A Case of Ischemic Stroke in a Patient with Middle Cerebral Artery Stenosis and Iron Deficiency Anemia

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Abstract: Recent studies showed that iron deficiency anemia was correlated with ischemic stroke in patients with carotid artery stenosis. It was also identified as the culprit of ischemic stroke in patients without any acknowledged risk factors for stroke. We presented a clinical case that highlights the importance of the management of anemia as etiologic factor of ischemic stroke, both as primary and secondary prevention.

Keywords: Anemia, cerebral ischemia, intracranial artery stenosis, stroke prevention, uterine fibromatosis.

1. INTRODUCTION

Recent studies showed that iron deficiency anemia was correlated with ischemic stroke in patients with carotid artery stenosis. It was also identified as the culprit of ischemic stroke in patients without any acknowledged risk factors for stroke. The aim of our study was to describe a case of ischemic stroke in a patient with left intracranial artery stenosis and iron deficiency anemia, condition which to our knowledge has not yet been described.

2. CASE REPORT

A 53-year-old right-handed female was reffered to the Neurology Department of the Emergency University Hospital in Bucharest with a history of mild aphasia and an initial computed tomography (CT) of the brain showing an ischemic stroke of the left middle cerebral artery (MCA). Her cardiovascular risk factors were dyslipidemia and smoking (15 pack years). The patient reported metrorrhagia in the past two years, with iron deficiency anemia (IDA) recently diagnosed.

The neurological examination revealed mild anomia and dysgraphia, and was otherwise unremarkable.

On admission, laboratory tests showed moderate mycrocitic hypochromic anemia, hyposideremia, low serum feritin level, normal total iron binding capacity and upper range of normal reticulocyte count (Table **1**). Her leukocyte count, biochemistry panel and reactive protein C value were in the refference range.

Table 1: Laboratory Results Demonstrating Iron Deficiency Anemia (MCV-mean Corpuscular Volume; MCH-mean Corpuscular Hemoglobin; ESR-erythrocyte Sedimentation Rate; TIBC-Total Iron-Binding Capacity; TSAT-Transferring Saturation)

Test	Value
Hemoglobin (g/dl)	7.9
MCV (fl)	66.6
MCH (pg)	19.6
ESR (mm/h)	66
Reticulocyte count (%)	1.5
Serum Iron (mcg/dl)	15
TIBC (mcg/dl)	367
Ferritin (ng/dl)	10.7
TSAT (%)	4.1

Cervical arteries Doppler ultrasonography (Figure **1**), transthoracic echocardiography and Holter electro-cardiography ruled out a large artery atherothrombotic or cardioembolic source.

A brain MRI was performed, demonstrating a T2 and FLAIR high signal region (Figure **2A**), corresponding to a T1 low signal region in the left insular and frontal cortices, markedly in the left middle frontal girus, without diffusion restriction on the SWI, consistent with a late phase of ischemic stroke. The 3D time of flight angiography revealed a low flow within the M2 segment of the MCA (Figure **2B**).

Cerebral CT angiography confirmed a narrowing region of the M2 division of the left MCA suggestive for

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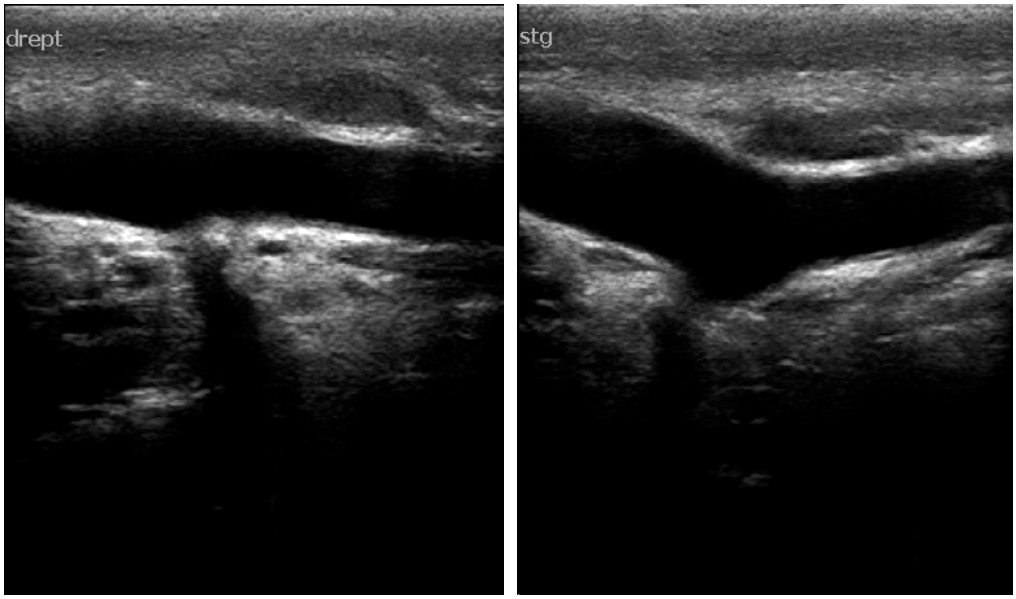

Figure 1: Doppler ultrasonography exam of the cervical arteries.

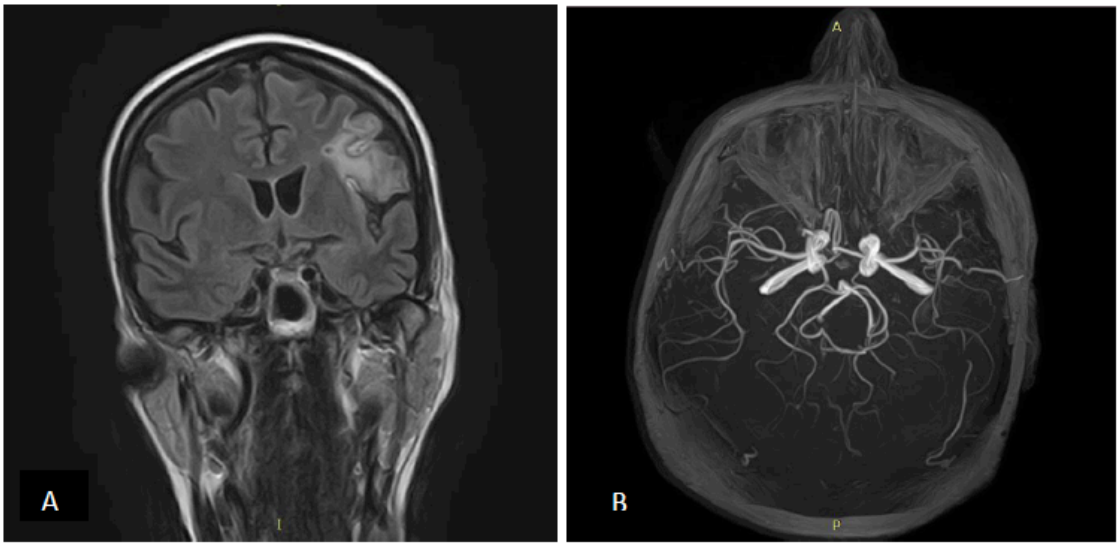

Figure 2A: FLAIR sequence MRI showing high signal of the left frontal cortex. **B**: TOF sequence MRI.

an haemodynamically significant arterial stenosis (Figure **3**) and normal carotid and vertebral arteries (Figure **4**).

A gynecological examination identified diffuse uterine fibromatosis.

In the following days, hemoglobin level markedly decreased, and erytrocyte blood transfusion and intravenous iron supplementation were administered, without discontinuation of the Clopidogrel antiplatelet therapy. The patient didn't experience any neurological symptoms during this period. She was transferred to the Gynecology department for surgical treatment of her uterine pathology.

At 11 months follow-up after the ischemic stroke, the patient didn't present any anamnestic or clinical findings suggestive of recurrent neurological ischemic events and her hemoglobin value was in the refference range.

3. DISCUSSION

A recent populational study describes the positive correlation of ischemic stoke and preexisting iron deficiency in adults [1] and the mechanisms proposed are hemodynamic and hypoxemic tissue damage and secondary thrombocytosis due to iron deficiency [1, 2]. Furthermore, two cases of ischemic stroke without any recognised risk factors for stroke were reported in

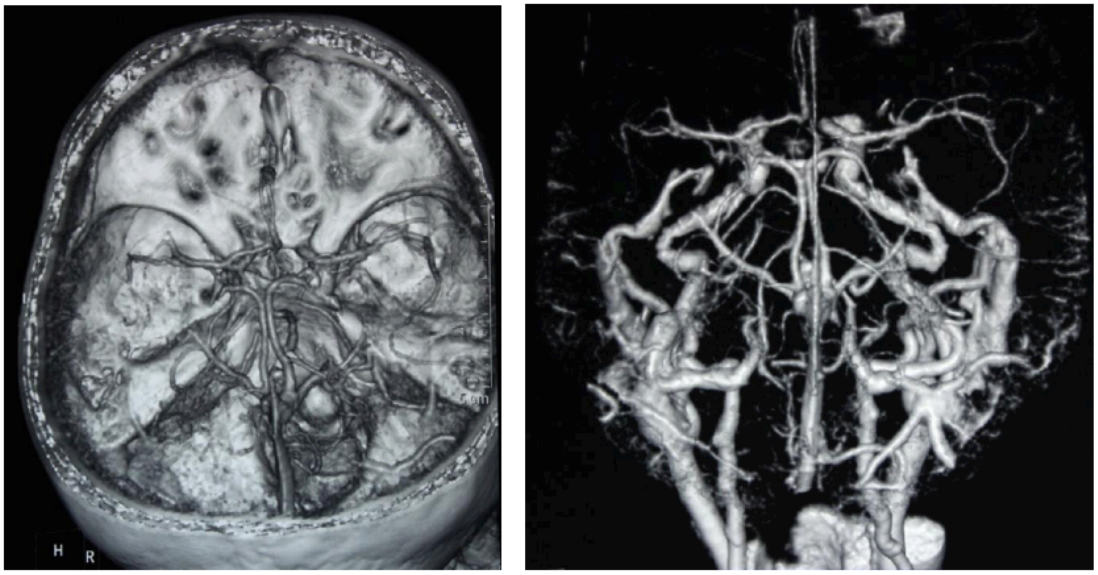

Figure 3: CT-angiography showing left MCA stenosis.

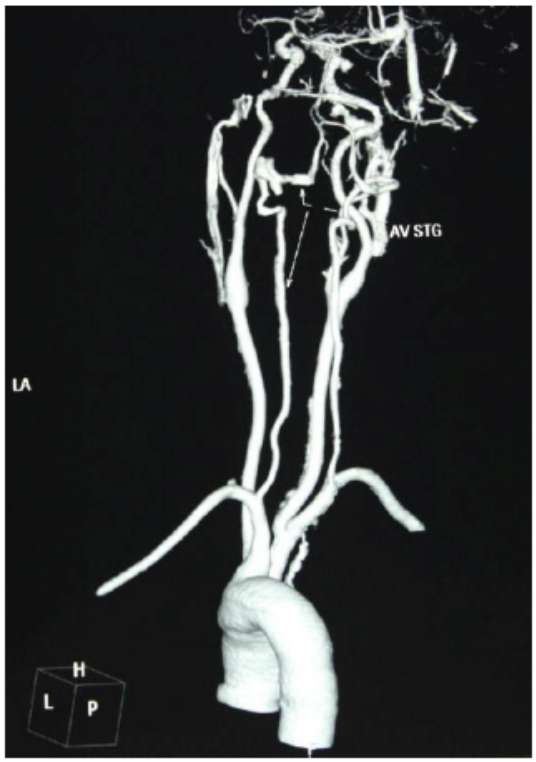

Figure 4: Cervico-cerebral CT-angiography showing normal carotid and vertebral arteries.

patients with IDA related to uterine fibromatosis [2]. Altought our patient did not exhibit an increase of the platelets number, a hypoxemic state due to worsening anemia might have contributed to her acute ischemic neurological damage. Anemia didn't increase the risk of hemorrhagic stroke [1]. The authors stress the importance of IDA management in acute settings [1]. In turn, a low hematocrit was suggested to enhance artery-to-artery embolism in patients with arterial stenoses [3], thus adding further pathophysiological link between anemia and ischemic stroke in our patient.

Studies have shown that the severity of intracranial artery stenosis is positively correlated with the development of collateral supply [4], which contributed to our patient's good clinical outcome.

In patients with intracranial artery stenosis, the risk of developing subsequent disabling ischemic events in

the same territory is high [5] and they were proven to benefit most from dual antiplatelet therapy with Apsirin and Clopidogrel as secondary prevention [6, 7]. Subsets of the SAMMPRIS study showed that endovascular treatment did not provide a benefit [8], instead it increased mortality and stroke events in the first month [9]. However, our patient undergone antiplatelete therapy with Clopidogrel alone, considering her bleeding risk profile.

CONCLUSION

Intracranial artery stenosis were proven to have an increased risk of recurrent ischemic events, which prompts for aggressive medical treatment as secondary prevention. However, there is no consensus of therapy in the literature. In a case where iron-deficiency anemia was a concurrent mechanism, its management is beneficial as primary and secondary prevention.

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