doi: 10.5262/tndt.2016.45

Page Kidney After Traumatic Hematoma

Travmatik Hematom Sonrası Page Böbrek

ABSTRACT

Currently, kidney transplantation is considered to be the most outstanding treatment method for end-stage renal failure. Page kidney may be a rare cause of secondary hyperreninemic hypertension. We reported a case with page kidney phenomenon because of subcapsular hematoma proceeding of trauma shrinked spontaneously and renal recovery occurred. This unusual complication of trauma can be seen in renal transplant patient and be in mind when hypertension and hematoma seen together.

KEY WORDS: Page Kidney, Subcapsular hematoma, Hyperreninemic hypertension, Renal transplantation

ÖZ

Günümüzde, böbrek nakli son dönem böbrek yetmezliğinin en seçkin tedavi yöntemi olarak kabul edilir. İkincil hiperreninemik hipertansiyon sebeplerinde Page böbrek nadir görülen sebeplerdendir. Bu olgu sunumunda travma sonrası oluşan ve kendiliğinden resorbe olan subkapsuler hematom sonrası görülen Page böbrek olgusu bildirilmiştir. Bu sıradışı travma komplikasyonu bazı böbrek nakli hastalarında görülebilirken hipertansiyon ve hematom birlikte görüldüğü zaman akla gelmelidir.

ANAHTAR SÖZCÜKLER: Page böbrek, Kapsül altı hematom, Hiperreninemik hipertansiyon, Böbrek nakli

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Received : 19.11.2015
Accepted : 17.02.2016

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INTRODUCTION

Page kidney represents a condition combined of acute hypertension and/or acute disruption of renal function owing to external compression of kidney (1). Hypertension is usually due to renin-angiotensinogen system (RAAS) activation induced by renal microvascular ischemia (2). As a result of this activation, salt and water retention occurs. If there are two functional kidneys, the creatinine level may not typically elevate (3). Otherwise, renal dysfunction might be considered in case of one functional kidney as renal allograft. Thus, we report a case with Page kidney phenomenon caused by a subcapsular traumatic hematoma.

CASE REPORT

A 25-year-old man presented to our hospital with a complaint of pain in his renal transplant region. He had received a living related renal transplantation (his mother) of chronic renal disease for unknown origin. He had remained on hemodialysis for 10 months before transplantation. The post-operative course and recovery was uncomplicated, he discharged on seventh day with a serum creatinine of 1.5 mg/dl on maintenance of immunosuppressive regimen of tacrolimus, rapamisin, prednisolon and other medication as metoprolol. There was no episode of rejection. His creatinine levels were in range of 1.74 to 1.35 mg/dl, tacrolimus levels were 3.05 to 6.85 ng/ml during follow up. At 25 months post transplantation he dropped down from a tree nearly 1 m high. After trauma, hematuria and graft pain began gradually. He admitted to hospital in the vicinity of his town because of these complaints. Abdominal ultrasound (USG),

and noncontrast computerized tomography (CT) scan were performed. Investigations revealed hematoma at the posterior part of graft kidney. In the light of findings the doctors did not prefer a surgical operation for the hematoma. Creatine kinase and uric acid levels were normal but his creatine levels stepped up therefore he was maintained on hemodialysis at the center when creatinine was 7.6 mg/dl. The patient wanted to continue his treatment at our hospital where he had underwent transplantation, so he admitted to our hospital. Physical examination revealed a blood pressure of 170/110 mm Hg, a regular pulse of 88 /min, and fever 37.5 C^0. The only abnormal finding on examination was tenderness on the graft area and pretibial edema in bilateral legs and ecchymosis in both limbs.

Laboratory findings on admission were as follows: hemoglobin was 11.5 gr/dl, white blood cell counts (WBC) 15.400/mm^3, platelets 148.000/mm^3. Blood biochemistry analysis revealed creatinine 5.9 mg/dl (normal range, 0.4-1.2 mg/dl), bicarbonate 16,2 mmol/l ,sodium 120 ml/mol, potassium 4,4 ml/mol, aspartate amino transferase (AST) 28 U/L (normal range,10-41 U/L), alanine amino transferase (ALT) 18 U/L (normal range, 10-40 U/L), C-reactive protein (CRP) 9.1 mg/dl (normal range, 0-10 mg/dl).Urine analysis revealed hematuria and leucocyturia. Urine culture was sterile. The urinary total protein/ creatinine ratio was 904 mg/d. A 74x40 mm hematoma was detected in renal ultrasound at the posterior of graft. There was no hydronephrosis. A renal Doppler USG was performed; the graft was evaluated as normal with normal Doppler flows. Intra renal systolic acceleration was normal. Resistive indexes upper/medium/lower were respectively 0.56/0.61/0.59. Renal artery flow form and speed were normal (87/29 cm/sec). Renal vein was normal. Tc-99m-MAG 3 nuclear scanning showed delayed and poor perfusion. Concentration function was decreased, excretion function was delayed and elongated. During 20 minutes imaging it was seen that the nuclear mass retained in renal cortex. The superior part of graft was determined normal where the medial and inferior parts were smaller than the former and there were hypoactive regions in latters. During hospitalization the patient's blood pressure ranged from 170/120 mm/Hg to 150/100 mm/Hg in the first week. He was not oliguric but renal function elevated day by day from admission so, hemodialysis sessions were started. During hemodialysis sessions ultrafiltration was also performed because of hypervolemia. At the fourth day pleural pain commenced. X ray was normal. Regarding to lung ventilation and perfusion scintigraphy, pulmonary thromboembolism was diagnosed. Low molecular weight heparin was started. Deep venous thrombosis wasn't established. He complained of diarrhea; amoeba trophozoites and cysts were detected in stool. Metronidazole treatment was begun. After 8 days later, urine flow started to be much more than before. Blood pressure diminished to 130/100 mmHg with same antihypertension medications. Serum creatinine steadily lowered to 1.65mg/dl nearly the same as his stable stage before. Renal function recovered on conservative management. Renal USG was repeated two weeks later and hematoma was 73x23 mm. RenalTc 99m -MAG 3 nuclear scintigraphy was also repeated. Renal perfusion delayed and diminished and concentration function was improved when compared with the previous scanning. Excretion function was delayed and elongated nevertheless; it was better than the former scan. Hypoactive regions in medial and inferior parts hold a little radioactivity. Immunosuppression was continued during hospitalization. He stayed twenty days at hospital. He was on hemodialysis just ten days for four times. He had got no problems with pulmonary thromboembolism.

His urine output was adequate, blood pressure was 130/90 mmHg when he discharged from hospital with low molecular heparin.

DISCUSSION

Page kidney was first described by Irvine Page in 1939, when he wrapped animal kidneys with cellophane and observed the development of acute hypertension (1). Hypertension has been ascribed to result from renal hypo perfusion and micro vascular ischemia from external compression of the kidney, and subsequent activation of the renin-angiotensin-aldosterone system (2). Page kidney is, therefore, somewhat akin to the Goldblatt model of renovascular hypertension, except that in the latter there is compression or stenosis of major renal vessels where as in page kidney there is an ischemic renal vasculature (4). Although the pathological mechanism underlying Page kidney is not completely understood, one study reports that inflammation and interstitial nephritis producing by extrinsic compression of the kidney ,results in hypertension rather than activation of the RAAS mechanism (5).

Common causes of Page kidney are: bleeding secondary to trauma especially after or during sports ,motor vehicle accident), bleeding secondary to interventions (post-operative kidney biopsy, extracorporeal shock wave lithotripsy, sympathetic nerve block), bleeding spontaneous (pancreatitis, warfarin therapy, polyarteritis nodosa, tumor, Wegener, renal cyst rupture), non-bleeding causes (pararenal lymphoceles, large simple cysts, retroperitoneal paraganglioma, perirenal pseudocysts, urinoma, peritransplant lymphocele (3), infiltrative disease that encasing both kidneys like Erdheim- Chester disease (6).

Native kidneys can be protected by thoracic wall and paraspinal musculature. In native kidneys there is a relatively large space between Gerato's fascia and the kidney compared to renal graft. Because renal graft has no gerato's fascia or surrounding cushion of fat, it is more susceptible to external force in iliac fossa. Furthermore, a little hemorrhage can also cause compression to renal graft.

In the literature trauma cases developing page kidney two of three percentage showed young men dominance, average age was 38 years (17-69 years), mean arterial blood pressure

was 177/95 (150-215/76-116 mm Hg) at diagnosis (4). Prior to 1991, cases of page kidney were rather hypertension cases investigated for hypertension reasons after trauma; after 1991, which was more invasive cases after renal biopsy performed the kidney transplantation. The authors, attribute the reason for these changes to high-quality imaging technology with advances in medicine, more invasive procedures and the presence of stronger protective equipment in sportive competitions (4).

In a retrospective study Smyth and his colleagues investiged 26 page kidney cases. They divided them into two groups as traumatic and nontraumatic reasons. They found that the trauma group was younger than the non-trauma group, systolic blood pressure was lower (mean: 162-184mmhg) and MDRD GFR was higher. Furthermore trauma cases required fewer antihypertensive medications than the non-trauma patients. Also, all patients that required nephectomy were in the nontraumatic group (2). Our patient was also similar in trauma group because he was young and his systolic blood pressure wasn't intractable.

Typical findings on Doppler Ultrasound include increased resistive indices and severe reduction of diastolic flow. If the hematomas are too small, the CT scan and MR imaging are also helpful for diagnosis (3). MR is more sensitive to the stage of blood product degradation and, the age of subcapsular hematoma (7). Nuclear scintigraphy has not already entered into routine use for Page kidney. Therefore more cases of Page kidney scintigraphic images are required for results. Our case, in this regard, is a case where the Tc-99m-MAG 3 nuclear imaging is also performed.

There are reports that spontaneous resolution of subcapsular hematomas (3,8). Hematomas less than 3 weeks old usually resolve spontaneously; otherwise nephrectomy or capsulectomy may be required for organized late hematomas (9). In this case because the hemorrhage was subacute, we decided to be conservative. Auspiciously, his hematoma shrinked in two weeks and hypertension and renal dysfunction improved fractionally.

In order to accuse hematoma for the renal dysfunction, other responsible etiologies should be ruled out. Post renal obstruction was excluded by renal graft USG. In renal graft Doppler USG resistive indexes were normal. There were no new liable drugs for acute interstitial nephritis. Hence hematoma was thought to be responsible for this creatinine elevation.

The acute presentation of a page kidney in a renal transplant has been called ''pseudorejection'' (3,10). As in our case, spontaneously shrinking of hematoma resulted in the recovery of graft. The Page kidney phenomenon may not only occur acutely but also may occur chronically with the development of a constricting fibrous casing (11). Surely diagnosing an early diagnosis in Page kidney is of paramount importance, since it enables evacuation surgical procedures, when indicated. Options for evacuation of the subcapsular hematoma may include laparoscopic surgery, surgical decortication (12) capsulotomy (13), interventional radiology, preventive techniques such as mesh hood fascial closure (14) or open exploration (13). Laparascopy has been described as a more recent treatment modality for Page kidney (15). Nephrectomy is indicated for arteriovenous malformation with renal hemorrhage, aneurysm, cyst rupture and hypernephroma (2).Percutanous drainage is the most successful method in cases of acute hematomas but is unlikely to resolve older (organized) hematomas (11). Also percutanous drainage needs to be performed before the formation of a fibrotic pseudocapsule (11).

Hypertension usually does not occur until the hematoma organizes into a restrictive fibrous capsule that compresses the kidney and interferes with intrarenal blood flow (16) The interval between injury and the development of hypertension may vary from days to years (7).Success with angio-converting enzyme inhibitors drugs and diuretics for management of hypertension has also been reported (18). Hypokalemia can be seen because of hyperaldosteronism (16).

Çiftci and et al. showed two cases native kidneys with large cyst with internal debris in a single kidney and new-onset hypertension after trauma. Ultrasound guided percutaneus drainage and then transperitoneal laparoscopic renal cyst decortications and evacuation of the cyctic structure made, antihypertensive drug was out of patient needs (12).

Finally, the presented case worth to be mentioned as an example to satisfactory response to conservative medication. Spontaneous resolution of hematoma apparently ameliorates renal dysfunction. The rationale for treating the patient with conservative approach was to avoid harmful consequences of the operation and successfully salvaged kidney function.

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