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ANTI-TNF-ALPHA INDUCED-SYSTEMIC AUTOIMMUNE MYOPATHIES: A CASE SERIES

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BACKGROUND

Anti-TNF-alpha therapies are widely used in vary rheumatic diseases, rarely and paradoxically, can induce autoimmunity, including systemic autoimmune myopathies (SAMs). Since it has been scarcely described, we reported four patients with anti-TNF-alpha induced-SAMs from our tertiary center.

CASE REPORT

Case 1: A Caucasian 38-year-old male with defined ankylosing spondylitis (AS). After 36 months of infliximab therapy, he developed a progressive, symmetrical and predominantly proximal limb muscle weakness (maximum creatine phosphokinase (CPK) of 1351U/L, electroneuromyography with myopathic pattern, muscle edema in thigh magnetic resonance). With a diagnostic of polymyositis (EULAR/ACR-2017), infliximab was stopped, and prednisone and azathioprine were introduced. After two months of this last scheme, there was both AS and polymyositis disease stabilization.

Case 2: A Caucasian 57-year-old female was treated sequentially with infliximab, etanercept, and adalimumab for 48 months. Her diagnosis was arthropathy (axial involvement) associated with inflammatory bowel disease (IBD). However, she developed a progressive proximal limb muscle weakness with CPK of 1268U/L and muscle biopsy compatible with inflammatory myopathy. Other drugs therapies were introduced (glucocorticoid, azathioprine, two cycles of rituximab) without good outcome. For the IBD, certolizumab was used, with no SAM control. Finally, she was treated with azathioprine and cyclosporine with stabilization of the IBD and SAM.

Case 3: A Caucasian 45-year-old male with AS. He had treated for 7 years with adalimumab, etanercept and infliximab. After 13 months of infliximab, he showed skin lesions (heliotrope and Gottron's papules), mechanic's hand, arthritis, without elevation of CPK. The perivascular lymphocytic dermatitis was observed in skin lesion biopsy. As a dermatomyositis (EULAR/ACR-2017), methotrexate, leflunomide and prednisone were introduced, but without good outcome. Finally, he received rituximab, achieving disease remission.

Case 4: A Caucasian 39-year-old female patient with rheumatoid arthritis. Because of refractory disease to various immunosuppressive drugs, received adalimumab. However, after two doses, she developed a progressive, symmetrical and proximal limb muscle weakness with CPK of 15000U/L, interstitial pneumopathy, and positive to anti-Jo-1 autoantibody. With a hypothesis of antisynthetase syndrome, she used different drug schemes (azathioprine, rituximab, abatacept, tocilizumab), but without success she died.

CONCLUSION

All patients undergo anti-TNF-alpha medications should be carefully evaluated and monitored continually for the potential development of diverse patterns of SAMs. Responsiveness and prognosis of the established treatment differed in each case. These agents should be used with caution in patients with preexisting autoimmune diseases and with induced-SAMs.