

Peri-hippocampal developmental venous anomalies (DVA) and memory loss: more than a normal variant ?

Letter to the Editor

Developmental venous anomalies (DVAs) are generally considered as normal anatomic variant and may occur across the entire brain. Occasionally, DVAs are associated with cavernomas, and those cavernomas may sometimes induce clinical symptoms including seizure or intra-cranial hemorrhage.

We present 4 cases patients undergoing imaging due to complains of progressive memory loss with peri-hippocampal DVAs. Evidently, this could be a simple coincidence, and the prevalence of peri-hippocampal DVAs in persons without memory loss is unknown.

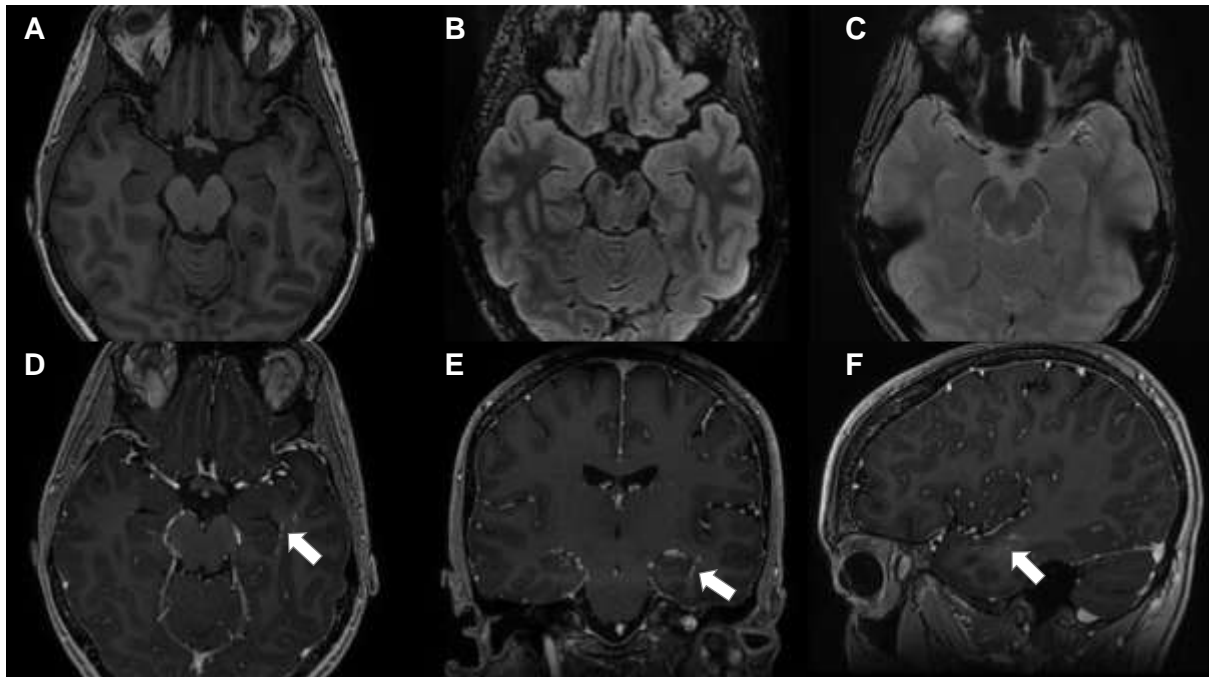
However, we want to formulate the hypothesis that peri-hippocampal DVAs might contribute to memory loss. This might be related to associated lesions, such as micro-cavernomas, which are not always visible on routine clinical MR imaging, yet which might impact the normal functioning of the hippocampus. Or, this could either be related to local alterations of blood flow (1), which might lead to a misbalance of arterial inflow and venous outflow and consequently could accelerate hippocampal neuronal loss. Alternatively, DVAs might lead to subclinical (local) epilepsy, which might interfere with the normal hippocampal function (2).

It is evident that based on the available evidence, we cannot confirm nor reject the hypothesis that peri-hippocampal DVAs might contribute to memory loss. The aim of this letter is to draw the attention of our colleagues to this hypothesis and imaging finding. This is of particular interest because most CT or MR protocols for workup of cognitive decline are performed without contrast administration. As it is very difficult or even impossible to detect notably smaller DVAs without contrast administration, the prevalence of peri-hippocampal DVAs in patients with memory loss might actually be under-estimated, notably if not specifically scrutinizing the scans for this sign.

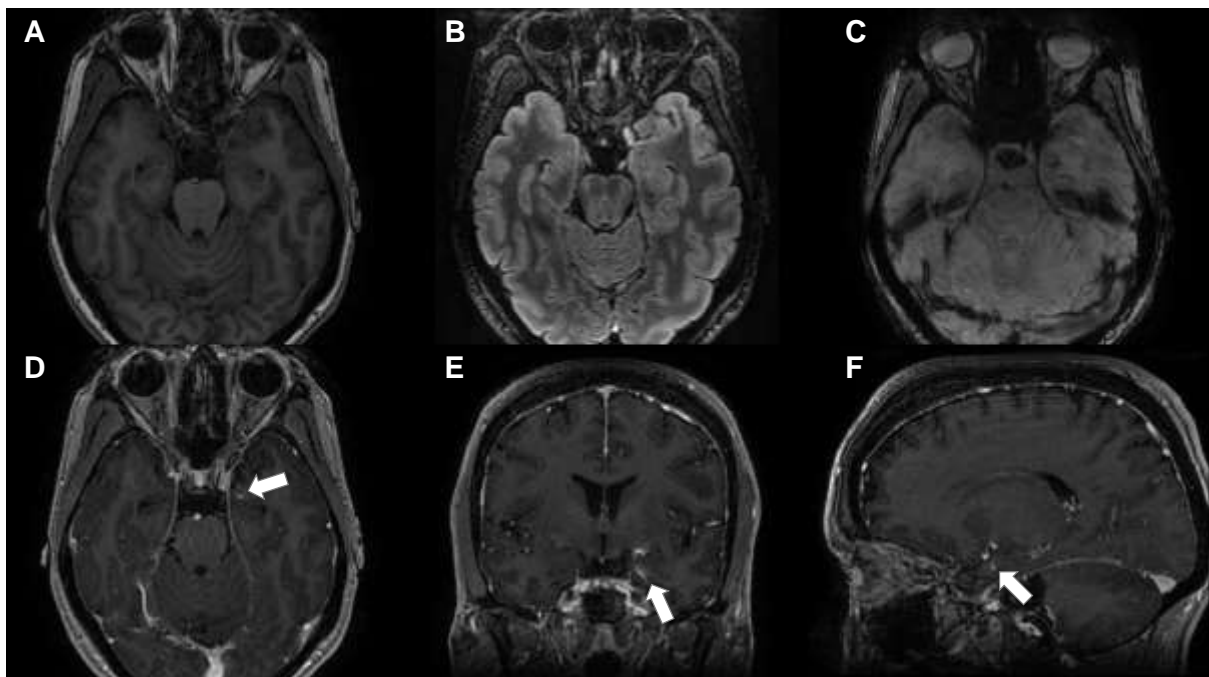
We hope that based on the presented cases, we can raise the awareness in our community of peri-hippocampal DVAs and memory loss, and subsequently create the evidence needed allowing us to confirm or reject the hypothesis of a potential interaction of peri-hippocampal DVA and memory loss.

References

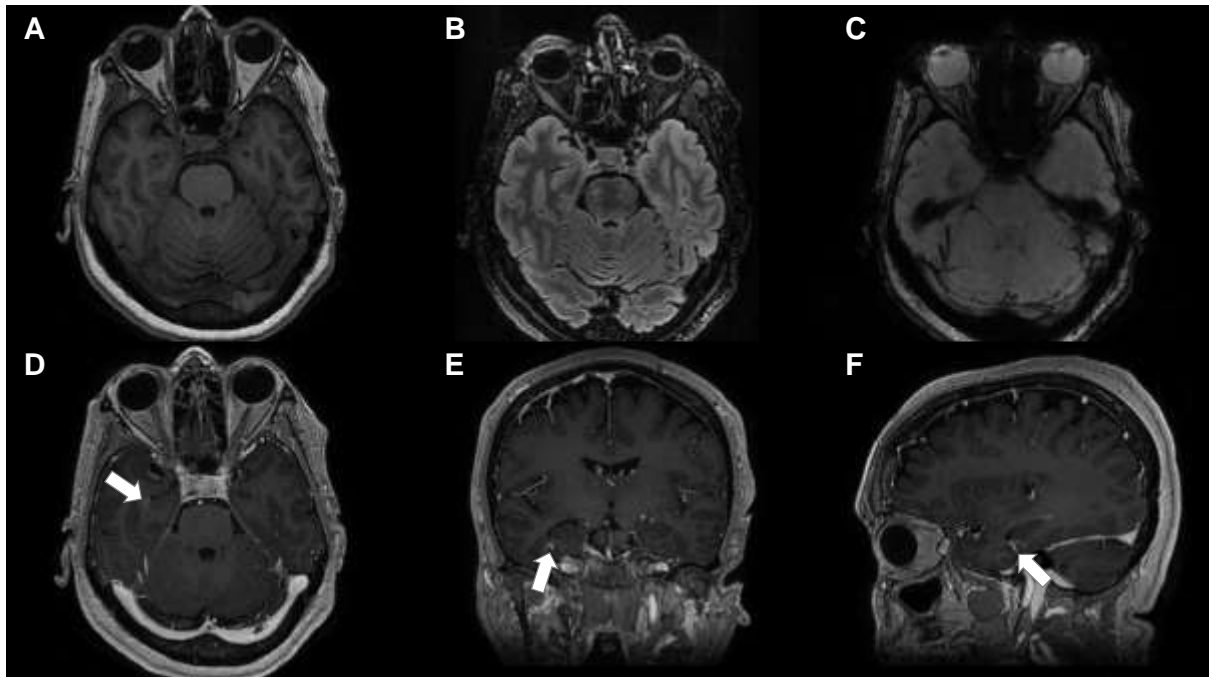
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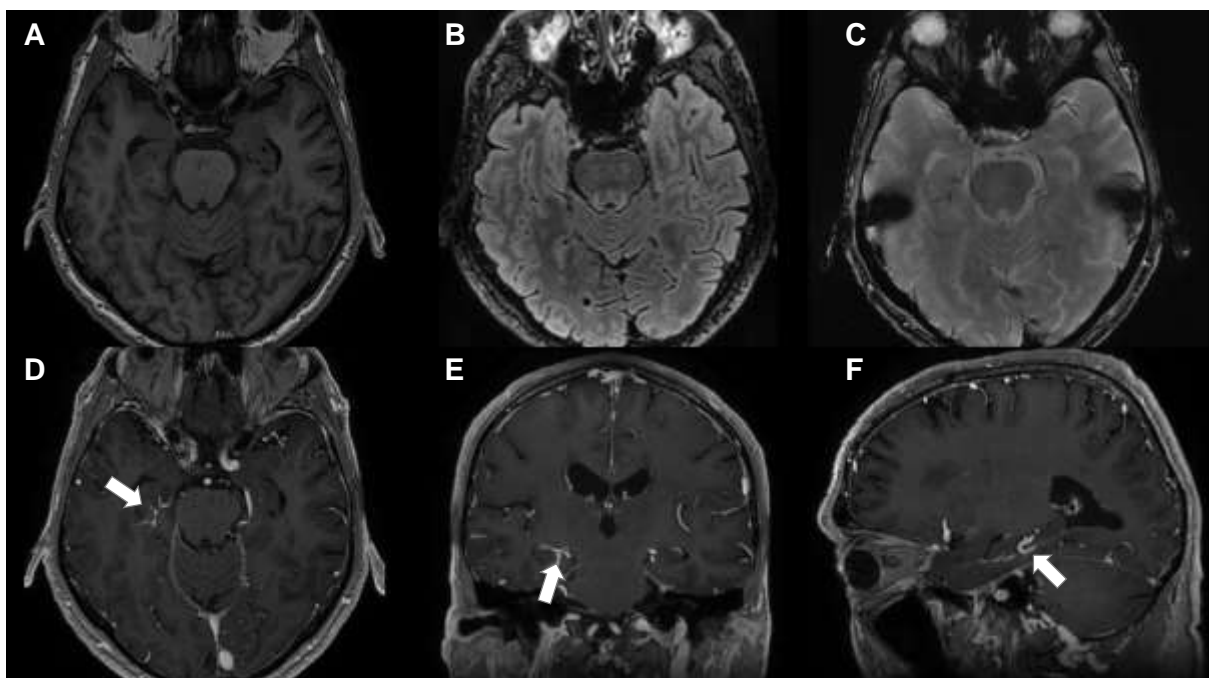
Case 1: 27 years-old female patient with memory loss and sleep disturbance. The left peri-hippocampal DVA is clearly visible on enhanced T1w scans (D-F) yet almost invisible on axial T1w (A), axial T2w FLAIR (B) and axial T2* (C). No significant hippocampal atrophy MTA score 0.



Case 2: 45 years-old female patient with memory loss and vertigo. Similar to the previous case, the left peri-hippocampal DVA is clearly visible on enhanced T1w scans (D-F) yet almost invisible on axial T1w (A), axial T2w FLAIR (B) and axial SWAN (C). No significant hippocampal atrophy MTA score 0.



Case 3: 54 years-old female patient with memory loss and psychotic symptoms since 15 years. Similar to the previous cases, the right peri-hippocampal DVA is clearly visible on enhanced T1w scans (D-F) yet almost invisible on axial T1w (A), axial T2w FLAIR (B) and only hardly visible on axial SWAN (C). No significant hippocampal atrophy MTA score 0.



Case 4: 76 years-old male patient with memory loss. The right peri-hippocampal DVA is clearly visible on enhanced T1w scans (D-F) yet almost invisible on axial T1w (A), axial T2w FLAIR (B) and only hardly visible on axial SWAN (C). Beginning hippocampal atrophy MTA score 2 on right and 1 on left hemisphere.

