“Amyloid Spells” - An Unusual TIA Mimic

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A n eighty-eight years old gentleman presented to the emergency department with two episodes of left sided paresthesias, grip weakness and slurred speech which resolved completely in two hours. His past history was notable for IHD for which he was on dual antiplatelets. There was no h/o injury to head. The examination was unremarkable save for mild left hemi-hypoesthesia. His MRI showed right fronto-parietal and left anterior frontal Subarachnoid Hemorrhage (SAH) which was blooming on SWI (Susceptibility weighted imaging) images (Figure 1a). FLAIR sequences (Figure 1b) showed hyperintensity in the same areas. Non-enhanced CT brain (Figure 1c) showed high attenuation acute SAH within right frontal region.

In patients presenting with an acute convexity SAH or CSS, recent or prior head trauma needs to be excluded. Reversible cerebral vasoconstriction syndrome occurs most commonly in younger patients and is associated with thunderclap headache. Other differentials for acute convexity SAH are- bleeding diathesis due to thrombocytopenia or coagulopathy, infectious aneurysms, transmural arterial dissection, cerebral venous sinus thrombosis, infectious and noninfectious vasculitis, Posterior Reversible Encephalopathy Syndrome, cavernous angioma and dural arterio-venous fistula.

Cerebral Amyloid Angiopathy (CAA) occurs due to deposition of beta amyloid in small and medium sized superficial cortical and leptomeningeal vessels; vascular disruption leads to micro bleeds, Lobar Hemorrhages (LH) and Cortical Superficial Siderosis (CSS); less commonly the amyloid may cause inflammation of the vessels leading to amyloid angiitis.

Some reports suggest that antiepileptics and migraine preventives may stop amyloid spells. In one small study four out of six patients with amyloid spells responded to antiepileptic therapy alone.¹

References


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