A 66-year-old lady with a background history of hypertension and non-insulin-dependent diabetes was admitted with an episode of confusion and slurred speech and was found to have small foci of restricted diffusion in the deep white matter of the left posterior frontal lobe in keeping with small embolic acute infarcts (Figure 1). As a part of her stroke work up, she was found to have a left ventricular thrombus in spite of preserved left ventricular systolic function. The cause of this thrombus remained uncertain and a targeted work up did not reveal any clear precipitant. She was initially commenced on warfarin anticoagulation that was subsequently switched to low molecular weight heparin due to poor international normalized ratio control.

She presented 2 months later with symptoms of headache and confusion and was found to have a small subdural haematoma in the right parietal region (Figure 2). There was no preceding trauma or local injury. After a thorough risk benefit evaluation exercise (this included a repeat transthoracic echocardiography that confirmed persistence of the left ventricular thrombus) and discussion with the patient, a decision to continue low molecular weight heparin anticoagulation was made.

Six months after the initial ischaemic episode, this lady represented with symptoms of headache and vertigo and a repeat computed tomography scan showed an area of acute right pontine haemorrhage (Figure 3). Her previous subdural haematoma had completely resolved in spite of being on therapeutic anticoagulation. We compared this region of pontine haemorrhage with her previous magnetic resonance imaging (MRI) scan and found that it topographically corroborated exactly with a previously diagnosed pontine microbleed (Figure 4).

While it remains possible that this bleed may have been secondary to ongoing anti-coagulation, there are elements in the presentation that do not support this hypothesis. First, her previous subdural haematoma had completely resolved in spite of her continuing anti-coagulation; secondly the unusual location of the bleed made this less likely. We hypothesize that the presence of an underlying microbleed played a significant role in her presentation and is likely to be attributable to her symptoms.

Discussion

Awareness of modern neuroimaging techniques is vital to recognizing and classifying causes of intracerebral haemorrhage. Cerebral microbleeds are a focus of intense medical research and the clinical significance of these lesions is yet to be clearly defined. These lesions are thought to represent extravasated perivascular haemosiderin deposits and are delineated on gradient-recalled echo T2*-weighted MRI imaging.1 The location of these lesions in either deep matter or brainstem is thought to represent a hypertensive microangiopathic process and cortical locations are believed to be secondary to cerebral amyloid angiopathy.2 Microbleeds are being increasingly detected in patients with ischaemic stroke,3 as putative causes of intracerebral bleeding4 and are being recognized as
a marker for the cognitive deterioration.\(^5\) Due to the inherent difference in MRI protocols and radiologic expertise used to diagnose these lesions, there have been recent developments of scoring systems that are said to have good inter- and intrarater reliability.\(^6\)

The implications of these lesions in terms of future bleeding potential remain uncertain and a number of retrospective and prospective studies have analysed this association in patients with ischaemic stroke\(^7\) and previous intracerebral haemorrhage.\(^8\) In a recent pooled analysis of patients with stroke (inclusive of ischaemic stroke and intracerebral haemorrhage), Lovelock et al.\(^9\) found an increased prevalence of microbleeds in this population group and the prevalence proportionately increased with the intensity of anti-coagulation (odds ratio (OR) 2.8 in anti-thrombotic free patients, OR 5.7 in anti-platelet users and OR 8.0 in warfarin users). In the warfarin-treated intracerebral haemorrhage subgroup, there was a clear excess of microbleeds. This risk, however, was not demonstrated in warfarin users in the ischaemic stroke subgroup.\(^9\)

Prospective data assessing the impact of

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Figure 1. Multiple embolic infarcts on MRI.

Figure 2. Right sub-dural haematoma.

Figure 3. Right Pontine bleed.

Figure 4. Pontine Microbleed.
microbleeds in patients with ischaemic stroke on antithrombotic therapy and concomitant microbleeds have been sparse, and the few published studies point towards an increased risk of intracerebral haemorrhage in this group.\textsuperscript{10} Others point towards an increased risk of ischaemic stroke in patients with pre-existing microbleeds;\textsuperscript{11} however, it must be noted that the study design and sought outcomes have been significantly different in these prospective analyses. Further prospective risk stratification by type of anti-thrombotic therapy remains unclear and it is hoped that the Clinical Relevance of Microbleeds in Stroke (CROMIS-2)\textsuperscript{12} study will shed light on this matter.

For now, we aim to increase awareness of this cerebral small vessel disease marker and highlight its hypothesized risk of cerebral bleeding. We anticipate that this marker will play a dominant role in anti-coagulation risk stratification in the future. Where anti-thrombotic therapy is mandated in the presence of cerebral microbleeds, this hypothesized bleeding risk becomes even more pertinent and as yet remains unquantified.

Conflict of interest: None declared.

References