Methods. – In Vellore, 132 patients with active disease were studied at 0 and 6/12 after therapy with steroids plus mycophenolate. In Lucknow, 46 patients were assessed at 0 and < 12/12 after therapy with steroids plus methotrexate or azathioprine while TADS (Takayasu Damage Score) was also used to assess development of damage.

Results. – In Vellore, ITAS2010 indicated satisfactory suppression of disease activity. However the ITAS.A score indicated continued disease activity. In Lucknow, at follow-up, the ITAS-A was higher than 2 in 79% of cases and only one in four had a value less than 1, even at 12 months. The mean score of TADS was 6 indicating marked damage.

Discussion. – The incomplete response to active induction therapy with persistent disease activity despite clinical improvement noted for ITAS.A was seen in two centres using different immuno-suppressive plus steroid regimes. Persistent activity would predict development of damage and indeed significantly elevated TADS scores were seen.

Conclusion. – ITAS.A, combining clinical data plus acute phase response, provides new information. The apparent incomplete response to therapy despite clinical improvement has major implications for therapy.

http://dx.doi.org/10.1016/j.lpm.2013.02.185

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 AD. 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 immuno-suppressive 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 aortic wall specimen obtained during the operation confirmed TA.

Results. – She stayed on a low dose of prednisolone and methotrexate (MTX) for nearly a decade. HTN required frequent adjustments of antihypertensive therapy and she has always had a mildly elevated CRP 10–25. Serial MRA from 2008 to 2011 showed slowly increasing thickness of the posterior wall from aortic arch down to descending thoracic aorta. Subsequent CT angiogram confirmed IMH starting from left subclavian artery following spiral dissection to the level of abdominal aorta at L3-L4 just before the graft (pic 1–2).

Discussion. – IMH is a variant of AD caused by ruptured vasa vasorum into the media wall. We hypothesise that fragility of the aortic media in TA is caused by vasa vasoritis [1] 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 immuno-suppressants from MTX to Mycophenolate Mofetil and to consider further escalation to biologic therapy in case of failure.

Reference
http://dx.doi.org/10.1016/j.lpm.2013.02.186

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, Baeberlein 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, 39, 31.9 and 2.5% of patients with TAB+ GCA, TAB- GCA, GCA controls and healthy individuals, respectively, with a threshold at the mean of healthy controls + 2 standard deviations (SD). With a threshold at the mean of healthy controls + 3 SD, anti-FTH1 antibodies were identified in 60, 34.5, 21.2 and 0% of the patients with TAB+ GCA, TAB- GCA, GCA controls and healthy individuals, respectively. By grouping TAB+ GCA, TAB- GCA patients with a threshold at 2 SD, the positive and negative likely ratios were at 1.96 and 2.5% of patients with TAB+ GCA, TAB- GCA, GCA controls and healthy individuals, respectively. Positive and negative likely ratios were at 1.96 and 0.58, respectively. In addition, in our population, anti-FTH1 antibody titer correlated significantly with CRP and no correlation was found with aortic and/or visual impairment.

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 AFG. The ability to identify a specific sub-group