

Spinal vascular malformation clinical features

Spinal vascular malformations can manifest a wide clinical spectrum of symptoms that range from progressive neurological deficits during the course of years to an insidious presentation from acute hemorrhage.

85% present as progressive neuro deficit (**back pain** associated with progressive **sensory loss** and LE weakness over months to years). Yet, **SVMs** account for <5% of lesions presenting as spinal cord “tumors.” 10-20% of SVMs present as sudden onset of **myelopathy**, usually in patients < 30 yrs of age,^{1) 2)} secondary to **hemorrhage** (causing **SAH**, **hematomyelia**, **epidural hematoma**, or **watershed infarction**).

Coup de poignard of Michon = sudden excruciating back pain with SAH (clinical evidence of SVM).

Foix-Alajouanine syndrome (subacute necrotic myelopathy): acute or subacute neurologic deterioration in a patient with an SVM without evidence of hemorrhage. Presents as spastic → flaccid paraplegia, with ascending sensory level and loss of sphincter control. Initially thought to be due to spontaneous thrombosis of the AVM causing subacute necrotizing myelopathy³⁾ which would be irreversible. However, more recent evidence suggests that the myelopathy may be due to venous hypertension with secondary ischemia, and there may be improvement with treatment⁴⁾.

► Clinical. Auscultation over spine reveals a bruit in 2-3% of cases. Cutaneous angioma over back is present in 3-25%; Valsalva maneuver may enhance the redness of the angioma⁵⁾.

Initial symptoms are sensory or disturbances related to gait; later, disturbances in **micturition**, defecation or erection may occur.

Clinically, this presents with progressive neurological dysfunctions that, if diagnosed in a timely fashion, can be at least halted and in part reversed.

see **Aminoff and Logue disability scale**.

The severity of the neurological dysfunction may be predicted by the extent of DSA- and MRI-documented venous congestion and cord edema. There was a strong positive relationship between initial and posttreatment neurological dysfunction⁶⁾.

¹⁾

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²⁾ ⁵⁾

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³⁾

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⁴⁾

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