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Significance of Striational Antibodies in the Context of Neuropathy (Choice 1)

Faisal Anwar and
Mark Flemmer

Department of Internal Medicine, Eastern
Virginia Medical School, Norfolk, VA, USA

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A 88-year-old Caucasian male diagnosed with prostate cancer and treated 1 year ago with transurethral resection of the prostate presents with a 3-month history of progressive fatigue/malaise, and ataxia with inability to ambulate. He was previously able to ambulate with assistance. On exam, he had a positive Romberg sign which has been present for years, as well as loss of proprioception and vibration sense in both lower extremities in addition to patellar areflexia and dysmetria. He was on many sedatives, which after holding during hospitalization, did not result in any improvement in his functional status. He was compliant with degarelix for prostate cancer treatment. A bone scan showed uptake in the anterior left 6th rib, anterior left iliac crest, and left ischium suspicious for metastatic disease. A lumbar puncture was performed to assess for chronic inflammatory demyelinating polyneuropathy, which was negative for any suggestive findings. An electromyogram was negative for demyelination but showed chronic axonal degeneration. A paraneoplastic antibody panel was ordered, and anti-striational antibody returned with a high titer (1:7680), with a reference range of <1:60. Other antibodies in the panel (including anti-acetylcholine receptor) returned within their respective reference ranges. Other causes for his neuropathy were ruled out, including metabolic or nutritional deficits, infection, and side effects of culprit medications. Testing for common autoimmune disorders was similarly unremarkable [1].

It has been known that paraneoplastic syndromes may affect any part of the nervous system from the cerebral cortex to the neuromuscular junction and muscle, most often affecting multiple areas. The pathogenesis is still not completely understood; the forgoing notion attributes clinical manifestations to antibody and T-cell responses against nervous system antigens. Specifically, it is the immune response directed against shared antigens ectopically expressed by the cancer and nervous system. In particular to anti-striational antibodies, these are more often detected in the context of myasthenia gravis and thymoma where they share

positivity with anti-acetylcholine receptor antibodies; they may additionally support an underlying autoimmune diagnosis. Furthermore, the striational antibody titer value was directly proportional to the positive predictive value for thymoma. To date, controlled studies investigating the association between striational antibodies and neurological/oncological disease are lacking. Nevertheless, the value of the striational antibody titer in the presence or absence of coexisting paraneoplastic antibodies can help in determining how aggressive to evaluate for underlying oncologic/autoimmune disease. The utility of paraneoplastic antibody screening is confounded by the fact that different antibodies can be associated with the same paraneoplastic neurologic syndrome, and conversely the same antibody may be associated with different syndromes. Nonetheless, in patients with a characteristic or suspicious oncologic history, an otherwise unexplained subacute neurological disorder, and a negative evaluation for other causes of neuropathy, paraneoplastic antibody screening is paramount in diagnosis [2,3].

Corresponding author: Dr. Faisal Anwar

✉ faisal.anwr@gmail.com

Department of Internal Medicine, Eastern
Virginia Medical School, Norfolk, VA, USA.

Tel: 7032207153

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