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

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Abstract

The frequency of cerebral infarcts associated to a hematologic infection remains difficult to be known and imprecise. The deficit of S and C protein is rarely responsible for cerebral infarcts. We thus bring a young Ivorian patient hospitalized in our neurologic service. She consulted for a left hemi corporal motor deficit of a brutal installation without a particular context. The clinical examinations found a left proportional hemiparesis at 2/5 in superior and inferior members according to the Medical Research Council scale (MRC) and a left painful hemi hypoesthesia. The rest of the examination was without particular. The Neurovascular imagery has objective various cerebral infarcts. Cardiovascular etiologies and hematologic checkups were strictly normal apart from the reduction of the prothrombin percentage (TP=32%). The C protein had reduced (C protein=47% NV>70) and the S protein had equally reduced (S protein=27% NV>60). Other factors of dependent Vitamin K coagulation, (factors II VII IX and X) was also reduce. In front of these results and the absence of other causes which can explain its pathology, the diagnoses of multiple cerebral infarcts due to a deficit in S and C protein had be retained. The patient benefitted a treatment of acetylsalicylic acid 100 mg/day and of vitamin K oral route; a symptomatic treatment and a physical reeducation. The evolution was marked six weeks after by a takeover of walk. A motor recuperation at the level of the left side of the body from 2/5-4/5 and a disappearance of a sensitive disorder of the same side. The rarity of deficits in S and C proteins is difficult to put in evidence their implication in the up come of a cerebral infarcts which can cause or lead to an interest to think in front of all vascular event of a young subject.

Keywords: Cerebral infarcts; C and S proteins; Hematologic disorders; Abidjan

Introduction

Th frequentness of cerebral infarcts associated to a hematologic affectio remains difficu 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]. Th S and C proteins are proteins normally present in the body. The have anticoagulant role and therefore prevent coagulation phenomena.

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

Observation

It concerns a young Ivorian woman aged 48 years who is righter, with a past pulmonary emboli in June 2012, hospitalized in our service for a left hemi corporal motor defici of brutal installation without a particular context. Th clinical examination had found a proportional left hemiparesis score 2/5 at the inferior and superior members according to the medical research council scale (MRC), and a left painful hemi hypoesthesia. Th rest of the examinations were normal.

Th IRM put in evidence hyper signals in sequence T2 and flair and a hypo signal in sequence T1 not respectively raised by gadolinium, of the left posterior, the right profound middle and the right anterior cerebral artery.

Th whole of these injuries were in favor of multiple cerebral infarcts.

Th etiologic cardiovascular and hematologic checkup were strictly normal a part from the reduced percentage of prothrombin (TP=32%).

Th C protein had fallen (protein C=47% NV>70) and S protein had equally reduced S protein=27% NV>60).

Th other factors of coagulation Vitamin K dependent (factors II VII IX and X) was equally reduced.

In front of these results, the multiple cerebral infarcts by defici in the S and C Protein were retained and a secondary prevention treatment was instituted associated to symptomatic treatment and hygiene dietetic measures. Thu the patient benefite from a plaquettery antiagregant (acid acetylsalicylic 100 mg/day and Vitamin K in oral route) and a physical reeducation.

Th evolution was marked six weeks afte by a recovery of walk and a motor recuperation at the level of the left body part from 2/5-4/5 according to the MRC scale.

We were equally noting a recuperation of a pain sensibility of the left body part.

Discussion

Th S and C proteins even the antithrombin are coagulation inhibitors. Th C protein afte activation inhibits coagulation, by degrading the factors V and VIII activated. Th S protein participates as co-factor of this degradation. A defici of 1 of these 3 inhibitors leads to a state of hypercoagulability [3]. Thes deficit are less frequent in the general population. Th results of prospective studies concerning limited cohorts are therefore contradictories. [4-6].Ther is no justificatio for practicing systematically the research of a defici 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. Thi 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 effectuate only 3 weeks afte 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 defici 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 defici in C protein. Th cerebral infarcts diagnosis was retained in our patient, in front of a left pyramidal syndrome; neuro vascular image ring. Etiology by defici 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.

Conclusion

Th rarity of deficit in C and S protein makes it difficu 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 firs intention etiology checkup was normal.

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