A TRICKY PROGRESSIVE WEAKNESS

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Case Report

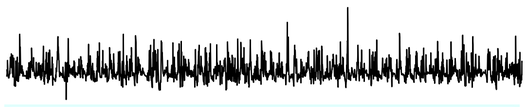
A 63 years old man, presented with 10 days story of weakness in lower limbs. He reported a previous pneumonia treated with antibiotics, one week before the onset. Symptoms have been worsened during the days before admission and forced him to use a stick to walk and to ask help to stand.
Neurological examination showed: pure motor paresis of lower limbs, with proximal strength graded 3/5 while spared distal movements, conserved flexion plantar reflexes and brisk tendon reflexes. His laboratory tests revealed a marked increase of CPK (14799), and GOT (741) and GPT (266).
Electrophysiological studies showed normal amplitude of motor and sensor nerve action potential and distal conduction velocity, EMG showed a reduced recruitment and amplitude of motor potential in iliopsoas and deltoid muscles but no myositis signs. Suspecting a myopathy, he was treated with intravenous infusion of 1 mg methylprednisolone for 5 days. On hospital day 3, the paresis became worse: consisting in inability to lift legs from the bed and areflexia. His CSF examination revealed 130 protein and cell count of 2. Viral-bacterial tests and serological tests for self-directed and paraneoplastic antibodies were negative.
Further ENG study showed prolonged F-wave latencies, poor F-wave repeatability and prolonged distal latencies, consistent with demyelination of nerve roots; normal recruitment muscle pattern, no fibrillation. He was treated with immunoglobulins 0.4 mg/kg for 5 days. During the first 5 days of therapy the weakness was spreading to the arms: proximal inability to keep arms lifted, conserved grasp strength; areflexia of upper limbs. He had no cranial nerves involvement, or autonomic dysfunction. Later he started a slow recovery, and 15 days after therapy neurological examination showed no strength deficit in upper limbs and ability to lift lower limbs up for few seconds. This is an example of GBS associated with myopathy; few cases are reported in literature with such a high level of CPK. They are associated with acute denervation due to direct axonal damage by viral infection or with concomitant myositis due to mycoplasma infection plus clarithromycin toxicity. We didn't find anyone of them. This case should teach to think about GBS even if the clinical pattern is uncommon and EEG not diagnostic 2 weeks from symptoms onset, in case of clues like albumin-cytological dissociation and prolonged F waves in a EEG control.

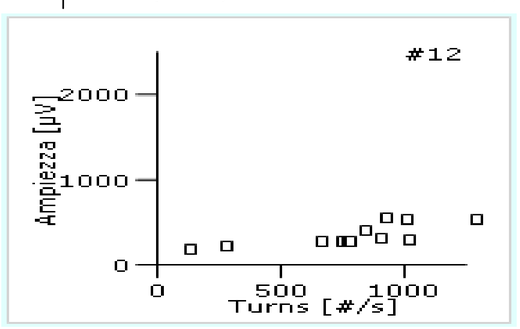
Discussion

This is an example of GBS associated with myopathy; few cases are reported in literature with such a high level of CPK. They are associated with acute denervation due to direct axonal damage by viral infection or with concomitant myositis due to mycoplasma infection plus clarithromycin toxicity. We didn't find anything of that. This case should teach to think about GBS even if the clinical pattern is uncommon, in case of prolonged F waves and albumino-citological dissociation.

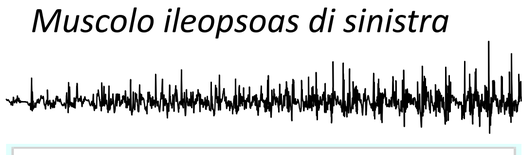
EMG at admission

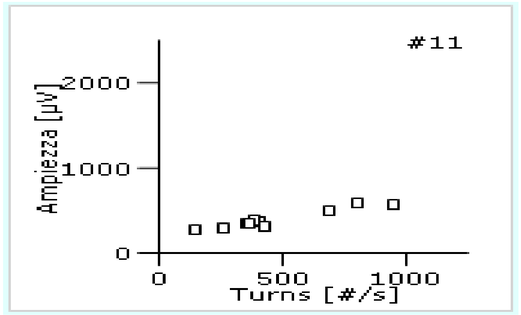
Muscolo deltoide di destra





Muscolo ileopsoas di sinistra



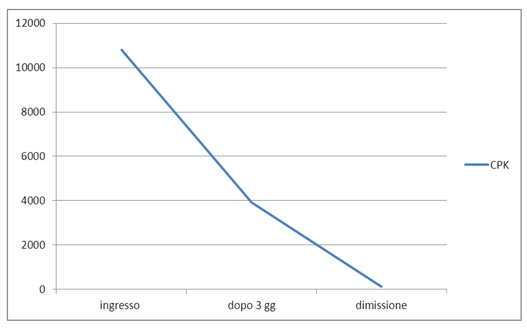


ENG during hospitalization

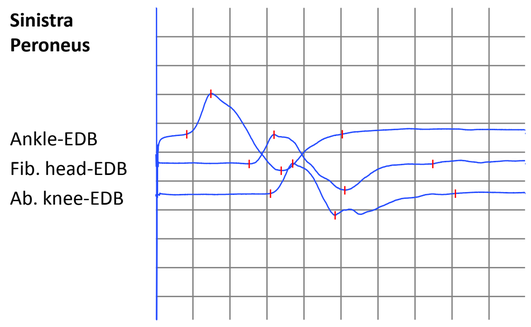
ENG di controllo

nervo	Latenza msec	Ampiezza mV	Velocità m/sec
Mediano sn	5.37	2.8	39.2
Ulnare sn	3.79	7	47
Peroneo sup ds	5.19	2.2	41.8
Peroneo sup sn	4.19	5.3	38
Tibiale ds	4.5	13	43.5
Tibiale sn	4.11	12.4	45.9
Surale ds	3.59	6.3	37.1
Surale sin	2.34	12	34.8
Mediano sensitiva sn	4.09	19	34.2
Ulnare sensitiva sn	3.03	10.9	39.6
Radiale sensitiva sn	2.12	16.3	44.8

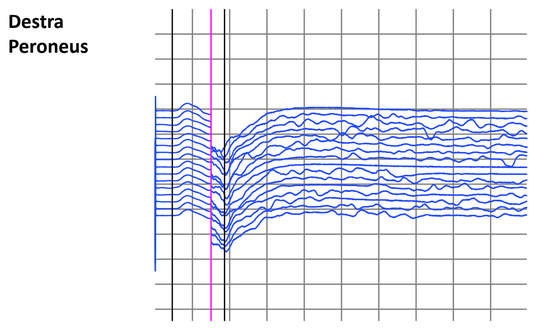
CPK



n. peroneo sup. destra



Onda F n. peroneo sup. destra



Proximal deficit

References

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