Watershed Cerebral Infarction in a Patient with Acute Renal Failure

Akut Böbrek Yetmezliği olan bir Hastada Watershed Serebral İnfarkt

Böbrek Yetmezliği ile Watershed İnfarkt / Watershed Infarction with Renal Failure

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Özet

Akut böbrek yetmezliği duygu durum değişikliği, konsantrasyon bozukluğu, tremor, stupor, koma, asteriksis,dizartri gibi nörolojik bulgulara neden olabilir. Bu bulgular aynı zamanda serebral infarktın da bir belirtisidir. Burada, kontrast ve anjiotensin dönüştürücü enzim inhibitörü kullanımına sekonder gelişen akut böbrek yetmezliği olan 70 yaşındaki kadın hastada görülen watershed serebral infarkt vakasını sunduk. Hasta dizartri nedeniyle magnetik rezonans görüntüleme ile değerlendirildi. Görüntülemede her iki serebral hemisferin frontal ve parietal derin beyaz cevherinde, iç sınır bölgesini etkileyen watershed serebral infarkt ile uyumlu milimetrik akut iskemik lezyonlar saptandı. Yatışının beşinci gününde böbrek fonksiyonları normale döndü (BUN 32 mg/dl, kreatinin 1.36 mg/dl) ve hasta taburcu edildi. Dizartri yirmi gün devam etti.

Anahtar Kelimeler

Akut Böbrek Yetmezliği; Dizartri; Watershed İnfarkt

Abstract

Acute renal failure can cause neurologic manifestations such as mood swings, impaired concentration, tremor, stupor, coma, asterixis, dysarthria. Those findings can also be a sign of cerebral infarct. Here, we report a case of watershed cerebral infarction in a 70-year-old female patient with acute renal failure secondary to contrast administration and use of angiotensin converting enzyme inhibitor. Patient was evaluated with magnetic resonance imaging because of dysarthria. Magnetic resonance imaging revealed milimmetric acute ischemic lesion in the frontal and parietal deep white matter region of both cerebral hemisphere which clearly demonstrated watershed cerebral infarction affecting internal border zone. Her renal function returned to normal levels on fifth day of admission (BUN 32 mg/dl, creatinine 1.36 mg/dl) and she was discharged. Dysarthria continued for 20 days.

Keywords

Acute Renal Failure; Dysarthria; Watershed Infarction

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Introduction

Acute renal failure can cause neurologic manifestations. Some of those symptoms are mood swings, impaired concentration, tremor, stupor, coma, asterixis, dysarthria, myoclonus [1]. Similar findings can be a sign of cerebral infarct. Here, we report a case of watershed (WS) cerebral infarction in a patient with acute renal failure secondary to contrast administration and use of angiotensin converting enzyme inhibitor.

Case Report

A 70-year-old female patient was admitted to the emergency department with abdominal pain and gastroenteritis which was continuing for one day. She had a history of hypertension and coronary artery disease and was taking perindopril 4 mg/day and metoprolol 50 mg/day for 3 days. She had a coronary angiography 3 days ago. Her medical history included no neurological or cerebrovascular disease. On her physical examination, blood pressure was 100/60 mmHg, pulse 88/min, axillary temperature 37.3 C there were no ralles, S3 or soufflé, bowel sounds has increased. Her laboratory parameters were as below; glucose 139 mg/dl, BUN 61 mg/dl, creatinine 4.51 mg/dl, Na 131 mEq/L, potassium 5.34 mEq/L, calcium 8.35 mg/dl, phosphorus 6.7 mg/dl, ALT 24 IU/L, AST 33 IU/L, amylase 44 U/L, lipase 20 U/L, total protein 5.76 g/dl, albumin 3.7 g/dl, hemoglobin 9.13 g/dl, platelets 423 K/mm3, leukocyte 18 K/mm3, CRP 121 mg/L, PH 7.34, HCO3 25. Examination of urine showed density 1012, protein 0.9 g/L, 4-6 erythrocytes and 2-4 leukocytes. Analysis of stool showed amebic cysts. She was admitted to the nephrology clinic due to acute renal failure secondary to use of angiotensin converting enzyme inhibitor, contrast nephropathy and gastroenteritis. She was treated with amlodipine 10 mg/day, metronidazole 1500 mg/day and hydrated by serum physiologic and dextrose. Diuresis was between 2000-2500 ml /day. Abdominal ultrasonography showed biliary sludge and kidneys were in normal range. On the second day after hospitalization she became dysarthric, uremic encephalopathy was not sought because her BUN and creatinine were in the previous range. Cranial magnetic resonance imaging was performed to exclude cerebrovascular accident. Magnetic resonance imaging revealed milimmetric acute ischemic lesion in the frontal and parietal deep white matter region of both cerebral hemisphere and in the left parietal post central gyrus, which clearly demonstrated WS infarction affecting internal border zone Figure1, Figure 2. Carotid and vertebral doppler ultrasonography showed no occlusion. Acetyl salicylic acid 150 mg/day was started. Her renal function returned to normal levels on fifth day of admission (BUN 32 mg/dl, creatinine 1.36 mg/dl) and she was discharged. Dysarthria approximatelly continued for 20 days.

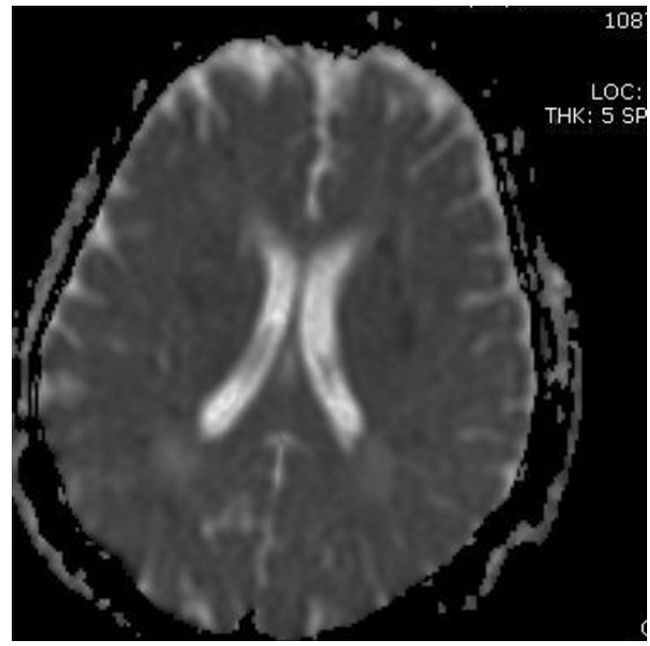


Figure 1. Diffusion-weighted magnetic resonance imaging.

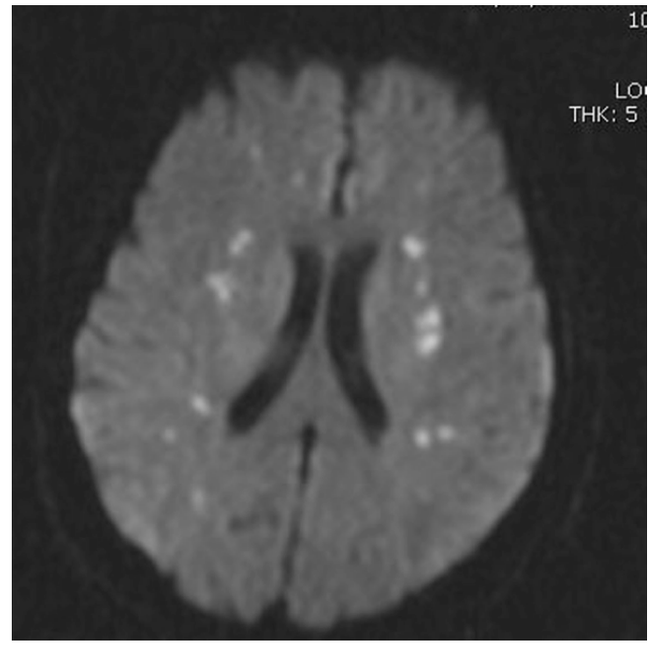


Figure 2. ADC map show multiple acute ischemic lesions in deep white matter region of both cerebral hemispheres

Discussion

Watershed infarctions involve the junction of distal fields of two nonanastomosing arterial systems. There are two distinct supratentorial WS areas. The first between the cortical territories of the anterior cerebral artery (ACA), middle cerebral artery (MCA) and posterior cerebral artery and those have been commonly referred to as cortical WS areas. The second, between the deep and the superficial arterial system of the MCA or between the superficial system of the MCA and ACA and those have been referred as the internal WS areas and it take place in the white matter along lateral ventricule [2]. The pathophysiology has not been fully elucidated. A commonly accepted hypothesis is that decreased perfusion in the distal region of the vascular territories leaves them vulnerable to infarction [3]. Severe hypotension can cause bilateral WS infarction. The symptoms of WS infarcts are syncope, episodic, fluctuating or progressive weakness of hand occasionally associated with upper limb shaking. In our case none of those symptoms were observed, dysarthia was the primary symptom. Our patient was using perindopril 5 mg/day for three days and she had a history of gastroenteritis. After all, episodes of hypotension could be the cause of WS infarction. On the other hand we don't know if she had hypotensive or hypertensive episodes during coronary angiography, which makes more vulnerable the WS infarction areas to subsequent events. It has been previously reported a case

of WS infarction in a hemodialysis patient who had recurrent episodes of intradialytic hypotension [4]. Renal failure can also cause neurologic manifestations such as mood swings, impaired concentration, tremor, stupor, coma, asterixis, dysarthria, myoclonus. Advanced uremic encephalopathy is usually characterized by a reduced level of consciousness, anorexia, asterixis, myoclonus, and upper motor neuron signs that result in disturbances of gait and speech. Twenty percent of patients with acute kidney injury in an intensive care unit setting develop neurologic impairment [1]. The treatment of those patients is renal replacement therapy. But in a patient with acute renal failure and stable renal function, as in our case, a careful search for other causes should be initiated before it is considered a clinical feature of uremia requiring renal replacement therapy.

In conclusion, in case of stable renal function, every patient with renal failure and symptoms of uremic encephalopathy shoud be evaluated for cerebral disorders. We could save the patients from unnecessary procedures and complications

Competing interests

The authors declare that they have no competing interests.

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