LASEQUE'S SIGN — AN IMPORTANT BEDSIDE DIAGNOSTIC SIGN IN ACUTE POLYRADICULOPATHY (GUILLAIN-BARRE SYNDROME)

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SYNOPSIS

Fourteen consecutive cases of Guillain-Barre Syndrome were examined for straight leg raising sign (Laseque's sign). Thirteen were positive (93%). The high percentage illustrates the importance of looking for this sign in all suspected cases of Guillain-Barre Syndrome where the underlying pathology is an acute inflammation and demyelination of nerve roots.

INTRODUCTION

It is widely accepted that the main site of lesion in Guillain-Barre Syndrome (GB Syndrome) is the site of union of anterior and posterior roots extending a short distance proximally and distally (Naymaker et al, 1949; Widerholt et al, 1964). There has been isolated reports therefore of radicular pain or sciatica in this condition (Brown, 1955). However the significance has not been highlighted. The frequency and consistency of radicular pain was looked for in 14 consecutive cases of GB Syndrome by the straight leg raising test, so that its value as a diagnostic aid could be evaluated.

MATERIAL AND METHOD

(a) The recognition and diagnosis of GB Syndrome rest on the strikingly characteristic clinical picture. Its features are a symmetrical flaccid paralysis with abolition of the tendon reflexes with subjective sensory symptoms without much objective changes. The patients are usually afebrile though they may have a preceding febrile illness or associated precipitating illness. Bilateral facial paralysis is common while other cranial nerves are uncommonly involved. Complete recovery is the rule but rarely the patient may die. An increase in cerebrospinal fluid protein with lack of increase of cells is usual but is not an invariable feature (Widerholt et al, 1964; Petch, 1949; Osler & Sidell, 1960).

(b) Laseque's sign is tested by making the patient lie flat on the back with both legs fully extended. In turn both legs should be passively flexed at the hip joint, full extension being maintained at both knee joints. Normally one should
be able thus without difficulty to lift the patient's leg to about 70° off the horizontal at the hip joint. Lasegue's sign becomes positive if the leg cannot be elevated to this extent due to pain and strong resistance. Two elements enter into this phenomenon i.e. element of contracture of reflex origin and pain of radicular origin (Monrad-Krohn). The pain characteristically is experienced from the gluteal region radiating down the thigh posteriorly.

Care is taken that pain due to pressure on muscles (myalgia, myositis) or contractions is not regarded as positive.

(c) Nerve conduction studies were performed on all patients, except Case 1, confirming the presence of peripheral neuropathy.

Results: See the Table.

Table: Lasegue's sign in 14 cases of acute polyradiculopathy

<table>
<thead>
<tr>
<th>Case No</th>
<th>Particulars</th>
<th>Diagnostic Summary</th>
<th>Lasegue's Sign</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F/28 Australian</td>
<td>Fever, weakness, hypotonia, areflexia, moderate sensory loss, bilateral C6 palsy, CSF Protein 110mg, Cells 0. Recovered.</td>
<td>+ve</td>
</tr>
<tr>
<td>2</td>
<td>M/42 Chinese</td>
<td>Flu-like illness, rapid weakness, proxima; more than distal, areflexia, bulbar palsy, CSF Protein 150, cells 2. Recovered</td>
<td>+ve</td>
</tr>
<tr>
<td>3</td>
<td>M/8 Malay</td>
<td>Preceding fever, hypotonia, weakness, proximal more than distal, hyporeflexia, CSF Protein 70, cells 24. Complete recovery.</td>
<td>+30°</td>
</tr>
<tr>
<td>4</td>
<td>F/24 Chinese</td>
<td>Flu-like illness, paresthesia, weakness, proximal more than distal, hyporeflexia, bilateral C3 palsy, CSF Protein 50, cells 0. Recovered.</td>
<td>+50°</td>
</tr>
<tr>
<td>5</td>
<td>F/58 Eurasian</td>
<td>Severe weakness, distal more than proximal, areflexia of lower limbs, mild sensory loss all limbs, CSF normal. Recovered.</td>
<td>+45°</td>
</tr>
<tr>
<td>6</td>
<td>M/58 Chinese</td>
<td>Mark ataxia, slight weakness of all limbs, areflexia, CSF Protein 120, cells 0. Slow recovery.</td>
<td>+45°</td>
</tr>
<tr>
<td>7</td>
<td>F/42 Chinese</td>
<td>Fever, complete paralysis, areflexia, respiratory arrest, no sensory loss, normal CSF. Now recovering (3 months)</td>
<td>+45°</td>
</tr>
<tr>
<td>8</td>
<td>M/17 Malay</td>
<td>Rapid weakness, distal more than proximal, areflexia only lower limbs, CSF normal. Complete recovery.</td>
<td>+50°</td>
</tr>
<tr>
<td>9</td>
<td>M/42 Malay</td>
<td>Underlying diabietic, acute weakness, areflexia, minimal sensory loss, CSF normal. Recovered.</td>
<td>+50°</td>
</tr>
<tr>
<td>10</td>
<td>M/23 Malay</td>
<td>Weakness with paresthesia, affecting only distal muscles. All reflexes absent. CSF normal. Recovered</td>
<td>-ve</td>
</tr>
<tr>
<td>11</td>
<td>M/40 Chinese</td>
<td>Acute bilateral sciatic pain, generalised weakness, areflexia, bilateral C7, CSF Protein 110, cells 20. Complete recovery.</td>
<td>+45°</td>
</tr>
<tr>
<td>12</td>
<td>F/60 Chinese</td>
<td>Total external ophthalmoplegia, bilateral C7, ataxia, areflexia, refuse L.P. Recovering.</td>
<td>+30°</td>
</tr>
<tr>
<td>13</td>
<td>F/50 Chinese</td>
<td>Acute onset of weakness, areflexia, bilateral facial palsy, CSF protein 70. Complete recovery.</td>
<td>+40°</td>
</tr>
<tr>
<td>14</td>
<td>F/35 Chinese</td>
<td>Acute bilateral sciatic pain, weakness hyporeflexia of lower limbs, bilateral C7 palsy, CSF normal Recovered.</td>
<td>+30°</td>
</tr>
</tbody>
</table>
DISCUSSION

The findings here are significant because 13 out of 14 cases (93%) examined showed positive Laseque's sign. 3 out of the 14 cases were mistaken initially to have prolapsed intervertebral disc until the full blown clinical picture developed within a few days later.

Haymaker and Kernohan reported 50 fatal cases in 1949. Their findings are that the peripheral nervous system is consistently affected; the spinal cord, brain stem, cerebellum and cerebrum being spared except for retrograde changes in the anterior horn cells and motor nuclei of the brain stem. The lesions of the peripheral nervous system is concentrated as mentioned earlier in the region where the anterior and posterior roots fuse, and in the adjacent portions. The sequence of events include swelling and irregularity of the myelin sheath, involvement of axis cylinders, appearance of lymphocytes, phagocytes and later proliferation of Schwann's cell sheath. Where changes are predominantly in the anterior root sensory changes are mild, where both are affected sensory loss is significant.

These findings are supported by other pathologists (Reye, 1954) subsequently. This would explain the high incidence of cases with positive Laseque's sign. There is bilateral radiculitis. However pain may be contributed by surrounding meningeal inflammation in some cases. Should weakness and hyporeflexia be confined to only the lower limbs with bilateral positive Laseque's sign then it is mandatory to exclude a cauda equina lesion e.g. massive disc prolapse by noting sensory dermatomes affected, testing for sacral hypoesthesia, bladder involvement and if doubt exist a myelogram. Meningitis will also give bilateral positive Lasegue's sign (in addition to Kernig's test) but the neck stiffness, different clinical picture, CSF pleocytosis clearly differentiate this condition from GB Syndrome.

Although we have not systematically followed up these patients to see when Laseque's sign returns to normal the overall impression is that it remains abnormal for at least one to two months in the moderate and severe cases of weakness. The one case where this sign was negative had no weakness of proximal muscles.

We conclude that this sign is of great value in the firm clinical diagnosis of Guillain-Barre Syndrome where the underlying pathology is demyelination of nerve roots, unlike most other forms of peripheral neuropathies. Absence of root pain, as elicited by Laseque's sign is unusual in Guillain-Barre Syndrome.

REFERENCES