TA is caused by vasa vasoritis leading to infiltration of media by inflammatory cytokines thus causing disruption of elastic fibres and facilitating formation of IMH. No doubt HTN also contributes into fragile media.

Conclusion—Based on the above we opted for switching this patient’s immunosuppressants from MTX to Mycophenolate Mofetil and to consider further escalation to biologic therapy in case of future radiological progression.

P116
Contribution of anti-ferritin antibodies to the diagnosis of giant cell arteritis

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Introduction—The diagnosis of giant cell arteritis (GCA) can only be ascertained by performing temporal artery biopsy (TAB). Recently, Baerlecken et al. [1] reported on the detection of antibodies directed at the human ferritin heavy chain (FTH1) in 92% of patients with GCA vs 1% of healthy controls. We decided to evaluate the diagnostic value of anti-ferritin antibodies in patients undergoing TAB for a suspicion of GCA.

Methods—We included 122 consecutive patients suspected of GCA. Blood sampling was performed at the time of TAB. Sera from 40 healthy individuals served as negative controls. We investigated for the presence of IgG directed against 19 FTH1 amino acids by using an ELISA test. Correlations between FTH1 antibodies and clinical manifestations were investigated using non-parametrical tests.

Results—Anti-FTH1 antibodies were identified in 72.5, 41.3, 31.9 and 2.5% of patients with TAB+ GCA, TAB- GCA, GCA controls and healthy individuals, respectively, with a threshold at 2 SD. By grouping TAB+ GCA, TAB- GCA, GCA controls and healthy individuals, respectively.

Conclusion—We therefore confirm the presence of anti-FTH1 in 72.5% of patients with histologically proven GCA. However, the detection of anti-FTH1 is not contributory to the diagnosis of GCA in a cohort of patients with suspected ACG. The ability to identify a specific sub-group

Reference


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P115
Chronic asymptomatic aortic dissection (AD) in Takayasu’s arteritis (TA); how to treat?

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Introduction—AD is extremely rare in TA and only a limited number of reports have been published. Little is known about the management of this rare complication and usually surgery is reserved for Stanford type A. A tight control of blood pressure is advocated for Stanford type B AD. In our case the diagnosis of chronic intramural haematoma (IMH) was made radiologically using serial MRA in a clinically asymptomatic patient. Despite that we changed patient’s immunosuppressive therapy as we believe IMH was caused by persistent low-grade vaso vasoritis.

Methods—A young Indian lady was diagnosed with TA based on a history of claudicant legs and left arm with subsequent angiogram revealing complete occlusion of the left subclavian artery and distal abdominal aorta. She underwent aortobifemoral arterial bypass grafting in 2002 with complete resolution of her symptoms. Histology of the abdominal aorta. She underwent aortobifemoral arterial bypass grafting as we believe IMH was caused by persistent low-grade vaso vasoritis.

Results—She stayed on a low dose of prednisolone and methotrexate (MTX) for nearly a decade. HTN required frequent adjustments of MTX for nearly a decade. HTN required frequent adjustments of immunosuppressants from MTX to Mycophenolate Mofetil and to consider further escalation of therapy despite clinical improvement has major implications for therapy.

Conclusion—The mean score of TADS was 6 indicating marked damage.

Discussion—The apparent incomplete response to therapy despite clinical improvement has major implications for therapy.

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