Bilateral Calcified Renal Artery Aneurysms: Not every Calcification is a Calculus!

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A 59 year old postmenopausal lady presented with back pain. She had hypertension, well controlled on a single antihypertensive and a normal renal function. Plain radiograph (Figure 1) showed “ring-like” calcification in the region of her kidneys. CT angiography revealed bilateral calcified saccular renal artery aneurysms (RAA) <2cms in size, arising from the segmental vessels at the hilum (Figure 2). Colour Doppler did not show any evidence of renal artery stenosis and renogram showed bilateral normal perfusion and function. Considering the low risk of complications in this patient, she was managed conservatively and was advised regular blood pressure monitoring and yearly follow up with imaging.

The incidence of RAA is estimated to be around 0.09-0.3% with only a fourth of these being bilateral. Poutasse had described 4 types of RAA: saccular, fusiform, dissecting and intrarenal; with the saccular type accounting for >75%.

Imaging is crucial for the diagnosis of RAA and presence of “ring-like” calcification on a plain abdominal radiograph should alert the physician.

As calcification in RAA is seen in >50% of the cases, further imaging is essential to distinguish it from renal calculi prior to embarking on therapy like extracorporeal shockwave lithotripsy. Angiography being the gold standard, affords a 100% diagnostic rate. However, non-invasive imaging like CT and MR angiography (MRA) are ideal with sensitivity and specificity of MRA being 78% and 100% respectively.

Majority of the RAA are small and asymptomatic but assume clinical significance when associated with hypertension or impending rupture. Factors which may predispose to rupture include size > 2cms, absent or incomplete calcification, uncontrolled hypertension and pregnancy. Endovascular treatment with stenting is the treatment of choice in most cases, with high rates of technical success and negligible complication and recurrence rates.

References

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“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. His coagulation profile, EEG and MRA were normal. The antiplatelets were withheld and he was initiated on Topiramate. There were no such further episodes on follow up after one month.

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.

In patients presenting with an acute convexity SAH or CSS, recent or prior head trauma needs be excluded. Reversible cerebral vasoconstriction syndrome occurs most commonly in younger patients and is associated with thunder clap 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.

CAA related Transient Focal Neurological Episodes (TFNE) can resemble transient ischemic attacks, migraine auras or seizures. These CAA related TFNE are associated with an early risk of symptomatic LH, hence requiring aggressive treatment of hypertension and avoidance of antithrombotic exposure.

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.

References


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