unfavourable long-term functional outcome is very likely for stroke.[12] Inflammation induced by pathogens or of autoimmune origin is particularly harmful to terminally differentiated organs with poor regenerative capacity, such as the heart and brain. Chronic inflammation is thought to impair heart failure, neurodegenerative diseases, and metabolic syndrome.[13]

Interestingly, increased resting heart rate has been linked to cognitive decline after ischaemic stroke.[14] This observation is supported by animal experiments demonstrating deleterious effects of chronic stress on ischaemic brain injury mediated by increased heart rate.[6] Interestingly, damage to the cerebral insular lobe (or 'insula') following stroke or subarachnoid haemorrhage causes autonomic dysfunction and sympathetic overactivation (stress), and may lead to neurocardiogenic damage and troponin elevation.[4,15] Therefore, not only may subclinical myocardial injury (as reflected by elevated cTnT) lead to cognitive dysfunction, vice versa it is also possible that brain injury (associated with cognitive dysfunction) causes cardiac damage.[15]

Finally, we think that an important contribution has been made to the heart and brain interaction. The roles of shared common harmful mechanisms (e.g. autonomic nervous imbalance, inflammation), subclinical (silent) stroke, and white matter disease need to be investigated to uncover the heart of the matter.

Conflicts of interest: none declared.

References

The list of references is available in the online version of this paper.

CARDIOVASCULAR FLASHLIGHT

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Very late bioresorbable scaffold thrombosis after discontinuation of dual antiplatelet therapy

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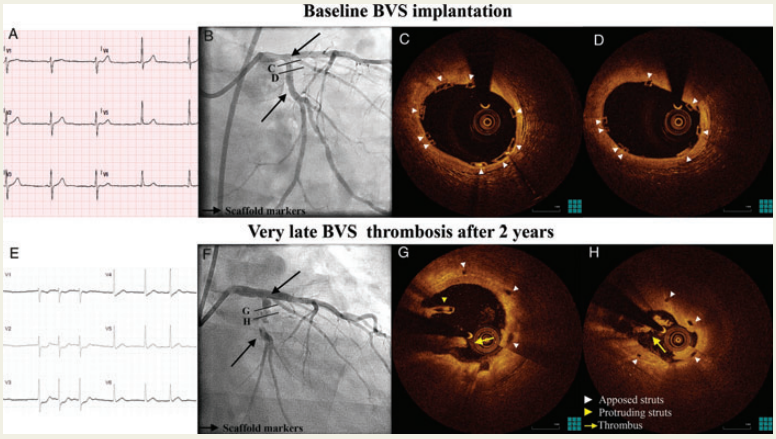
A 57-year-old gentleman was admitted with unstable angina with dynamic ECG changes (E), 4 days after discontinuation of dual antiplatelet therapy (DAT) with aspirin and clopidogrel. He had undergone staged percutaneous coronary intervention with bioresorbable vascular scaffold (BVS; ABSORB 1.1, Abbott Vascular, Santa Clara, CA, USA) implantation in the ostial left circumflex artery (LCx) 2 years ago (*Panels A–D*), followed by everolimus-eluting metal stent implantation in the distal left main and proximal left anterior descending artery, with balloon dilation of the LCx ostium. Fractional flow reserve of the LCx post-intervention was 0.88.



At presentation, angiography showed a filling defect in the scaffolded segment (*Panel F*). Optical coherence tomography revealed intracoronary thrombus and scaffold pattern irregularities (*Panels G* and *H*). The patient was treated with thrombectomy and everolimus-eluting metal stent implantation and discharged on aspirin and prasugrel.

To our knowledge, this is the first reported case of very late BVS thrombosis. Bioresorbable scaffolds have been suggested as an alternative to metal platform drug-eluting stents, aiming to reduce very late stent complications, by providing transient support and being completely integrated into the vascular wall after this period. Although, at 24 months significant bioresorption is expected to have occurred with restoration of local vasomotion, there is still evidence of scaffold struts. In our case, scaffold thrombosis was observed together with scaffold pattern irregularities. The possibility that the observed irregularities were induced by the balloon dilation performed during left main metal stent implantation, subsequently leading to thrombosis, cannot be excluded. Importantly, the event occurred 4 days after DAT discontinuation, suggesting a need for individualized duration of DAT in complex cases treated with BVS.

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