Acute Bilateral Anterior Cerebral Artery Territory Infarction: Case Report

Akut İki Taraflı Anterior Serebral Arter Bölge İnfarktı

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Yazışma Adresi/Correspondence: Hakan Levent GÜL, MD Konya Training and Research Hospital, Clinic of Neurology, Konya, TÜRKİYE/TURKEY leventgul2003@yahoo.com **ABSTRACT** The ratio of anterior cerebral artery (ACA) infarction is 0.3-4.4% of all cerebral infarctions. Simultaneous bilateral ACA infarction is even more rare. Bilateral infarction can result from unilateral occlusion of anomalous cerebral vasculature. We report a rare seen case of acute bilateral ACA territory infarction. Magnetic resonance anjiograms showed the unilaterally hypoplastic ACA starting from A1 segment. Acute bilateral anterior cerebral artery territory infarction can be the result of unilateral cerebral artery occlusion. Cerebral vasculature anomalies may show unusual presentations of cerebrovascular infarctions and these cases are not as rare as believed.

Key Words: Cerebral infarction; anterior cerebral artery; magnetic resonance imaging; abnormalities

ÖZET Anterior serebral arter (ACA) infarkt oranı, tüm serebral infarktların %0.3-4.4'üdür. Eş zamanlı bilateral ACA infarktları çok daha nadirdir. İki taraflı infarktlar anormal vaskülarizasyon gösteren tek taraflı tıkanıklık sonucu olabilirler. Biz nadir görülen iki taraflı ACA bölge infarktını rapor ettik. Manyetik rezonans anjiyografi, A1 segmentinden başlayan tek taraflı hipoplastik ACA-'i gösterdi. Akut bilateral anterior serebral arter bölge infarktları tek taraflı serebral arter tıkanıklığı sonucunda oluşabilir. Serebral vaskülarizasyon anomalileri olağan olmayan serebrovasküler infarktlar şeklinde prezente olabilir ve bu olgular inanıldığı kadar nadir değildir.

Anahtar Kelimeler: Serebral infarkt; anterior serebral arter; manyetik rezonans görüntüleme; anormallikler

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erebrovascular infarction is a common and well-recognised clinical disorder. Anterior cerebral artery (ACA) territory infarction is 0.3-4.4% of all cerebral infarctions. ¹⁻⁴ Simultaneous bilateral infarctions are even more rare. ^{1,2,4} Bilateral ACA territory infarctions are mostly caused by vasospasm after subarachnoid hemorrage of the anterior communicating arteries. Surgical procedures, atrial fibrillation and head trauma are other etiologic factors. But also embolism or thrombosis of the A1 segment and contralateral hypoplasia or aplasia of ACA may result as bilateral ACA territory infarction. ^{1,2,4}

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CASE REPORT

A 66-year-old woman with hypertension and coronary heart disease has admitted to our hospital because of sudden loss of consciousness. She had tetraparesie dominantly at the right side and lower limbs. Plantar responses were bilaterally extensor. Her blood pressure was 160/100 mmHg and electrocardiography revealed atrial fibrillation. Her biochemical parameters were in normal range. Computed tomography was normal (Figure 1). Neurological deterioration continued and akinetic mutism had devoloped during therapy.

MR imaging showed hyperintense lesions in bilateral caudate nucleus, anterior lib of internal capsule, part of putamen and bilateral median frontal lobes (Figure 2).

MR anjiogram showed total occlusion of the right ACA and hypoplastic left ACA starting from A1 segment (Figure 3).

Six months after onset of stroke, control examination was performed. There was no change in the patient's right hemiparesia but a positive progression was observed at the left side. She had still akinetic mutism.

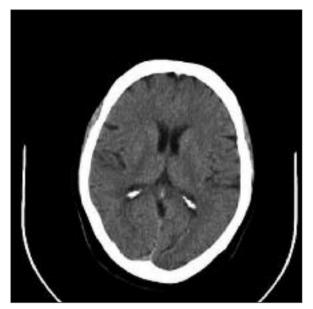


FIGURE 1: Computed tomography was normal.

DISCUSSION

ACA territory infarctions are 0.3-4.4% of all cerebral infarctions. ¹⁻⁵ Cardiogenic embolism or artery to artery embolism can result as ACA territory infarctions according to Bogousslavsky and Regly Gacs et al. reported contralateral or ipsilateral occ-

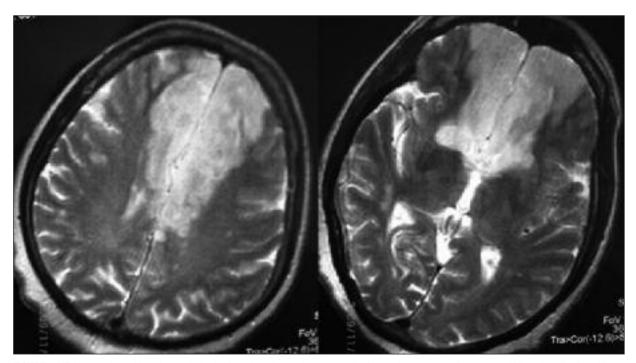


FIGURE 1: MR imaging showed hyperintense lesion in bilateral caudate nucleus, anterior lib of internal capsule, part of putamen and bilateral median frontal lobes.

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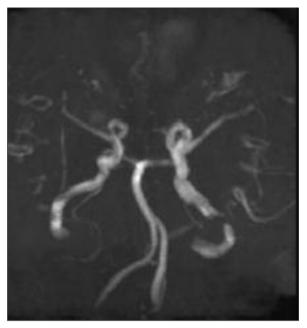


FIGURE 3: MR anjiogram showed total occlusion of the right ACA and hypoplastic left ACA starting from A1 segment of ACA

lusions of the internal carotid artry (ICA), distal extensions of ICA thrombosis and local thrombosis caused by vasculitis as other causes.⁷ Kang SY et al. reported ACA atherosclerosis in 61 patients, cardiogenic embolism in 10 patient and internal carotid artery-ACA embolism in 6 patients among 100 patients as the risc factors of ACA territory infarctions.⁸

Bilateral ACA territory infarctions are rare. Bogousslavsky and Regly⁶ reported 27 cases of ACA territory infarction among 1490 patients of cerebral infarction but only two cases had bilateral ACA territory infarction. Kumral et al. reported two cases of bilateral ACA territory infarction among 48 cases of ACA territory infarction.⁹ Kahilogullari et al. reported a case which had one median trunk of azygos ACA among 16 postmortem cases and they reported that this variation could have the po-

tential of bilateral ACA territory infarction.¹⁰ But we have still no report about the frequency of bilateral ACA territory infarction. Bilateral ACA territory infarctions are generally caused by vasospasm after rupturation of an anevrysm of an anterior communicating artery or distal ACA resulting as subarachnoidal hemorrage.^{1,2}

Embolization to the ACA may occur following some unusual hemodynamic circumstances, such as unilateral ICA occlusion, an azygos ACA, or a hypoplastic A1 segment. In these cases, embolus originating from the heart, aorta or carotid arteries are prone to reach the distal ACAs through the proximal ACA when there is increased blood flow. Bilateral ACA and ICA stenosis, or a small caliber AComA (anterior communicating artery), which could limit distal perfusion can cause unilateral or bilateral ACA territory infarction. The size of the lesions depends on the anatomical patterns of the anterior circle of Willis, the location of arterial boundary zones, and the site of occlusion. 11

As in our case embolism or thrombosis of the A1 segment with hypoplasia or aplasia of contraleral A1 segment may cause ACA territory infarction bilaterally.^{1,2} Unilateral hypoplasia of the A1 segment can be the predisposing factor for bilateral simultenous ACA territory infarction.

Neurological outcome in these patients are usually poor. Paralysis are observed in lower limbs and akinetic mutism can be frequently seen as in our case. 1-3

CONCLUSION

Anomalies of cerebral vasculature are not rare (the incidence of anatomical variations was 26%¹²) and while investigating unusual presentations of cerebrovascular infarctions, we should keep these anomalies in mind.

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