AKINETIC MUTISM FOLLOWING BILATERAL ANTERIOR CEREBRAL ARTERY TERRITORY INFARCTION DUE TO ANEURYSM: A CASE REPORT

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ABSTRACT

Bilateral anterior cerebral artery (ACA) territory infarction is rare localization in stroke which should always prompt a search for an anterior communicating artery (ACoA) aneurysm. The common neurological manifestations are contralateral weakness predominating in the lower extremity, behaviour disturbance, motor inertia, muteness, incontinence, grasp reflex, diffuse rigidity, akinetic mutism. We describe a 38-year-old woman presented with a left sided hemiparesia and decrease of speech for last days. She was a smoker and morbidly obese. She had no any diagnosed disease. Her neurological examination had weakness of left extremeties affected leg more than the arm and akinetic mutism like as no spontaneously speech and move and grasp reflex. CT showed bilateral ACA infarction which included cingulate gyrus, the right side more than left and subarachnoid hemorrhage in the interhemispheric fissure. MRI angiography showed the appearance of ACoA aneurysm. We report a patient with bilateral infarction in the ACA which a rare localization and clinicians must be alert to exist ACoA aneurysm which may bleed, different symptoms and signs like as akinetic mutism, primitive reflexes.

Key Words: Bilateral anterior cerebral artery infarction, aneurysm, akinetic mutism, primitive reflexes.

ANEVRİZMAYA BAĞLI BİLATERAL ANTERİör SEREBRAL ARTER BÖLGE ENFARKTINI TAKİBEN GELİŞEN AKİNETİK MUTİZM: OLGU SUNUMU

ÖZET


Anahtar Sözcükleer: Bilateral anterior serebral arter enfarktı, anevrizma, akinetik mutizm, primitif refleksler

INTRODUCTION

Bilateral anterior cerebral artery (ACA) territory infarction is rare localization in stroke. Less than 3% of all cerebral infarcts involve the cerebral artery territory. Bilateral ACA territory infarction should always prompt a search for an anterior communicating artery (ACoA) aneurysm.
The common neurological manifestations are contralateral weakness predominate in the lower extremity, behavior disturbance, motor inertia, muteness, incontinence, grasp reflex, diffuse rigidity, akinetic mutism. We describe a young woman with bilateral ACA territory infarction.

**CASE**

A 38-year-old right-handed woman presented with a left sided progressive hemiparesia for two days at the hospital. She noted that was admitted to her local hospital with sudden onset severe headache and nausea four days ago. She had high blood pressure (200/100 mmHg) in the emergency department and treated with antihypertensive therapy. Her complains were regressed and the patient was discharged from hospital with medical advice and following of tension at the same day. Over the following 2 days her headache continued with less intensity and no nausea. She examined by family practice, her blood pressure was normal. She was followed without additional antihypertensive therapy following 2 days. The patient started to have paresthesia left sided and progressed to hemiparesia who was admitted to our clinic. She reported no loss of consciousness, convulsions and incontinence. Especially her family noted decrease of speech for last days. She was a smoker and morbid obese (body mass index: 42.5 kg/cm²).

**Figure 1A.** CT scan shows a bilateral symmetrical hypodensity in areas supplied by the ACA which included cingulate gyrus.

**Figure 1B, 1C.** CT scan shows hypodensities were lying through superior frontal gyrus the right side more than left and hyperdensity in the interhemispheric fissure diagnosed as a subarachnoid hemorrhage (white arrow).

She had no any diagnosed disease and medical therapy before. Her blood pressure was very high (190/110 mmHg). Her neurological examination had weakness of left extremities affected leg more than the arm and akinetic mutism like as unresponsiveness with open eyes, no spontaneously speech and move and grasp reflex. Computed tomography (CT) of the brain showed a bilateral symmetrical hypodensity in areas supplied by the ACA which included cingulate gyrus (Figure 1A), hypodensities were
lying through superior frontal gyrus the right side more than left and hyperdensity in the interhemispheric fissure diagnosed as a subarachnoid haemorrhage (Figure 1B, 1C).

Magnetic resonance imaging (MRI) revealed that bilateral ACA infarct right more than left side with oedema. Infarction areas are hyperintense on FLAIR imaging and hyperintensity in the interhemispheric fissure consistent with subarachnoid hemorrhage (Figure 2A). T2-weighted MRI scan showed hyperintense areas in the cingulate gyrus, paracentral lobe, trunk of corpus callosum and medial side of superior frontal gyrus (Figure 2B). Diffusion MRI showed hyperintensity in bilateral cingulate gyrus (Figure 3) and hypointensity consistent with acute ischemia on apparent diffusion coefficient imaging. MRI angiography on admission showed the appearance of AcoA aneurysm (Figure 4). Therefore we planned to perform digital subtraction angiography to treat the aneurysm.

There was no abnormal result of her etiological investigations which included biochemical, coagulation, thrombotic and genetic laboratory tests, electrocardiography, echocardiography, Doppler of carotid and vertebral arteries. During her hospitalization, she was treated with antihypertensive therapy. On her 8th day of admission, she started to talk and move with no induced. The patient’s weakness symptoms were improved particularly on the upper extremity.

**DISCUSSION**

Bilateral ACA territory infarction is usually due to vasospasm that occurs as a complication of subarachnoid haemorrhage caused by the rupture of one or more aneurysms of the AcoA distal ACAs [1]. Simultaneous bilateral cerebral infarction can be the result of a unilateral cerebral artery occlusion and this can potentially mimic a space-occupying lesion. Bilateral ACA occlusion with
resultant frontal lobe lesions, as occurred with our patient can produce akinetic mutism and left hemiparesis which were distinctive symptoms in our patient. Akinetic mutism is a state of limited verbal and motor responsiveness to the environment in those without paralysis. Patient looks apathetic, indifferent, detached and frozen. The patient may also make brief, monosyllabic, but appropriate response to questions. The lesions causing abulia and akinetic mutism often involve the cingulate gyrus, frontal pole/gyrus rectus, supplementary motor area, or the caudate [2,3]. The cingulate gyrus and supplementary motor area lesions as occurred with our patient can produce akinetic mutism. Especially on her 8th day of admission she started to talk and move with no induced. Abulia and akinetic mutism to be 44% after either unilateral or bilateral ACA distribution infarcts [4]. A similar proportion was found in a larger series with these symptoms being most frequent after bilateral lesions (67%) but was not uncommon after left- or right-sided strokes (51% and 25% respectively) [3].

When the arm is affected, this is usually attributed to extension of the ischaemic area to the internal capsule, although in some cases it may also be in consequence of supplementary motor area infarction [5]. As well the patient had weakness of arm, whose MRI showed infarction involvement of supplementary motor area, despite of not including internal capsule. There is often no sensory deficit, but even when present it is usually mild. In our patient we examined had mild sensory deficit, too. Infarction of the frontal lobe or basal ganglia can result in the grasp reflex seen in patients with ACA infarcts. A grasp reflex may be elicited if the motor deficit in the hand is not too great. Our patient had grasp reflex, too who had hemiparesis with predominantly lower extremity weakness than the upper.

We report a patient with bilateral infarction in the ACA which a rare localization and clinicians must be alert to exist AcoA aneurysm which may bleed, different symptoms and signs like as akinetic mutism, primitive reflexes.

REFERENCES