

Sneddon Syndrome without Antiphospholipid Antibodies

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Figure 1: Sneddon syndrome.

Clinical Image

Madame F, 55 years old with a story; four miscarriages and two fetal deaths in utero. At 40 years old, she had a brutal motor deficit in the left lower limb and then in the homolateral upper limb associated with left hemifacial paraesthesia with complete recovery 10 years ago. She was readmitted for the installation of a left hemiplegia, a left central facial paralysis, a dysarthria with a NIH score of 9 associated with a livedo racemosa, branched, with large irregular open meshes, asymmetric diffusion affecting the legs; thighs, legs and feet. The brain scan shows an infarction of the superficial territory of the right Sylvian artery. On the balance sheet a negative determination of anti-phospholipid antibodies. The diagnosis of Sneddon syndrome is retained, the NIH score rose to 2 under curative anticoagulation followed by secondary prevention. Sneddon syndrome is a rare disease of unknown cause. Its frequency is difficult to estimate and seems underestimated [1]. It is characterized by the association of reticular and cerebrovascular accidents, sometimes responsible for deficits. The other neurological manifestations are polymorphic: headache,

epilepsy, paraesthesia, dizziness, facial paralysis, pseudo-bulbar syndrome, long-term dementia. Biologically, antiphospholipid antibodies (APL) are found in 40% of cases [2].

The occurrence of a stroke in young people, especially in the presence of a racemosalivedo, should suggest the diagnosis of Sneddon syndrome (Figure 1).

Conflicts of Interest

Author declares that there is no conflict of interest.

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