# Neurovascular complication of Hematologic Disorders: Case of Deficits in S and C Proteins about an Observation in Abidjan Ivory Coast

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**Introduction**:
The frequentness of cerebral infarcts associated to a hematologic affection remains difficult to be known and imprecise; it is estimated at 1% as concerns vascular cerebral accidents and between 2% to 16% for stroke of young subject [1,2]. The S and C proteins are proteins normally present in the body. They have anticoagulant role and therefore prevent coagulation phenomena.

We bring here a recent case of cerebral infarcts associated to a deficiency in S and C proteins observed in a young Ivorian patient hospitalized in our neurology service of Cocody Teaching Hospital in Abidjan.

**Results**: The S and C proteins even the antithrombin are coagulation inhibitors. The C protein after activation inhibits coagulation, by degrading the factors V and VIII activated. The S protein participates as co-factor of this degradation. A deficit of 1 of these 3 inhibitors leads to a state of hypercoagulability [3]. These deficits are less frequent in the general population. The results of prospective studies concerning limited cohorts are therefore contradictories. [4-6].There is no justification for practicing systematically the research of a deficit in S and C proteins and of antithrombin, it can be proposed during a cerebral infarcts on a young case when there exists personal and familial past history of thrombotic arteries and or venous evocates or miscarriages or an open foramen oval [3] of which was the case with our patient, who is young of age and had a pulmonary emboli in the past. This checkup is carried out in the absence of antivitamin K treatment for S and C proteins, and the treatment by estrogens for the S protein. In the case of abnormality it has always be controlled on the second sample for antivitamin K reduces the percentage of S and C protein that is why the dosage does not have to be effectuated only 3 weeks after the stop of the treatment. In our patient no anticoagulant treatment preceded the S and C proteins.

A Japanese study concerning 26800 patients presenting the cardiovascular pathology puts I evidence the up came or early rise of the cerebral infarcts in patients presenting a deficit in C protein [7]. A Meta-analysis concerning 18 studies cases/controls put in evidence a possible correlation between the up came of cerebral infarcts and the presence of a deficit in C protein. The cerebral infarcts diagnosis was retained in our patient, in front of a left pyramidal syndrome; neuro vascular image ring. Etiology by deficit in S and C proteins was retained in front of the results of dosage of these proteins with a low blood percentage and absence of other causes which can explain the pathology.

**Conclusions**: The rarity of deficits in C and S protein makes it difficult to put in evidence their implication in the up come of cerebral infarcts. However this etiology in front of all cerebral infarcts is there to privileged in young patient with cardiovascular risk factors and of which the first intention etiology checkup was normal.