An Interesting Case Of Painful Quadriparesis

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INTRODUCTION

Patients with polymyositis usually present with symmetrical, proximal muscle weakness in the upper and lower extremities, usually painless weakness with elevated skeletal muscle enzyme levels; and have characteristic electromyography (EMG) and muscle biopsy findings.

CASE REPORT

40 year old female, with history of stroke at age of 27 yrs, presented with weakness of both upper and lower lower limbs with pain since 9 months. H/o CVA 13 yrs ago, Rt hemiparesis, not received treatment, with minimal residual deficit. H/o fall on the head and neck injury 5 yrs ago. Third pregnancy second trimester spontaneous abortion.

ON EXAMINATION

Bilateral facial hyperpigmentation [melasma].
Bilateral ptosis, No opthalmoplegia.
Rt UMN facial nerve palsy (old). Tone mild hypertonia. Power of 3/5 in all four limbs including neck and trunk muscles. Other systems normal.

MANAGEMENT

INVESTIGATIONS

CBC -
Hb -12.3, TLC- 6300cells/cu.mm with normal differential count. Platelet Count-1,80,000 cells/cu.mm, ESR-12, RFT, LFT & ELECTROLYTES, THYROID profile are NORMAL. ANA & APLA are NEGATIVE. CPK 54, LDH 410.
Mantoux – Negative,
HIV Spot & HBs Ag Spot – Negative.
Radiology-
MRI BRAIN SPINE: L4-L5 disc bulge
NCV of all four limbs: grossly NORMAL study.
EMG: shows myopathic pattern.
MUSCLE BIOPSY: end stage muscle disease

TREATMENT

Patient was treated with oral steriods (prednisolone 60 mg/day) vitamin B12 supplementation, after 7 days she improved and is able to walk with support.

DISCUSSION

Polymyositis is an idiopathic inflammatory myopathy that causes symmetrical, proximal muscle weakness, painless weakness, elevated skeletal muscle enzyme levels; and characteristic electromyography (EMG) and muscle biopsy findings, an immune-mediated syndrome secondary to defective cellular immunity that is most commonly associated with other systemic autoimmune diseases, more common in women than in men, especially those aged 45-60 years. Our patient presented at 40 years of age with painful muscle weakness involving initially distal muscles and then proximal muscles of all four limbs. On examination we thought of a differential diagnosis of compressive myelopathy at C5-6, chronic inflammatory demyelinating polyneuropathy and polymyositis. Routine investigations were normal and autoimmune markers (ANA & APLA) were negative. MRI spine was not showing evidence of compression and further EMG showed myopathic pattern. So, muscle biopsy was done, which showed end stage muscle disease, a terminal histological pattern to any longstanding myopathy.

CONCLUSION

Based on age of presentation and muscle biopsy findings we arrived at the diagnosis of polymyositis

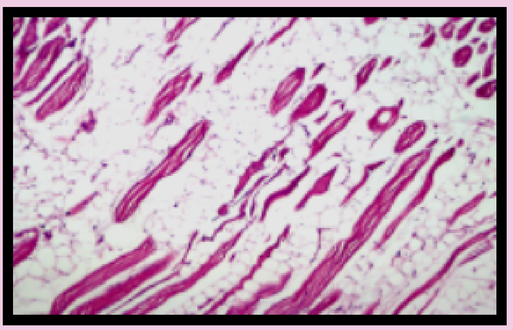

Fig. 1-
Extensive Adipose Tissue

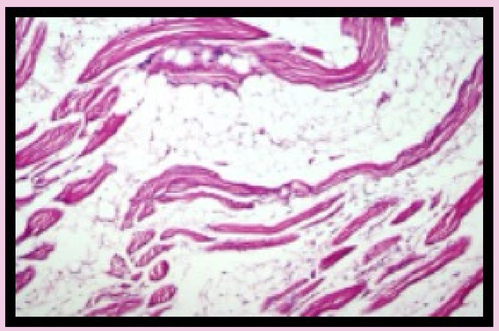

Fig. 2-
Marked Myofibre Atrophy

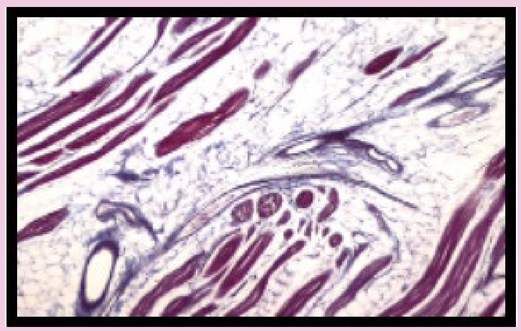

Fig. 3-
Masson's Trichrome (MT)

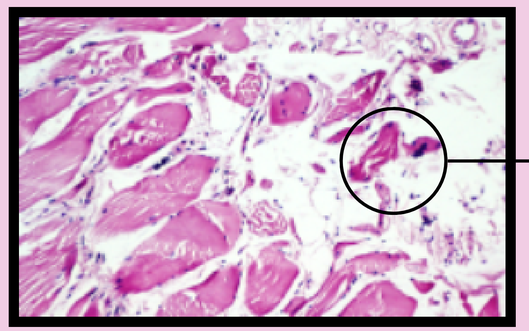

Fig. 4-
Atrophic myofibres with clamped nuclei

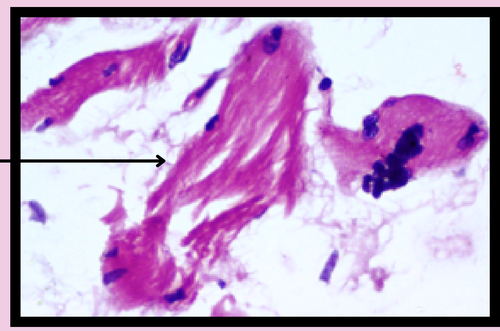

Fig. 5-