Spontaneous intracerebral lobar hemorrhage

Spontaneous intracerebral lobar hemorrhage was popularized in 1980 after a report delineating 4 clinical syndromes associated with intracerebral hemorrhage in each of the cerebral lobes (occipital, temporal, frontal, and parietal) as in contrast to hemorrhage of deep structures (e.g. basal ganglion, thalamus, and infratentorial structures).

Lobar hemorrhages are more likely to be associated with structural abnormalities than deep hemorrhages.

Lobar intracerebral hemorrhage, is the major clinical manifestation of cerebral amyloid angiopathy.

Epidemiology

Primary lobar hemorrhages (usually due to cerebral amyloid angiopathy) are typically seen in elderly. Younger patients may also develop lobar haemorrhages, but in such cases they usually have an underlying lesion (e.g. cerebral arteriovenous malformation).

Accounts for 10–32% of nontraumatic ICHs.

They may also be more common in patients with high alcohol consumption.

Etiology

Spontaneous intracerebral lobar hemorrhage etiology

Clinical presentation

Patients typically present with acute neurological deterioration, often with decreased GCS.

Headache may be present.

Diagnosis

Lobar hematomas represent around half of all supratentorial hemorrhages and have high mortality and morbidity. Their management depends on the underlying cause. Apart from local causes such as vascular malformation, which are rare and can usually be easily excluded thanks to imaging, the vast majority of lobar hematomas equally frequently result from either hypertensive arteriolopathy (HA) or cerebral amyloid angiopathy (CAA). Distinguishing between CAA and HA is important for prognostication (risk of recurrence nearly sevenfold higher in the former), for decision-making regarding, e.g., antithrombotic therapies (for other indications) and for clinical trials of new therapies. Currently, a non-invasive diagnosis of probable CAA can be made using the MR-based modified

Boston criteria for cerebral amyloid angiopathy, which have excellent specificity but moderate sensitivity against histopathological reference, leading to the clinically largely irrelevant diagnosis of “possible CAA”. Furthermore, the Boston criteria cannot be applied when both lobar and deep MRI hemorrhagic markers are present, a not uncommon situation. Here we propose to test whether new CT and MR-based imaging biomarkers, namely finger-like projections of the hematoma and adjacent subarachnoid hemorrhage on acute-stage CT or MRI, and remote punctate diffusion-weighted imaging ischemic lesions on acute or subacute-stage MRI, have the potential to improve the performance of the Boston criteria. Furthermore, we also propose to test whether clinical-radiological biomarkers may also allow a positive diagnosis of HA to be made in lobar hematomas, which, if feasible, would not only further reduce the prevalence of “possible CAA” but also permit a diagnosis of HA and/or CAA to be made in the presence of mixed deep and lobar MRI hemorrhagic markers [1)].

With large hemorrhages, it may be difficult to distinguish between lobar and deep ICH.

CT

CT is usually the modality first obtained and demonstrates a hyperdense collection of blood, located superficially within the lobes of the brain (i.e., not in the basal ganglia). The haemorrhages vary widely in size from only a centimetre or so (often asymptomatic) to extremely large collections.

Extension into the subdural or subarachnoid and even intraventricular space (the latter is far more common in basal ganglia haemorrhages) may be seen.

CT angiography

It is becoming increasingly used in the workup of patients, not only to assess for an underlying abnormality, but also to evaluate for the presence of a spot, the so-called CTA spot sign, that is indicative of ongoing bleeding. The presence of such a spot sign correlates, not surprisingly, with a growth of the haemorrhage in the first few hours following the scan and is, again not surprisingly, associated with a poor outcome.

CT perfusion

Studies have demonstrated the presence of the spot sign on dynamic-enhancement CT (DECT or CT perfusion) to be an even stronger predictor of hematoma expansion 5-6, i.e. the most robust factor in predicting outcome.

MRI

MRI is usually obtained when concern exists that the bleed if from an underlying region. Findings depend on the size and age of the bleed (see ageing blood on MRI).

In cases of primary lobar haemorrhage, multiple small areas of susceptibility-induced signal drop-out may be evident in keeping with previous micro haemorrhages, suggestive of cerebral amyloid angiopathy (CAA).

The presence of single lobar haemorrhage is still part of the Boston criteria for CAA.

Differential diagnosis

The term lobar haemorrhage is often used to denote a primary haemorrhage. As such the differential includes:

an underlying tumour (e.g. glioblastoma, cerebral metastases) an underlying vascular malformation (e.g. cerebral arteriovenous malformation) haemorrhagic transformation of a cerebral infarct haemorrhagic transformation of the venous infarct

Treatment

see Lobar intracerebral hemorrhage treatment.

Outcome

Lobar hemorrhages may also have a more benign outcome than ganglionic-thalamic hemorrhages.

Case report

A 68-year-old man presented with intracranial hemorrhage in the right frontal lobe, which rapidly increased the day after admission. Arishima et al. performed hematoma removal with a biopsy of the cortex around the hematoma. The day after the operation, a subcutaneous hematoma over the craniotomy appeared, and the computed tomography showed a recurrent hemorrhage with an acute subdural hematoma.

They were aware of a bleeding tendency, and a detailed hematologic examination by hematologists revealed autoimmune acquired factor XIII deficiency due to an antifactor XIII antibody. Specimens taken around the hematomas were pathologically diagnosed as cerebral amyloid angiopathy (CAA) on immunohistochemical examination.

They considered that acquired factor XIII deficiency had induced lobar hemorrhage in the frontal lobe affected with CAA, and the coagulation disorder induced postoperative rebleeding. The patient died from repeated lobar hemorrhage 3 years after the surgery. There is no routine screening coagulation test including the active partial thromboplastin time and the prothrombin time for factor XIII deficiency. It is important for neurologists and neurosurgeons to be aware of this rare disease in patients with a bleeding tendency [2].

[1]

Baron JC, Jensen-Kondering U, Sacco S, Posener S, Benzakoun J, Pallud J, Oppenheim C, Varlet P, Turc G. Can novel CT-and MR-based neuroimaging biomarkers further improve the etiological diagnosis of

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2)

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