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Category: Présentation de cas clinique / Clinical case presentation
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Title: **A dwarf overshadowing a giant**

A 75-year-old woman known for hypertension presented to Hospital with acute-onset left hemibody weakness, dysarthria and headache. CT head showed acute right frontoparietal intracerebral hemorrhage, and CT angiography (CTA) showed multiple areas of segmental stenosis and thrombosis in both vertebral arteries. Bloodwork revealed only mild normocytic anemia. Her presentation was attributed to cerebral amyloid angiopathy (CAA), and she was discharged on antihypertensives and prophylactic enoxaparin.
She returned to hospital three days later with acute-onset left-sided vision loss, left hemiplegia and facial twitch. CT head revealed new right frontal lobe intracerebral hemorrhage and posterior right occipital lobe infarct, suggesting a cortical etiology to her blindness. Her presentation was again attributed to CAA. Anticoagulation was held and she was started on levetiracetam. Unfortunately, the following day, her vision loss progressed to bilateral involvement. MRI brain showed extensive acute bilateral occipital and cerebellar infarcts, and raised the possibility of vertebral artery vasculitis based on the previously seen focal stenoses and irregularities bilaterally. Rheumatology was consulted; history was remarkable only for a transient history of jaw claudication three months prior; she denied cognitive disturbances, scalp tenderness, shoulder/hip girdle pain, or constitutional symptoms. Physical exam was unremarkable. Bloodwork revealed elevated CRP(32) and ESR(89); antiphospholipid panel is pending. She underwent PET scan, which revealed extensive uptake in bilateral vertebral, subclavian, maxillary and femoral arteries, consistent with Giant cell arteritis (GCA). She was started on high-dose prednisone, however prognosis remains guarded.

This case highlights several important points. Although stroke is an uncommon manifestation of GCA, with an estimated prevalence of 2-6%, clinicians should be aware of red flags suggestive of the diagnosis. These include bilateral vertebral artery stenoses, involvement restricted to extracranial vessels, and elevated inflammatory markers. Intracranial vessel involvement is rare, explained by the hypothesis that inflammation in GCA involves the elastic lamina in arterial walls, and that intradural portions of arteries have little-to-no elastic tissue, however several cases of intracranial GCA have been reported. Apart from secondary hemorrhage in supratentorial ischemic areas, intracranial hemorrhage due to GCA is also exceedingly rare, with only a few case reports found where hemorrhage occurred in the setting of aneurysmal rupture.

It remains unclear whether our patient's intracranial hemorrhages can be explained by GCA, or whether concomitant CAA acted as a red herring. Several reports of this association have been noted, with the hypothesis that GCA in this setting constitutes a foreign body response to amyloid proteins.