

MUSCOLAR AND NERVE INVOLVEMENT IN COURSE OF A POSSIBLE LYMPHOMA

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Introduction: There are some different possible mechanisms of peripheral neurological involvement in course of lymphomas:

- 1: Endoneural infiltration by neoplastic B-cells causes segmental demyelination and axonal degeneration: NEUROLINFOMATOSIS;
- 2: Inflammatory or dys-immune neuropathy (GBS or CIDP) generated by HL>NHL auto-Ab (anti-Hu) or infections in immunodeficient patients;
- 3: Haematological metastasis that closes the vessels by local intravascular proliferation or exerts direct compression: stroke/embolus in PNS (or CNS) especially in intravascular lymphomas or angiotropic lymphoma (vasculitic-like multiple mononeuropathy);
- 4: Direct neoplastic nerve infiltration (typical in HIV+);
- 5: Vasculitis and crioglobulinemia (type I-II)/ amiloidosis (monoclonal paraproteinemia)/ antinerve monoclonal Ab (antimyelin associated glycoprotein and GM1 ganglioside) in NHL.

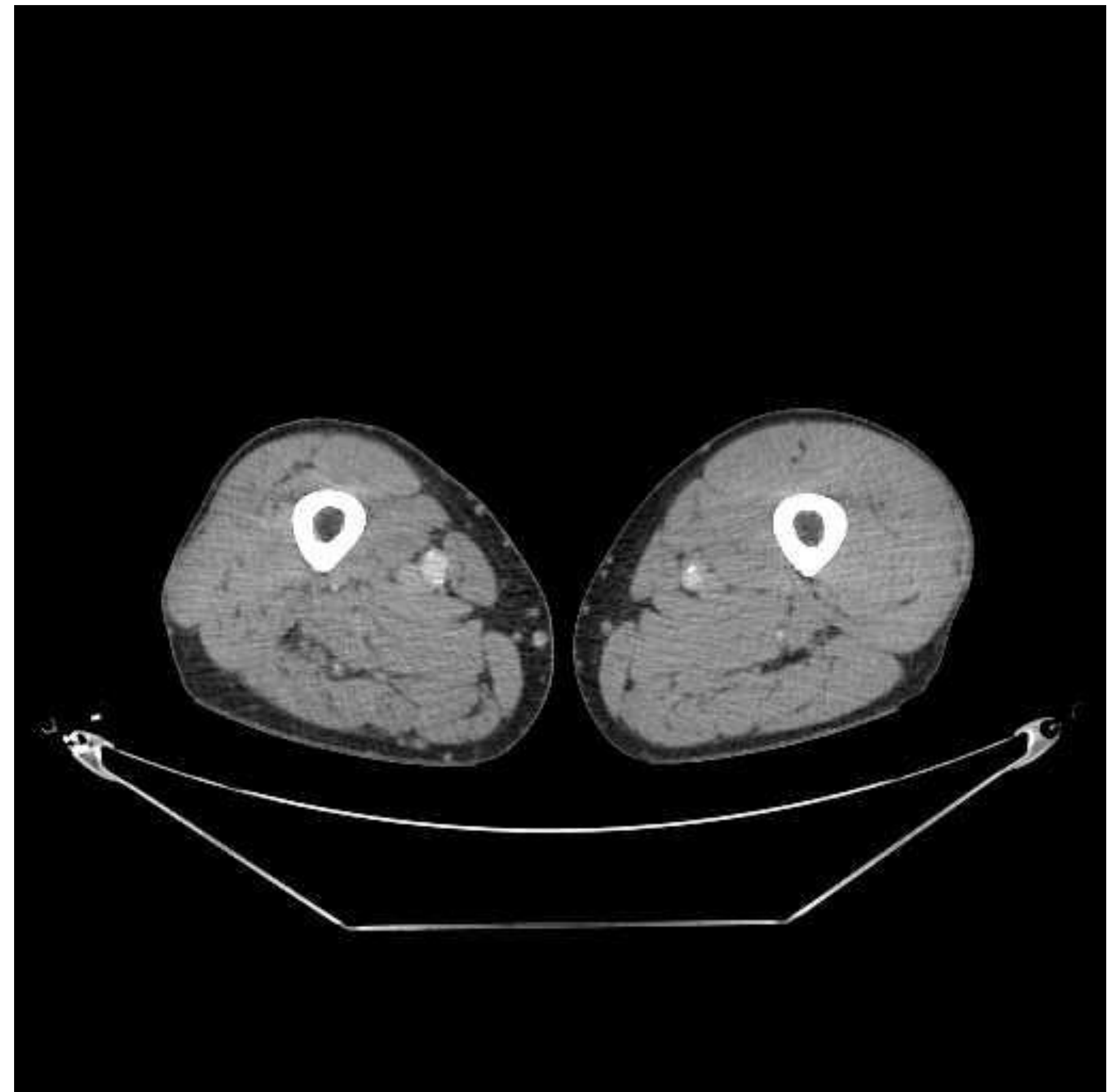


Fig 1

Case report: 56 y-o man, schizophrenic and HCV+ patient taking 600 mg/die of quetiapine, in July 2013 suddenly developed bilateral ulnar and medial nerve palsy. EMG showed acute denervation. In CSF we found 85 mg of protein and 3 cells. Cervical MRI showed C6-C7 herniation without any medullary or radicular compression. We performed nerve biopsy: severe loss of myelinated and amyelinated fibers with epineural low grade B-type lympho-proliferative malignancy (B-lymphoplasmocytic or marginal zone NHL). At the osseo-medullary biopsy (OMB) performed repeatedly after demonstrated localization of polyclonal B-type: No NHL was found. After 2 months (October 2013) he developed transient, partial III right cranial nerve palsy: at that time cerebral gad-MRI was normal. One year later (July 2014) he experienced abrupt onset of left thigh pain with sciatic paralysis. A CT-scan performed 24 hours after the onset showed posterior muscular loggia dimensional augmentation (Fig 1), as in the compartmental syndrome. After fasciotomy, performed only 36 hours later, sciatic left palsy persisted. 4 months later (November 2014) the patient developed left finger foot embolism from popliteal aneurysms.

The onco-haematologist suspected that the polyclonal lymphoid infiltration was secondary to crioglobulinemia in HCV+; to draw the diagnosis of B-cell NHL at OMB infiltration should be monoclonal, and it is still not. Therefore no specific therapy was started.

Ten months later (September 2015) the patient developed subacute urinary retention (no obstructive reason was found); we scheduled to perform uroflusometry and spinal cord MRI

Discussion: There are very few clinical reports of compartmental syndrome in course of lymphomas, but when the neoplasm directly infiltrated the muscle. Although the diagnosis of neuroinfomatosis can be made on nerve biopsy, there is still no systemic signs of NHL. After 56 months from clinical onset the cause of muscle and nerve involvement (multi-neuropathy) remains still undetermined.

Conclusion: The actual diagnosis of axonal multiple neuropathy in course of crioglobulinemia HCV-related seems the most probable. No clinical explanation was made for the muscle involvement. The urinary retention is still under investigation. The evolution toward B-cell NHL is still considered very likely.

References:

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