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Original Research Paper

Pathology

COEXISTENT ARTERIOVENOUS MALFORMATION WITH VASCULITIS - CASE REPORT

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ABSTRACT Distinction between arteriovenous malformation and true primary angiitis has therapeutic implications.

KEYWORDS : AV Malformation, Vasculitis, Reactive, Transmural

INTRODUCTION

Arteriovenous malformation (AVM) is an abnormal tangle of blood vessels without a network of capillaries in between. Vasculitis is primary, multifocal inflammation of the vessel wall. Their distinction is important due to therapeutic differences.

Case Study

A 22-year-old female presented with generalised tonic clonic convulsions and holocranial headache for two months. Blood investigations were within normal limits. Digital subtraction angiography (DSA) showed AVM 4.5 x 5.5 cm having feeders from right middle cerebral artery and recurrent artery of Heubner. Histopathology revealed AVM (Figure 1 and 2). Intervascular areas showed siderophages and reactive astrocytes.)

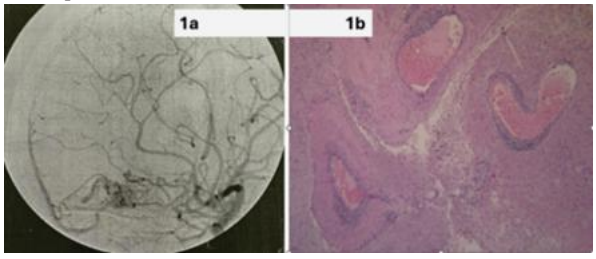


Figure 1a- DSA Image of Arteriovenous Malformation, 1b- Varying Calibre Vessels of Varying Wall Thickness

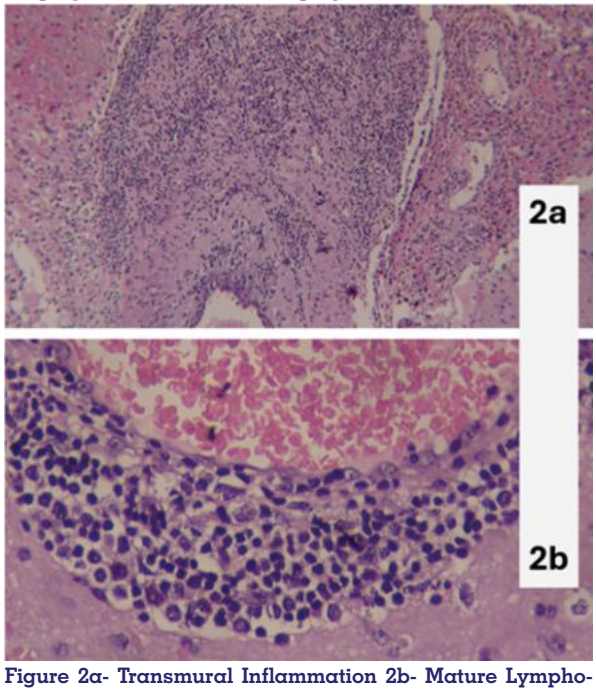


Figure 2a- Transmural Inflammation 2b- Mature Lymphocytes and Plasma Cells.

The patient did not recur following surgical resection. The clinicians were advised to investigate for lupus anticoagulant and acute phase reactants, in view of female gender and young age of the patient.

True primary Vasculitis (PACNS) involves multifocal inflammation of the vessel wall, often requiring long-term immunosuppressive therapy. It is sub classified as large vessel vasculitis (Takayasu, Giant cell arteritis), medium-size vasculitis (Kawasaki, polyarteritis nodosa) and small vessel vasculitis (Wegener's granulomatosis, Behcet syndrome).

When AVM ruptures, the neuroparenchyma reacts with perivascular inflammation. This can be mistaken for PACNS. After AVM is treated by surgery, embolization, or radiosurgery; the inflammation typically resolves.

AVM lacks a normal capillary bed, causing high-pressure arterial blood to surge directly into fragile veins. This hemodynamic stress triggers a localized response of neutrophils and macrophages. They release pro-inflammatory proteins (IL-6, and TNF-alpha)

In rare cases, secondary cerebral angiitis has been documented *after* the surgical treatment of a ruptured AVM. [1,2,3]

In our case, siderophages and gemistocytes suggest prior episodes of rupture. However, the inflammation was transmural with destruction of internal elastic laminae which corresponds better with PACNS.

CONCLUSION

Histological differentiation between these two entities has therapeutic importance. Based on microscopy, we prefer a diagnosis of coexistent AVM with vasculitis.

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