Lumbosacral plexus neuropathy

General information

Analogous to idiopathic brachial plexitis 1)

It is controversial whether this actually exists in isolation without diabetes.

DM is a major risk factor and thus justifies the continued classification into diabetic and nondiabetic forms ²⁾.

Often starts with LE pain of abrupt onset, followed in days or a few weeks by weakness with or without muscle atrophy. Sensory symptoms are less prominent, and usually involve paresthesias. Objective sensory loss is only occasionally seen. There may be tenderness over the femoral nerve.

Etiology

Other etiologies are similar to that for brachial plexus neuropathy except that under tumor, a pelvic mass should also be included (check prostate on the rectal exam).

Evaluation

Evaluation is as for brachial plexus neuropath, except that instead of a brachial plexus MRI, a lumbar MRI and pelvic CT should be done to rule out masses.

EMG is key to diagnosis: evidence of patchy denervation (fibrillation potentials, and motor unit potentials that are either decreased in number or increased in amplitude or duration and polyphasic) involving at least 2 segmental levels with sparing of the paraspinal muscles is highly diagnostic (once diabetes, etc. have been ruled out).

Differential diagnosis

May be confused with femoral neuropathy or L4 radiculopathy when quadriceps weakness and wasting occurs. Similarly, L5 radiculopathy or peroneal neuropathy may be erroneously suspected when foot drop is seen. Straight leg raising may occasionally be positive. Conspicuously absent are: back pain, exacerbation of pain by Valsalva maneuver or back motion, and significant sensory involvement.

See differential diagnosis of foot drop and other causes of sciatica.

Outcome

Recovery from pain precedes the return of strength. Improvement is generally monophasic, slow (years), and incomplete.

1)

Evans BA, Stevens JC, Dyck PJ. Lumbosacral Plexus Neuropathy. Neurology. 1981; 31:1327–1330

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