Currently, patients with an uncomplicated acute type B aortic dissection (ABAD) are treated conservatively. Despite adequate anti-hypertensive treatment, delayed aortic dilatation will develop in 20–50% of patients with uncomplicated ABAD, which can lead to the catastrophic event of aortic rupture. In light of this, some randomized controlled trials have studied the importance of prophylactic thoracic endovascular aortic repair (TEVAR) in uncomplicated ABAD to prevent such complications [1]. These studies failed to show that TEVAR was beneficial in the short term. Recently, however, a more positive long-term outcome after TEVAR has been demonstrated. Therefore, several epidemiologic, clinical or morphological predictors have been studied in recent years to identify ABAD patients at high risk of aortic enlargement, who may benefit from early surgical or endovascular...
intervention. Moreover, various biomarkers such as white blood cells (WBC), C reactive protein (CRP), D-dimmers and matrix metalloproteinases (MMPs) have also been investigated as potential predictors of outcome in patients with ABAD [2].

On the other hand, similar biomarkers are present in the systemic inflammatory response, known as postimplantation syndrome (PIS), which is frequently induced by the endovascular treatment of aortic pathologies (e.g., AAA). This syndrome is usually transient during the early postoperative phase and is characterized by fever, leukocytosis, coagulation disturbances and an increase in inflammatory markers in patients’ blood [3]. Suggested causes for the postimplantation syndrome after endovascular procedures in the aorta include injury to the vascular endothelium during endograft implantation, manipulation with introducers and catheters inside the aneurysmal thrombus, activation of biological mediators by the prosthetic material, thrombosis of the aneurysm sac after the aneurysm exclusion and volume of new-onset thrombus after TEVAR [4, 5].

Gorla et al. [6], in their well-designed and interesting study, investigated the postimplantation inflammatory response after TEVAR for ABADs. The study revealed an incidence rate of 15.8% for PIS with increased values of WBC, CRP, IL-6, fibrinogen and D-dimers. In particular, D-dimers and IL-6 peaked at 24 h, CRP and WBC at 48 h, and fibrinogen at 72 h. All-cause mortality did not differ significantly between PIS and non-PIS patients during the index hospitalization as well as after 4 years. Interestingly, a significant difference was depicted with regard to major adverse events. In a mean follow-up of 4 years, a partially thrombosed false lumen, which led to increased reinterventions, was observed more frequently in the PIS group when compared with the non-PIS group. In spite of the important topic that this study deals with and the interesting findings, the small patient sample size could affect the validity of the results of Kaplan–Meier analysis. In addition, in patients with ABAD and aortic rupture or malperfusion syndrome, the pathways involved in an inflammatory response are not totally attributed to the implantation of the endograft. Aortic dissection itself and its complications (e.g. visceral malperfusion syndrome, aortic rupture, limb ischaemia) are linked to an inflammatory cytokine release [7]. Thus, the acute inflammation response triggered by dissection could have led to an overestimation of PIS.

The inflammatory response after TEVAR and its clinical impact on patients’ outcome has not been studied and only limited data are available. In a study by Eggebrecht et al. on patients who underwent TEVAR, a significant increase in WBCs, CRP, fibrinogen and d-Dimers was detected which persisted up to 20 days postoperatively [8]. Another study on PIS after TEVAR detected a significant increase in temperature, WBCs, CRP, IL-10 and IL-6 at 24 and 48 h after endograft implantation, compared with baseline. No significant differences were observed in serum levels of IL-8, TNF-α, creatinine, urea and cystatin C from baseline to 24 and 48 h after stent-graft implantation [9]. In contrast to the study by Gorla et al., no clinical complications related to this postoperative inflammatory response were noted. However, it should be noted that the later study predominantly included patients with thoracic aneurysms and those not with ABAD.

The findings of the study by Gorla et al. raise two additional important points that merit further investigation:

Firstly, the association of alterations in inflammatory response following ABAD endovascular repair with an unfavourable aortic remodelling. The authors described that a partially thrombosed false lumen was observed more frequently in the PIS group when compared with the non-PIS group. Swartbol et al. [10], in a small in vitro study, found that after endovascular repair of abdominal aortic aneurysms (EVAR), the inflammatory cascade is initiated by IL-6 release from aneurysmal thrombus formation. Kakisis et al. [5] evaluated the impact of pre-existing as well as new-onset thrombus on the inflammatory response after EVAR. The volume of new-onset thrombus was associated with the development of PIS after EVAR, whereas chronic mural thrombus appeared to be an inert material [6]. Although both studies underline the association of aneurysmal thrombus formation with the inflammatory response after TEVAR, it is unclear whether thrombosis of the false lumen can be associated with PIS after stent grafting of patients with ABAD. This hypothesis remains to be validated in future studies.

A further important issue is the prognostic value and the clinical impact of alterations in the inflammatory response following TEVAR for ABAD. Studies describing the outcome of patients undergoing TEVAR have not confirmed an association between PIS development and worse outcome [8, 9]. It is, however, well established that partial false lumen thrombosis after TEVAR for ABAD leads to an increased risk of aortic expansion and death. Independent predisposing factors for partial false lumen thrombosis have been suggested to be visceral branches arising from the false lumen, re-entry tears and the maximum diameter of the aortic false lumen [1]. Gorla et al. found an increased incidence of partially thrombosed false lumen, which led to reinterventions, in the PIS group in comparison to the non-PIS group. Other predisposing factors for false lumen patency, however, are not discussed.

In summary, acute aortic dissection and PIS are accompanied by an increase in inflammatory cytokines and coagulation/fibrinolysis alterations, the clinical impact of which is not fully elucidated. The study by Gorla et al. suggests that patients with acute type B aortic syndrome treated with stent graft may develop an increased incidence of multiple adverse events at the 4-year follow-up if the repair is associated with PIS. Further investigation of the pathophysiological interlinks as well as of the clinical impact is needed on a larger number of patients.

REFERENCES