Vascular involvement may occur in about 20% of patients with Behçet’s syndrome (BS) at initial presentation, however, this may reach up to 40% during the follow-up in the same cohort (1). The bulk of this involvement is formed by venous disease which usually manifests with thrombosis (2). Lower extremity veins, vena cava and dural sinuses are among the most commonly affected. Less commonly affected sites such as right side of the heart and pulmonary arteries can also be considered as venous structures. Retinal vasculitis which occurs in about 50% of patients with posterior uveitis also primarily involves the retinal veins. Not only are the veins the primary target in BS, but they also have this important characteristic of occurring in association (2). This may all be at the same time or accumulating one by one with each relapse. Ultimately, it is not unusual for a BS patient to have chronic thrombosis in the leg veins and vena cava inferior and fresh aneurysms in the pulmonary arteries. Although, we are quite aware of the serious morbidity and mortality caused by the vascular involvement, we are unable to predict its development or tell whether there will be relapses or not. Currently, apart from knowing their relative high frequency among young males, there are no clues to further guide us.

In this issue of Clinical and Experimental Rheumatology, Ambrose et al. (3) investigates the capability of magnetic resonance imaging (MRI) in evaluating vein wall thickness of patients with BS. They study thickness and signal enhancement of the popliteal vein by two blinded, experienced observers among 5 BS patients, 4 of whom have a previous history of vascular involvement, and 7 healthy controls. This is a small pilot study with several points to be clarified such as i. what kind of venous thrombosis the patients had; ii. whether popliteal vein had been involved previously, and iii. what was the correlation with ultrasonography. Nevertheless, the study is interesting in that MRI may have the potential to detect increased vein wall thickness in BS subjects compared to healthy controls, acting as a surrogate marker for vessel wall inflammation. Thus far, imaging of venous involvement depended mainly on detecting the intraluminal thrombus formation.

Vessel wall MR imaging has also been applied to the aorta, the carotid artery and the peripheral arteries (4). To depict the vessel wall the blood signal needs to be suppressed (“black blood” [BB]). BB imaging is commonly performed using double inversion recovery (DIR) preparation or spatial saturation (SPSAT) of upstream blood followed by data acquisition such as fast spin echo (FSE) readout. DIR and SPSAT blood suppression rely on the inflow of blood with nulled signal into the imaging volume. Their effectiveness is therefore a function of volume thickness, blood velocity and flow pattern (4). These techniques provide excellent blood suppression for 2D imaging of arteries with fast blood flow such as the aorta, but may be less effective for 3D volumetric imaging and in the presence of recirculating or slow blood flow such as that observed near the carotid bifurcation and in the venous system. Also slow and laminar flow requires large gradient switching, potentially leading to low wall signal due to eddy current effects. Another issue is the acquisition time which could be approximately 10 minutes that may lead to inherent artifacts such as blurring of images and may potentially affect image interpretation. Also, as the authors stated, the slow-moving blood adjacent to the vein wall might cause an apparent thickening of the walls. As depicted in Figure 2 (3), the vein wall appears thicker in
the anterior aspect of the vessel wall and thinner in the posterior aspect, both in healthy control subjects and the BS patients. This asymmetric appearance in MR is probably the effect of phase encoding direction that was chosen in an anterior to posterior direction. Based on the above problems addressed, the use of this technique in vein wall imaging could be promising but needs to be proven in further studies.

The Ambrose study raises several further questions: Could the vein wall thickness predict future vascular involvement? Can we differentiate thickness from enhancement? Can we tell whether the vessel wall disease is acute, chronic or burnt out? What would be the optimum vein to assess? Could MRI also detect the inflammation in the surrounding tissues of the vein which is often observed by vascular surgeons in BS patients (5)?

References