An alternative cause of stroke in a young patient with beta-thalassemia

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We describe an interesting case of a young man with subacute stroke on a background of beta-thalassemia. The case demonstrates the need to investigate for cardiogenic embolism, instead of assuming hypercoagulability due to iron overload.

Presentation

A 35-year-old man was admitted with a 4-day history of left sided weakness and blurred vision in the left eye. He had an associated headache and had vomited once, and had initially attributed the symptoms to a migraine, which led to the late presentation. He had a history of beta-thalassemia, splenectomy, and migraines. He was a non-smoker.

Examination:

- Reduced visual acuity in the left eye, and mild left sided ptosis
- Mild left sided weakness (MRC 4/5), worse in the lower limb
- Soft systolic murmur, loudest at the apex

Investigations

- **Bloods:** macrocytic anaemia (Hb 105g/L, MCV 104.5fL), and raised Ferritin (726 ug/L) and iron saturation (82%)
- **Blood film:** target cells, Howell Jolly bodies, and spherocytes (as seen above) in keeping with beta-thalassemia and splenectomy
- **Fundoscopy:** left inferior branch retinal artery occlusion
- **Carotid ultrasound & MR angiography:** normal
- **MRI brain:** restricted diffusion in anterior limb of the left internal capsule and posterior corpus callosum (see below)
- **Echocardiogram:** diffuse calcification (thalassemia-induced cardiomyopathy), and a hypermobile mass in the left ventricular outflow tract

Fig. 1

Diffusion weighted images showing two foci of restricted diffusion in the left corona radiata and corpus callosum.

Fig. 2

Fig. 3

T2 Flair imaging of the same ischaemic foci.

Fig. 4

A sagittal view, with extensive calvarial thickening.

Diagnosis and Management

The patient was diagnosed with left retinal artery occlusion and a left lacunar stroke, caused by cardiogenic emboli due to thalassemia-induced cardiomyopathy.

His left sided weakness was explained by contralateral pressure on the corpus callosum on the right.

He was commenced on anticoagulation and had no further events.

Conclusion

Thalassemia does not in itself predispose to stroke, but hypercoagulability-induced thromboembolism due to iron overload is often accepted as causing cerebral ischaemia in thalassemia.

However, this case demonstrates the importance of considering cardiogenic embolism due to siderotoxic cardiomyopathy. Patients with known thalassemia must therefore have urgent cardiac imaging.

Thrombolysis can be applied in the acute setting, and anticoagulation is appropriate for long term management of a ventricular thrombus.