diagnosed as due to *Loxosceles* spider.

Patient in a regular general state, with a mild dyspnea, not icteric or cyanotic. Blood pressure 90 x 50 mm Hg., pulse of 72 beats/min.; axillary temperature 37.2°C. There was a circular lesion of the skin and the subcutaneous sue of the left buttock with a diameter of approximately 2.0cm., with phlogistic signs and a melicercide crust. The pulmonary examination showed a slight inferior drawing with medium subcrepastion stertorous breathing in the lower left lung. The cardiovascular apparatus had rythmical noises and normophonetic heart sounds. The abdomen was normal. The patient lead a delay bladder catheter. The patient was anxious with contact difficulties due to the dyspnea. Applying the extension of superior members procedure these upper limbs fell globally and rapidly reflexes absent in the lower limbs, present and symetrical in the upper limbs. Hypotonia in the lower limbs. The superficial abdominal reflexes, the cremasteric and the plantar reflexes were absent. Superficial anesthesia of the great toes. Rigidity of the neck. Urinary retention. The cranial nerves were normal. The hematological examination disclosed a normochromic anemia and leukocytosis of 15,000/mm³. The other routine laboratory investigations were normal. The results of the cerebrospinal fluid examination are shown in table I. Normal descendent iodized oil perimyelographya.

Evolution: The neurological form, first represented by a crural paraparesis with a sensory syndrome, which pointed to damage at the lumbosacral segment, developed rapidly with a severe flaccid paralysis of the lower limbs. The motor manifestations assumed an ascending character, which attained moderately the upper limbs, but the sensory disorders reached the fourth thoracic level, and consisted of thermal, tactile and painful anesthesia and loss of position sense in the great toes. Death occurred on the third day after the onset of the spinal involvement.

The general pathological aspect showed a bilateral pneumonia and an acute splenitis. The brain weighed 1,400g. The external examination of the brain and the verticofrontal cuts showed no abnormality. The spinal cord was completely softened from the third cervical to the first sacral segment. The meninges and the blood vessels showed no modifications.

The only microscopic modifications were in the spinal cord, both in the white and in the gray matter, being more marked in the thoracic and high lumbar levels. At these points, the spinal cord was entirely necrotized, formed by tissue debris. In these, the only type of cells
### TABLE I

Results of the Examinations of the Cerebrospinal fluid

<table>
<thead>
<tr>
<th>Date</th>
<th>Puncture</th>
<th>Aspect</th>
<th>Global LE</th>
<th>citology ER</th>
<th>Specific % L M N</th>
<th>Chloride</th>
<th>Biochemical mg/dl</th>
<th>Pandy</th>
<th>Takata</th>
<th>Immunological Reactions</th>
<th>Spinal Syphilis</th>
<th>Cysticercosis</th>
<th>Bacteriological Examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>24/12/79</td>
<td>SOD</td>
<td>Turbid</td>
<td>1365</td>
<td>213</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Neg.</td>
<td>Neg.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26/12/79</td>
<td>LD</td>
<td>Colorless</td>
<td>1469</td>
<td>170</td>
<td>2</td>
<td>1</td>
<td>93</td>
<td>745</td>
<td>78</td>
<td>44</td>
<td>+ V</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27/12/79</td>
<td>SOD</td>
<td>Turbid</td>
<td>3360</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>93</td>
<td>655</td>
<td>58</td>
<td>67</td>
<td>Neg.</td>
<td>Neg.</td>
<td></td>
</tr>
<tr>
<td>27/12/79</td>
<td>LD</td>
<td>Turbid</td>
<td>1280</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>97</td>
<td>651</td>
<td>28</td>
<td>156</td>
<td>+ + V</td>
<td></td>
<td></td>
</tr>
<tr>
<td>29/12/79</td>
<td>LD</td>
<td>Turbid</td>
<td>42700</td>
<td>1365</td>
<td>2</td>
<td>1</td>
<td>97</td>
<td>659</td>
<td>29</td>
<td>490</td>
<td>+ + V</td>
<td>Neg.</td>
<td></td>
</tr>
</tbody>
</table>

SOD. Suboccipital; LD. Lombar; LE. Leukocytes; ER. Erythrocytes; L. Lymphocytes; M. Monocytes; N. Neutrophils; Neg. Negative; + weakly positive; ++. Strongly positive; + V. positive red.
recognizable were polymorphonuclear leukocytes. Inwardly or amidst the tissue debris most of the blood vessels showed fibrinoid necrosis. In the segments where the necrosis was milder, the spinal parenchyma was feeble, spongelike, areolated, being impossible to differentiate between the gray and the white matter. Focal collections of polymorphonuclear leukocytes were scattered randomly on the tissue, mostly on the perivascular areas. The little nuclei still visible did not allow any cell identification. Exceptionally neuronals ghosts were present (figure 1). A ring like nervous tissue, better structured, was visible in the spinal periphery, near to the piamater.

Fig. 1 — Microscopic aspect in the spinal cord: necrotized areas and polymorphonuclear leukocytes are seen; the cells are mostly on the perivascular region. Hematoxylin and eosin stain. A=X 78 B=X 200.

In the upper cervical and the low sacral segments, the appearance was practically normal, with the presence of some rare cuff of limphocyte cells surrounding the intraparenchymatous vessels.

The whole of the spinal cord, the meninges and the nervous roots had no histological alterations, with the exception of some rare and discrete lymphatic perivascular sheaths in the meninges.

**DISCUSSION**

In this case, the concomitance of the venomous accident (loxoceelism) with the persistance of the bubbly lesion on the left glutes region, which maintains the toxine inside it, and the development of the necrotizing myelitis deserve considerations on a possible correlation between the
events (Limber et al, 1962). Considering the antigenic aspect, it is known that the venom of certain spiders can lead to immediate and late immune reactions, consuming the complement C₃ (Futrell et al, 1978, Majeski et al, 1976, Smith et al, 1970). The severity of the systemic lesions depends on the period of inoculation and on the resistance of the host (Gajardo-Tobar, 1966). The pathological aspect of this case does not allow us to assert the relation between the venomous lesion in the gluteal region and the myelitis, but this possibility cannot be rejected because the toxine diffusion of the inoculate in the spinal cord can be biphasic and can occur through the lymphatic vessels, foreign arterial adventitia and veins, and may ascend by the Batten plexus, and spinal.

RESUMO: Os autores apresentam caso de paciente de 56 anos de idade picado por aranha, Loxosceles, desenvolveu lesão bolhosa no local. Após seis dias houve aparecimento de mielite necrosante ascendente que evoluiu rapidamente para êxito letal. São enfatizadas as dificuldades para se estabelecer o diagnóstico etiológico nas mielites de causa indeterminada e são feitas tentativas de correlação entre o acidente peganhento e a lesão espinal.

Os mecanismos responsáveis pela gênese da lesão espinal e o comportamento antígenico complexo de toxina são discutidos.

PALAVRAS-CHAVE: Mielite aguda. Picada de aranha.

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