Zabramski classification

Cavernous malformations can be grouped into four types based on MRI appearances using the Zabramski classification [1].

The Zabramski classification of cerebral cavernous malformations has been proposed as a way of classifying cerebral cavernous malformations, and although not used in clinical practice it is useful in scientific publications that seek to study cavernous malformations.

The classification was proposed in 1994 , and at the time of writing (June 2016) remains the most commonly used classification of cavernous malformations in the literature.

Based on MRI characteristics:

Type	MRI Features	Description
Type I	Hyperintense on T1 and T2, no hemosiderin rim	Recent hemorrhage with blood degradation products
Type II	Mixed signal intensity on T1 and T2, hemosiderin rim	“Popcorn-like” lesion with chronic hemorrhage and thrombosis
Type III	Hypointense on T1 and T2	Chronic resolved hemorrhage, gliosis, and hemosiderin deposits
Type IV	Only visible on GRE/SWI sequences	Tiny capillary telangiectasia-like lesions (micromalformations)

Type I: subacute haemorrhage

T1: hyperintense

T2: hypo or hyperintense

Type II

: most common type - classic “popcorn” lesion

T1: mixed signal intensity centrally

T2: mixed signal intensity centrally

Low signal rim with blooming on T2* sequences

Type III

: chronic haemorrhage

T1: hypointense to isointense centrally

T2: hypointense centrally

low signal rim with blooming on T2* sequences

Type IV

: multiple punctate microhaemorrhages

T1: difficult to identify

T2: difficult to identify

T2* Gradient Echo: “black dots” with blooming

Difficult to distinguish from small capillary telangiectasias

It is worth noting a couple of point about type IV cavernous malformations.

Firstly, the classification proposed in 1994 by Zabramski predates SWI and as such some authors have suggested that care should be taken in assuming that lesions only seen on SWI (but not on T2* sequences) are type 4 cavernous malformations.

Secondly, how these differ from micro haemorrhages from other causes (e.g. cerebral amyloid angiopathy) is unclear in the absence of genetic evidence of familial multiple cavernous malformation syndrome, as the absence of central T1 / T2 abnormality makes a definite diagnosis of cavernous malformation (in the absence of histology) difficult.

1)
Zabramski JM, Wascher TM, Spetzler RF, Johnson B, Golfinos J, Drayer BP, Brown B, Rigamonti D, Brown G. The natural history of familial cavernous malformations: results of an ongoing study. J Neurosurg. 1994 Mar;80(3):422-32. doi: 10.3171/jns.1994.80.3.0422. PMID: 8113854.