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Dear Doctor,

Greetings from Micro Labs. We are happy to bring you "Stroke Newsletter" which contains latest and interesting news in the field of Neuropsychiatry.

This issue contains:

1. **Examination of the associations of baseline and 24-h perihematomal edema shape features with 3-month outcomes**
2. **Evaluation of prevalence of stroke-associated pneumonia and its predictors among hyperglycaemic patients during acute ischemic Stroke**
3. **Evaluation of risk factors for dysphagia in patients with acute and chronic ischemic stroke**
4. **Case report on acute cerebral microbleeds detected on high resolution head CT presenting with transient neurologic events**

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Examination of the associations of baseline and 24-h perihematomal edema shape features with 3-month outcomes

Introduction

Shape sphericity at baseline emerged as an independent prognostic factor in acute intracerebral hemorrhage patients

Perihematomal edema (PHE) is defined as increases in water content in the brain tissue next to an intra parenchymal hematoma, and they could be used as a simple imaging indicator or as a potential surrogate marker to evaluate secondary brain injury following intracerebral haemorrhage.[1] PHE volume has been identified as an indicator of prognosis for intracerebral hemorrhage (ICH) patients. However, other groups reported that the post-ICH PHE expansion was more prognostically useful than the baseline PHE volume. Beyond volume it was worth considering other shape features which might hold insights and provide a more comprehensive basis for evaluating the PHE expansion.[2] This study aimed to evaluate the prognostic correlates of different PHE shape features at baseline and 24-h follow-up as well as their interval changes with 3-month outcomes.

Methods

An observational study was conducted based on the retrospective data. This was a multi-centric trial dataset, which included 796 patients, manually segmented ICH and PHE on baseline and follow-up CT scans and extracted 14 shape features. Using Spearman correlation, study investigated the relationship between association of baseline, follow-up, difference (baseline/follow-up) and temporal rate (difference/time gap) of PHE shape changes with 3-month modified Rankin Score (mRS). Study determined if PHE shape features independently predict outcome adjusting for patients' age, sex, NIH stroke scale (NIHSS), Glasgow Coma Scale (GCS), and hematoma volume using multivariable analysis.

Results

After p values ($p<0.001$) were adjusted for multiple comparison, shape parameters related to admission PHE maximum diameters in each of the three planes, major and minor axes, volume,

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surface area, and sphericity at baseline were correlated with 3-month mRS (Figure 1). For 24-h follow-up and baseline (but not the 24-h features themselves) the shape feature difference and temporal rate of changes were also associated with outcome. Baseline sphericity (p=0.047) was predictive of 3-month mRS after adjusting for multiple comparisons. After adjusting for age, sex, admission NIHSS, GCS, treatment, ICH Volume, and Time-to-treatment as covariates, none of the features on 24-h follow up scan, or shape feature difference, or temporal rate of changes were predictive of the 3-month mRS. Multivariable logistic regression revealed that baseline PHE shape Sphericity (p=0.029) and Voxel Volume (p=0.026) were independent predictors of favourable outcome, defined as 3-month mRS 0-to-3, in addition to admission NIHSS (p<0.001), ICH Volume (p=0.002), and age (p<0.001). When follow-up, difference, and rate shape features of PHE were included in a multivariable model, the PHE shape Sphericity (p=0.033), Voxel Volume (p=0.033) at baseline, and maximum 2D diameter Axial (p=0.040) at follow-up were independent predictors of favourable outcome defined as 3-month mRS 0-to-3.

Discussion

In this study, patients with acute supratentorial ICH, it was found that many metrics of baseline PHE size, including volume, maximum diameter along the three orthogonal planes, and major axes diameters as well as shape sphericity were associated with 3-month outcome. It was found that when adjusted for other predictors of outcome, metric of baseline PHE size as well as sphericity were independent predictors of outcome. According to study findings, a less spherical appearance of PHE was linked to a worse prognosis, regardless of baseline symptom severity, age, hematoma size, or PHE volume.[2] Selim M et al. reported that rate of PHE growth was a stronger prognostic factor than baseline PHE volume.[3] Temporal rate and difference between baseline and follow-up CT scans in this study showed a significant correlation with the outcome.

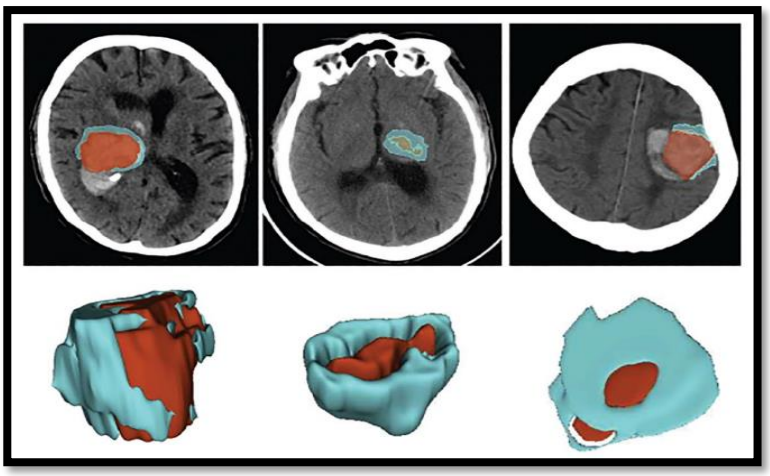

Figure 1. Representation of peri-hematomal edema (blue) sphericity feature

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Conclusion

Finally, shape sphericity became a significant predictive factor in addition to baseline PHE volume with less spherical PHE shape associated with worse 3-month outcome even after adjusting for baseline clinical predictors, hematoma and PHE volumes. PHE shape features could be useful for therapy triage and prognostication for therapies aimed at secondary brain injury.

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Evaluation of prevalence of stroke-associated pneumonia and its predictors among hyperglycaemic patients during acute ischemic Stroke

Introduction

Age above 60 years old and NIHSS > 14 were associated and significantly increased the odds of stroke-associated pneumonia among diabetes patients with hyperglycemia during acute ischemic stroke.

Stroke-associated pneumonia (SAP) is described as pneumonia that develops within the first 7 days following the commencement of a stroke. SAP is a frequent and harmful complication of stroke. Its incidence has been estimated to range from 2.34 to 44%. Hyperglycemia (HG) during acute ischemic stroke (AIS) is defined as random blood glucose > 7.8 mmol/L during the acute ischemic attack and was also linked to SAP.[1] Experimental studies suggested that hyperglycemia might be detrimental to brain structures and metabolism. Hyperglycemia enhance the inflammatory reaction, leading to increased blood–brain barrier permeability, swelling, and bleeding.[2] The purpose of this study

was to evaluate the prevalence of SAP in patients with AIS and predictors of SAP among patients with HG during AIS.

Methods

A retrospective cross-sectional study was conducted among patients with acute ischemic stroke. A total of 412 patients with AIS were included. Diagnosis of SAP was based on the SAP diagnostic criteria by Centres for Disease Control and Prevention. Patients were divided into two groups based on their blood glucose levels: normoglycemia and hyperglycemia. Multivariate logistic regression analysis was conducted using SPSS software, version 22 (IBM Corp., Armonk, NY) to evaluate SAP predictors among patients with HG. The Kaplan-Meier log-rank test was employed to compare the survival rate from unfavourable functional outcomes between hyperglycemic patients with and without SA.

Results

Out of 412 patients with AIS 69 (16.74%) developed SAP. Of the 69 SAP patients, 51 (73.91%) were found to have HG during AIS where 50 (72.46%) of the patients were older than 60 and 40 (57.97%) were males. In this group, 52 patients (75.36%) and 35 patients (50.72%) had pre-stroke diabetes mellitus and concurrent hypertension, respectively. only 19 (27.53%) and 15 (21.73%) of the SAP patients had pre-stroke hyperlipidaemia (HPLD) and ischemic heart disease (IHD) respectively. There was an increase in patients with SAP developed leucocytosis 34 (49.27%). Among the 343 patients in the group that did not have SAP during AIS 165 (48.10%) of the patients with no SAP had DM prior to the stroke. About 93 (27.11%) and 31 (9.03%) of no SAP group had HPLD and pre-stroke IHD respectively. In the SAP and no-SAP groups, the proportion of patients with an NIHSS score greater than 14 at admission was 19 (27.53%) and 39 (11.37%), respectively. In comparison, the final model for the normoglycemic group retained lacunar and pre-stroke

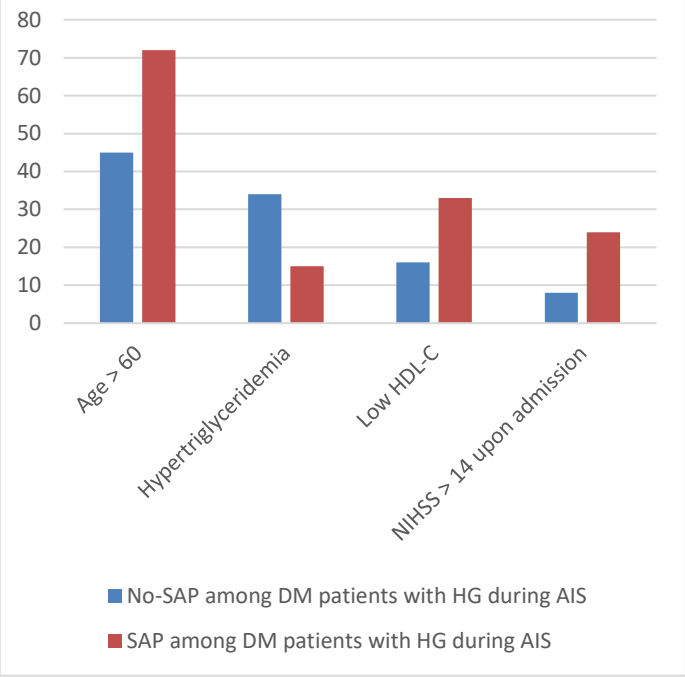

Figure 1. Associated factors with SAP during AIS among patients with prior stroke DM

IHD as significant variables. In the normoglycemic group, lacunar IS significantly reduced the likelihood of SAP with an aOR of 0.17, but pre-stroke IHD significantly increased the odds of SAP with an aOR of 6.23. The risks of SAP among DM patients with HG during AIS were significantly increased for age above 60, in patients developed leucocytosis and NIHSS > 14 (Figure 1).

Discussion

One of the most severe side effects of AIS was SAP, which has a significant mortality rate. The incidence of SAP increased among IS patients with HG in the current study. Hyperglycemic condition reduces the bactericidal ability of leukocytes and, therefore, increases the chances of pulmonary infection.[1] A previous study also reported the result which was consistent with the current study that hyperglycemia was an independent risk factor for SAP.[2] In the current investigation, among patients with HG during AIS, being older than 60years was an independent predictor of SAP. This result was consistent with earlier research showing an association between ageing and an increased risk of post-stroke pneumonia.[4] Furthermore, among patients who experienced HG during AIS, severe stroke (NIHSS > 14) has been found to be one of the SAP-independent predictors. Moreover, leucocytosis was discovered to be an independent predictor of SAP.

Conclusion

Compared to patients with normoglycemia, those with HG had a two-fold higher prevalence of SAP. The variables that were associated with SAP in HG patients during AIS were leucocytosis, NIHSS > 14, and age more than 60. These results highlighted the higher risk of SAP in acute ischemic stroke patients with HG.

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Evaluation of risk factors for dysphagia in patients with acute and chronic ischemic stroke

Introduction

Activities of daily living (ADL) score, rate of pulmonary infection, and Brunnstrom's stages were good predictors of dysphagia among all enrolled participants.

Stroke is one of the primary causes of disability and death globally. Dysphagia is a condition characterized by difficulties in the passage of food or liquid from the mouth through the pharynx, oesophagus, and stomach. Dysphagia may lead to poor dietary intake, dehydration, malnutrition, and pulmonary complications, which can lead to poor prognosis in stroke patients.[1] Dysphagia occurs in 2.3–16.0 % of the general population and 26.19 % of elderly individuals. According to studies, 40% to 60% of people who suffer an acute ischemic stroke have dysphagia, and over 50% of those patients still have the condition 6 months later.[2] This study aimed to evaluate the prevalence and risk factors for dysphagia at different stages following an ischemic stroke.

Methods

A retrospective cohort study was conducted on patients with acute and chronic ischemic stroke. A total of 399 patients with ischemic stroke were included in the study. The International Classification of Diseases, Tenth Revision (ICD-10) was used to identify ischemia infarction in patients using computer tomography (CT) or magnetic resonance imaging (MRI). Water swallowing test (WST) was used for screening of their swallowing function as per standard protocols. To find out how different laboratory, clinical, and demographic characteristics related to dysphagia, statistical analyses were conducted on the obtained data. Multiple logistic regression was used to adjust for potential confounding or risk factor variables. The activities of daily living (ADL) and lymphocyte counts that were used to predict dysphagia were analysed and compared using the area under the ROC curve (AUROC).

Results

Dysphagia was found in 165 of the 399 patients, indicating that 41.4% of cases occurred within 180 days following an ischemic stroke. 72 (36.7%) of the 186 patients in the acute stage and 93 (43.7%) of the 213 patients in the chronic stage, had dysphagia. Patients who experienced dysphagia within 180 days after the stroke had higher NIHSS scores (9.67 ± 6.42 vs 6.78 ±

4.29, P < 0.001), higher NLR counts (4.42 ± 3.44 vs 3.45 ± 2.21, P = 0.001), and lower ADL scores (32.21 ± 24.04 vs 50.20 ± 24.00, P < 0.001). Additionally, aphasia (P < 0.001) and lung infection (P < 0.001) were more common in patients with dysphagia. Individuals diagnosed with acute phase dysphagia exhibited higher pH values (7.42 ± 0.03 vs. 7.41 ± 0.04, P = 0.027), blood platelet (PLT) counts (266.44 ± 89.38 vs. 232.06 ± 65.69, P = 0.045), and diaphragmatic position (P = 0.042); lower lymphocyte counts (1.36 ± 0.47 vs. 1.88 ± 2.35, P = 0.004), and LMR counts (2.58 ± 1.04 vs. 3.08 ± 1.81, P = 0.022). In the acute stage, a significant decrease in Brunnstrom's stages of hand was still observed. In the chronic stage, a significant decrease in Brunnstrom's stages of upper extremities was also seen. Within 180 days of the stroke onset, there was also a significant decrease in Brunnstrom's stages for the hand and upper extremities (Figure 1). Furthermore, among all included subjects, the ADL score, the rate of pulmonary infection, and Brunnstrom's stages (hand and lower extremities) were all highly predictive of dysphagia. Furthermore, the ADL score exceeded the lymphocyte count in terms of predicting value for dysphagia, according to ROC analysis.

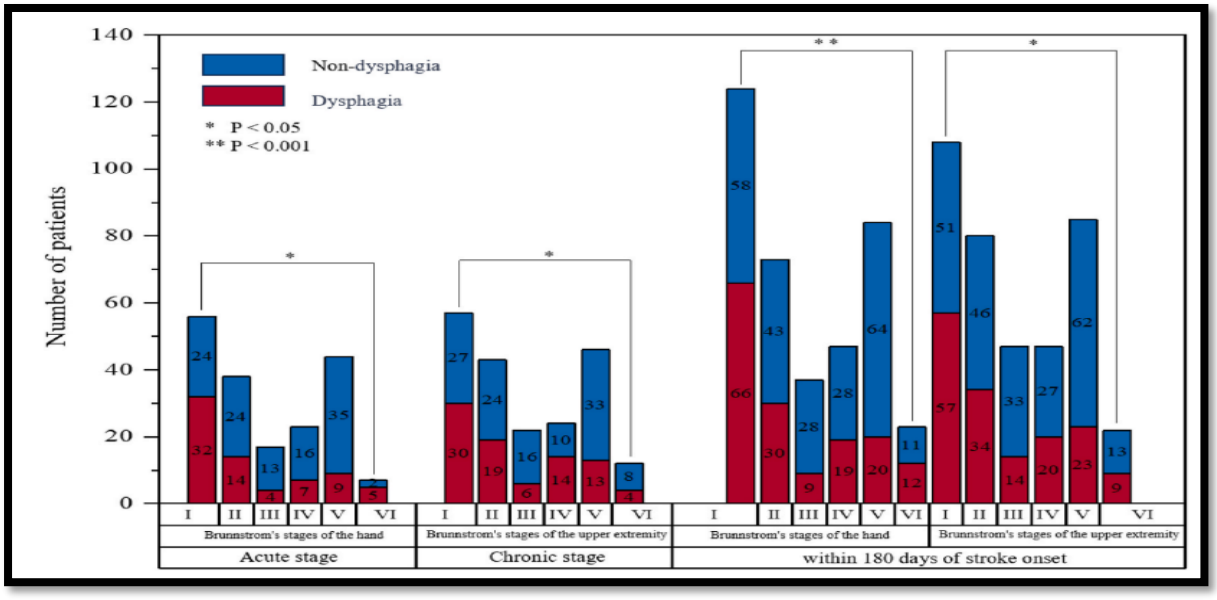

Figure 1. Comparison of all enrolled patients in Brunnstrom's recovery stages, both with and without dysphagia

Discussion

According to this study, the prevalence of acute-stroke dysphagia was 36.7%, whereas the prevalence of chronic dysphagia was 43.7%. Compared to patients without dysphagia, those with post-stroke dysphagia were found to be older (P = 0.002) and to be more likely to have malnutrition (P = 0.011). Furthermore, among all included subjects, the ADL score, the rate of

pulmonary infection, and Brunnstrom's stages (hand and lower extremities) were all highly predictive of dysphagia. Significantly, this study showed that, in all three time periods analysed, the activities of daily living (ADL) score was an independent predictor of dysphagia following a stroke. The prevalence of post-stroke dysphagia was greater in patients with lower ADL scores. In particular, post-stroke swallowing problems that cause feeding difficulties can lead to a deterioration in nutritional status, respiratory infections, generalised muscular atrophy, and eventually severely impair day-to-day functioning. In the scientific community, nutritional status and mRS scores have also drawn more interest.[2] In the last three years, Nayeon et al. have shown that the NIHSS score, the mRS score, and low body mass were independent indicators of dysphagia.[3] Mahmoudinezhad et al. showed that community-dwelling older adults stroke patients with an mRS≥3 had lower nutritional status, smaller midarm circumference, higher NIHSS scores, and were more likely to have comorbid dysphagia.[4]

Conclusion

This study identified the prevalence and risk factors for dysphagia in ischemic stroke patients at different time-windows. In clinical settings, a low ADL score (<40) may be a useful and accurate predictor of post-stroke dysphagia.

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Case report on acute cerebral microbleeds detected on high resolution head CT presenting with transient neurologic events

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Introduction

CMBs are usually asymptomatic, detected incidentally on brain MRI, which is represented by subclinical markers of heightened risk of stroke and dementia.

Cerebral microbleeds (CMB) are a typical MRI (Magnetic resonance imaging) finding that indicate the existence of cerebral microhemorrhage (CMH) in the brains of elderly individuals. It has been demonstrated that CMB was linked to poor functional outcomes for stroke patients, increased risk of ischemic strokes and intracerebral hemorrhage, cognitive decline, and memory impairment.[1] Although CMBs are thought to be clinically hidden and mostly function as a subclinical marker for dementia and stroke risk, recent research has shown that they can manifest as acute symptoms. It was critical to identify acute microbleeds as the underlying cause of acute cerebrovascular syndrome since this may need a different course of treatment, especially when antiplatelet or anticoagulant medication was involved.[2] This report described two cases who presented with symptomatic acute CMBs.

Case presentation

A 70 year's old male patient with the history of diabetes, hypertension, hyperlipidemia and obstructive sleep apnea presented with a transient episode of left upper limb weakness and numbness five days prior. Further, standard unenhanced head CT scan revealed nothing unusual but a minor hyperdensity in the right centrum semiovale/corona radiata, consistent with a small acute haemorrhage, was visible on the high-resolution head CT with coronal re-constructions (1.25 mm slice thickness). Brain MRI revealed the existence of a CMB on T2*GRE weighted images, which was absent on a brain MRI performed three years prior. On the T1 weighted sequence, the lesion was isointense. On the diffusion weighted image (DWI) sequence, it showed hypointense signal encircled by a rim of hyperintense signal. Another report of 82 year's old female patient with the history of hypertension, prior remote large ischemic stroke with residual mild left hemiparesis and vision loss, and post-stroke epilepsy, presented with worsening of her pre-existent left sided weakness. Patient suddenly experienced with brief shaking of the left side followed by paralysis and fall to the ground. Upon examination in the emergency department revealed severe hypertension (blood pressure 200/130 mm Hg), slight left hemiparesis, visual loss consistent with her long-term impairments. An ordinary unenhanced head CT scan revealed nothing unusual, but a high-resolution CT scan with a 1.25 mm slice thickness revealed a small hyperdensity in the right thalamus/internal capsule area that had not been visible in a head CT scan performed two years prior. The lesion seen on the CT scan was in the same place as the CMB seen on the same day's

brain MRI. On DWI, the lesion showed some restricted diffusion. Patient was treated with antiepileptic drugs and antiplatelet therapy.

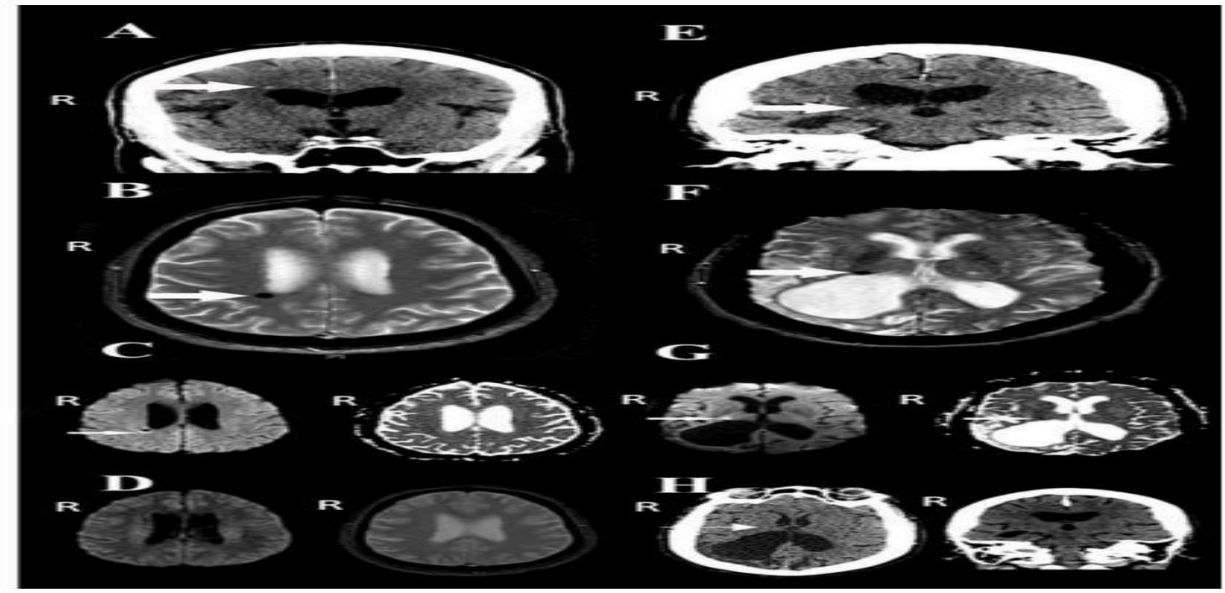

Figure 1. Case 1 images are in the left column (panels A–D) and case 2 in the right column (panels E–H), where A and E represent Coronal reconstructions using high resolution unenhanced head CT demonstrates areas of small acute hemorrhage (arrow). B and F. Axial T2*GRE image demonstrating cerebral microbleeds (arrows). C and G. Axial diffusion weighted image (DWI) in the left and apparent diffusion coefficient (ADC) in the right for each case demonstrating hypointense lesion surrounded by hyperintense rim in case 1 (arrow) and hyperintense DWI signal with corresponding hypointense ADC signal for case 2 (arrows). D. DWI (left) and T2*GRE (right) images predating the appearance of CMB show absence of the lesions for case 1. H. Axial and coronal high resolution (1.25 mm slice thickness) unenhanced head CT predating the occurrence of CMB in case 2 showing absence of the acute hemorrhage (arrow).

Discussion

This case report presented two main findings: first, it showed that breakthrough partial seizures in a patient with controlled epilepsy can be caused by acutely developed CMB, and second, it showed that high resolution unenhanced head CT can detect acute CMBs. The incident manifested as a transient ischemic attack in the first case and focal motor seizure in the second patient. Since both of our patients had a history of hypertension and the CMB was found in deep areas, the underlying aetiology may have been hypertensive vasculopathy. The acute incident and CMB were linked to a hypertensive crisis in the second instance, which improved

with blood pressure management. CMBs were typically asymptomatic and were accidentally identified during brain MRIs. Despite this, they were not quiet since they were subclinical indicators of an increased risk of dementia and stroke.[2] Teo et al. described a case in which a significant intracerebral haemorrhage occurred in the same region as the CMBs after the CMBs first appeared as TIA.[3]

Conclusion

Ischemic events were more likely to occur in patients with CMB. For patients who have previously had CMB, a more individualised approach should be used when selecting the best treatment method.

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