

Protein S Deficiency and Ischemic Stroke

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Abstract

The protein S deficiency is an ischemic stroke main risk factor in black young people. The authors report three patients of 37, 45 and 56 years old of age who presented ischemic stroke with hemi body deficit (two lefts and one right). No personal or family history of any cardiovascular diseases and risk factor were found except protein S deficiency with 15

Index terms—

1 Introduction

stroke constituted the principal causes of hospitalisation in African neurological unit. The ischemic strokes are more frequent than haemorrhage, 63% to 80.9%, [1][2][3], with high frequency of multiples infarcts, 13.50% and leucoaraiosis, 58.75%. [2] The main risk factors were high blood pressure, heart diseases, diabetes, infectious and other indeterminate causes. Since the discovered of the protein S, many last studies established it's implication in thrombotic states. The hereditary protein S deficit was found in 12.4% and acquired in 21.5%. Recently, the deficit on fibrinolysis factor or coagulation inhibitor (proteins S and C) were found as the important aetiology of ischemic stroke in young people, with frequency range from 13.8% to 16%. [4][5][6] The authors reported three ischemic strokes with protein S deficiency as the only risk factor in African young people. All patients and their relatives have given informed consent. The study was approved by the local ethic committee. Table ?? show the patients clinical characteristic II.

2 Observation a) Case 1

A 37 years old trader woman with a long personal history of left leg stepped and lameness a year ago due to intramuscular injection came to our visit because of weakness of half right body. Her waist was 1,64m with 60 Kg of weight. The neurological exam revealed half right paresis with 3/5 muscular force in leg and 4/5 in arm, with hyper reflexivity, and Babinski sign. None body sensibility disturbance was noted. All routine blood analysis was normal except low protein S with 15% (normal: 80-130%). Hearts recording and ultrasonic exam were normal. Ct-scan showed left fronto parietal hypo density directed to ischemic stroke. No other risk factor was found. The patient received enoxaparin 0.6mlx2/day, relieved by salicylic acetyl acid 250 mg/day and daily kinesitherapy. After three months of treatment a slight regression of neurologic symptoms was noted with Rankin scale at 2/6. At eight months later the patient died by stroke relapse.

3 c) Case 3

A 56 years old carpenter man, with no personal and family history of cardiovascular diseases came to our observation with four hours of brutal left half body deficit. His height was 1,70m for 58 kg weighted and 130/80 mmHg for blood pressure. Neurological examination revealed, speech disturbance, left hemi paresis predominant on arm and face (2/5 arm, 4/5 leg). The left reflexes were low with Babinski sign, and left half body sensibility disturbed was noted. Routine blood analysis were normal except low protein S at 32% (normal: 80-130%). Hearts recording and ultrasonic performed were normal. Doppler of supraortic vessels was normal. Ct-scan and MRI revealed right thalamus, corona radiata and parietal cortex ischemia with cortical and sub cortical atrophy that directed to ischemic stroke with multiples infarcts. Enoxaparin 0.6ml/day was started and relieved by clopidogrel 75 mg/day associated to daily kinesitherapy. The patient had completely health recovery after six months of follow up.

4 III.

5 Discussion

The main known risk factors of ischemic stroke in young African people are high blood pressure, heart diseases, diabetes, alcohol, tobacco, and lipids. Our b) Case 2 A 45 years old man, with no cardiovascular diseases diagnosed beforehand and other stroke risk factor came to our visit because of rapid left half body deficit and dysphasia. He weighed 70 kg for 1,72m of size and 140/90 mmHg of blood pressure after several controlled. Neurological examination revealed left hemi body palsy. The left reflexes were low with Babinski sign, right half body sensibility was normal. All routine blood analysis were normal except low protein S at 22% (normal: 80-130%). Doppler of supraaortic trunk, electrocardiogram and heart ultrasonic recording were normal. Ct-scan and MRI revealed right internal capsule and lenticular nucleus infarcts. Enoxaparin 0.7ml two times daily was started and relieved by clopidogrel 75 mg/day. A daily kinesitherapy was made. The patient remains with partial deficit recovery after eight months.

IV.

6 Conclusion

The protein S deficiency constitutes an ischemic stroke risk factor in black young people, and must be researched forwards all cerebral infarct to reduce the indeterminate stroke aetiology. A wide prospective study is necessary to measure the real prevalence and frequency of coagulation disturbance factors in our countries, in the setting of stroke prevention. Legend: M: male; F: female; SAA: salicylic acetyl acid.

three patients had not presented any of this main risk factor. It was necessary to make the research of coagulation inhibiting factor as protein C and S. We only make the research of protein S because patients had no much money and this test was not available done in our country. The patients had not forwards used anticoagulant and oestroprogestatif treatment or other treatment that could lead to diminish the protein S level. The deficit in protein S was not acquired in our patients. We did not make the protein S and CT scan control because the needy patients had to pay themselves. We did not also make the patient and their family biological and genetic survey but we retain a constitutional deficit in protein S as the ischemic stroke risk factor in these three patients. The protein S deficiency can be asymptomatic and explained the absence of familial thromboembolic disease that could be related. The deficiency in protein S as the cerebral infarct aetiology is rarely reported in young African people. [4] The estimation of the frequency of the protein S deficient in general population, and in black African in particular is difficult because of the lack of epidemiologic data and the studies were extremely rare. The frequency of protein S deficiency range around 11% to 34% in black African and Caribbean population. [5,7] This large diversity of the prevalence and the frequency must due to population heterogeneity and rare studies. The age of first onset reported range around 15 to 45 years old in patients with ischemic stroke, and the abnormalities seemed to be autosomal dominant transmission. [4,6,8] This high prevalence of protein S deficiency must exist in African countries but still unknown.

1

N°	Age/Sex	Clinical features	CT-scan/ MRI	Protein S level	Treatment	Follow up
01	37 F	Right hemi paresis with 3/5 in leg and 4/5 in arm, high reflex, Babinski sign	Left fronto parietal hypo density	15% (N: 80-130%)	-Enoxaparin 0.6mlx2/day, -SAA 100 mg/day	Rankin scale at 3/6, stroke relapse, died.
02	45 M	-Left half body deficit and speech disturbance, -The right reflexes were abolish with Babinski	Right internal capsule and lenticular nucleus infarct,	22% (N: 60-130%)	Kinesitherapy. -Enoxaparine 0.7ml/day relieved by Clopidogrel 75 mg/day.	Partial recovery after eight months
03	56 M	-Left hemi paresis (2/5 arm, -Right thalamus,		32%	Kinesitherapy -Enoxaparin	Complete health

Figure 1: Table 1 :

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