Page Kidney: Possible complication of renal biopsy.

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Abstract:

Page kidney is an un-usual complication of renal biopsy, resulting from marked compression of renal parenchyma by the perinephric haematoma leading to compromise of blood supply to renal micro vasculature. It presents as new onset of hypertension or marked elevation of blood pressure in previously normotensive patients. The phenomenon is usually reversible and responds well with resolution of perinephric haematoma. We here in report a case of Page kidney diagnosed on history and clinical presentation supported by radiological findings and resolution of hypertensive emergency with resolution of haematoma.

Keywords: *Page kidney, perinephric haematoma, renal biopsy, hypertension, complications.*

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Introduction:

Renal biopsy is relatively a safe procedure and its importance in diagnosing and treatment of different renal pathologies cannot be denied. With the increase in expertise and availability of advanced imaging techniques the risk of complications has decreased. There is 1.2% chance of developing any serious complication (requiring blood transfusion, angiography or nephrectomy). Mild to moderate amount of peri nephric collection may occur in 3-33.3%.[1] It may lead to significant pressure on renal parenchyma resulting in activation of renin angiotensin aldosterone system (RAAS). Page kidney is defined as an external compression of kidney due to any cause significant enough to decrease the perfusion of kidney leading to renal ischemia and activation of RAAS. We herein present a case of page kidney resulting as a complication of renal biopsy which was managed conservatively.

Case Report:

A 45 years old male presented with 3 months history of fatigue, abdominal pain and now vomiting. He had used some herbal medicines and NSAIDS for his symptoms. He was evaluated three weeks ago for unexplained anemia and referred to us when found to have serum creatinine of 15mg/dl. His blood pressure was 100/70mmHg, pulse 94bpm and rest of the physical examination was unremarkable besides pallor. Ultrasound (USG) abdomen revealed normal sized kidneys with preserved corticomedullary distinction and mild increase in cortical echogenicity. His hemoglobin (Hb) was 9.6. Urine analysis revealed proteinuria 3+, 6 – 8 RBC's and no cast. Immunologic workup was negative including ANA and normal C3, C4; negative HIV and Hepatitis B and C serology. ANCA serology was not done due to cost.

Considering rapidly progressive glomerulonephritis (RPGN), intravenous methylprednisolone was started along with hemodialysis (HD). After four sessions of hemodialysis , renal biopsy was done and two cores of renal tissue were taken from lower pole of left kidney under real time USG guidance. Coagulation profile, bleeding time, clotting time and platelet counts were normal 12 hours prior to the biopsy procedure. As a protocol strict bed rest for next 24 hours was advised. Six hours after the procedure a portable USG did not reveal any perinephric collection. Patient remained pain free and passed normal colored urine during this time and later shifted to ward. At 30 hours of procedure patient complained of left sided flank pain and B.P. was 160/110mmHg. Oral nifedipine retard 20mg was given. Urgent USG revealed small perinephric hematoma (upper pole, 18ml; lower pole, 14ml). He was shifted to ICU for B.P. monitoring and management. His increased blood pressure required use of IV isosorbide

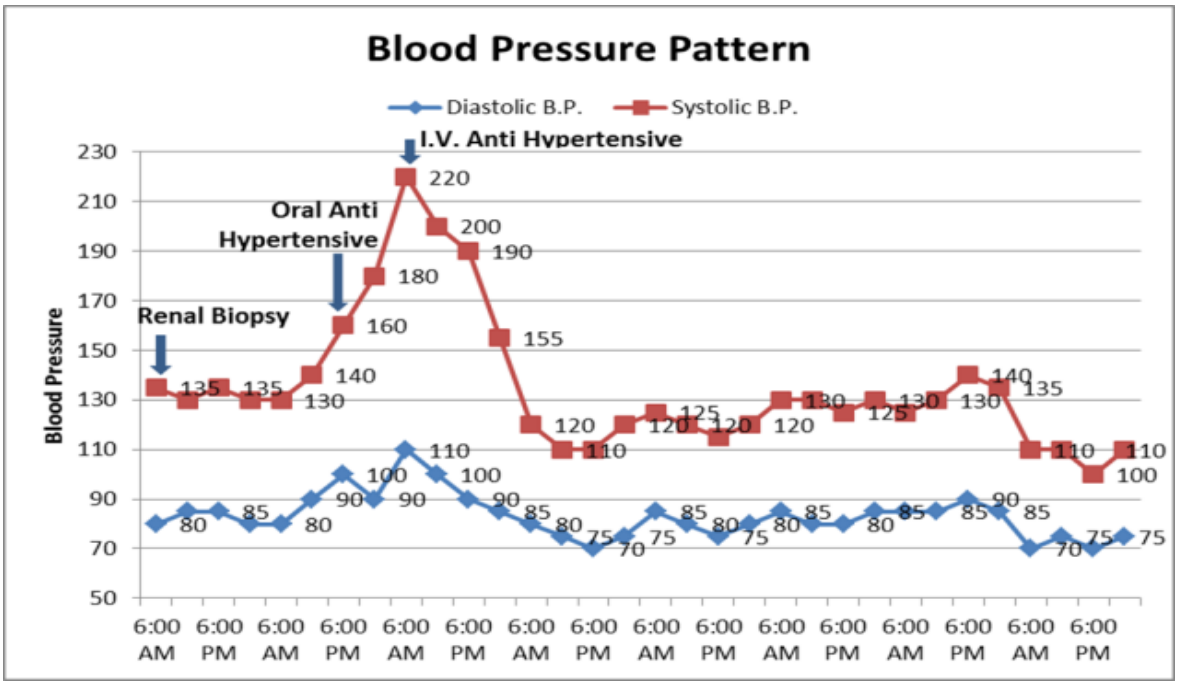

Figure 1: Blood pressure pattern of the patient post renal biopsy, increasing suddenly at 36 hours and gradual settling over next 24 hours.

dinitrate, oral prazosin, captopril and calcium channel blockers for appropriate control. His blood pressures remained high for 24 hours and then started settling down, **Fig 1**. HD (heparin free) was done on 3rd post biopsy day and USG repeated 24 hours later revealed further increase in hematoma as 200 – 250 ml of organized perinephric collection. After blood pressure control CT scan KUB was done which confirmed a perinephric collection of 200 ml, **Fig 2**. Surgical consultant advised conservative management. Patient was kept on appropriate analgesics and was advised strict bed rest. Serial USG were repeated on 4th, 7th, 9th and 11th post renal biopsy day with gradual decrease of hematoma from 160 ml, 125 ml, 70 ml and 25ml respectively. Blood pressure remained stable during this period with single antihypertensive agent.

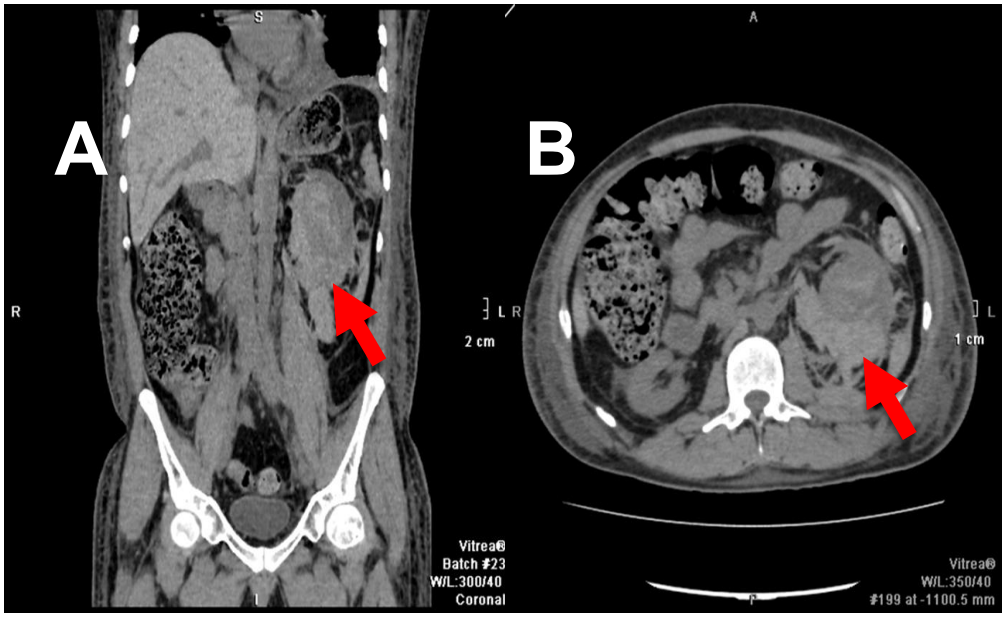

Figure 2: CT Abdomen **A:** (Coronal section) and **B:** transverse section, revealing hyperdense area around left kidney. Representing collection of blood and inflammatory perinephric fat stranding.

The Hb of patient dropped during this episode requiring two pints of pack cell volume transfusion, **Fig 3**. Meanwhile, patient was continued on dialysis, his renal pathology revealed advanced crescentic GN with severe interstitial fibrosis and fibrocellular crescents. Immunofluorescence was negative. A diagnosis of pauci immune renal limited RPGN was made. Considering severe interstitial fibrosis and life threatening perinephric hematoma plasmapheresis was not offered. Patient was discharged on 12th post renal biopsy day on tapering doses of prednisolone and advised to continue dialysis at his home town and renal transplant in future. He unfortunately expired 3 months later in his hometown due to complications related to sepsis.

Discussion:

Our patient had page kidney as a complication of renal biopsy. In fact, Page Kidney was first time described by Irvine Page in 1939.[2] He postulated that applying external pressure on kidney can cause a significant decrease in the micro vascular blood supply of kidney and activation of RAAS with significant rise in blood pressure. In his experiments, Page wrapped a canine

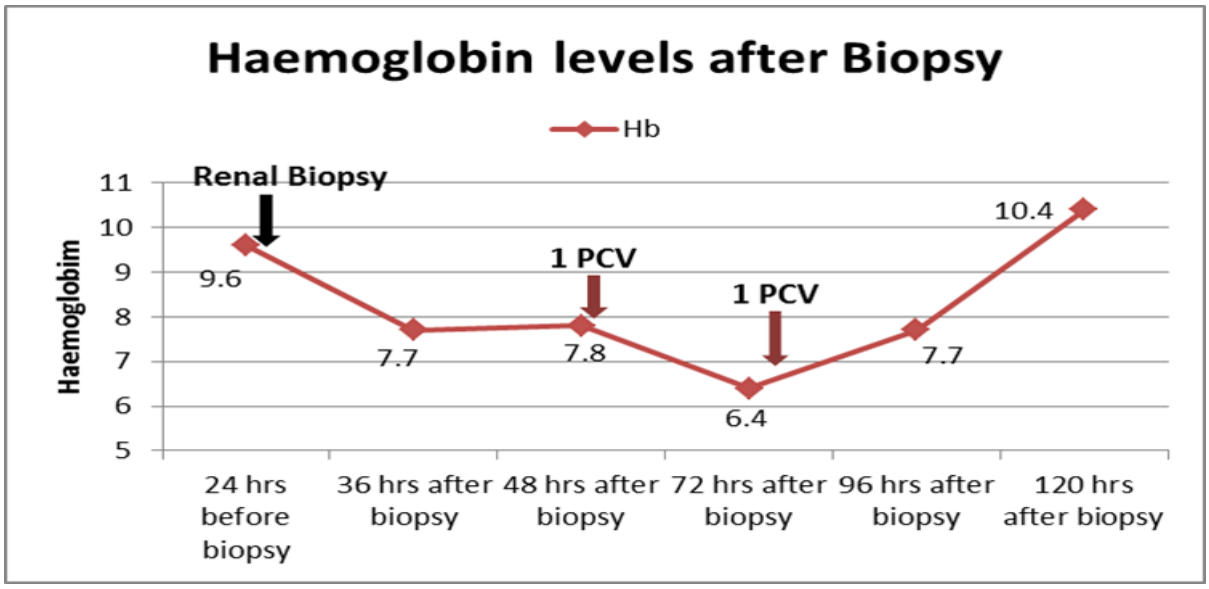

Fig. 3: Hemoglobin levels of the patient. Hemoglobin fall suddenly after renal biopsy suggestive of blood collection around the renal parenchyma.

kidney with cellophane paper and showed that it leads to constrictive perinephritis as a result of intense foreign body reaction to the cellophane paper. The constrictive perinephritis led to the vascular compromise to microvascular circulation and then hypertension. This was the result of Ag-II mediated vasoconstriction and increased salt and water retention which subsequently caused hypertension. It remained a more of an experimental model until the first case of an American footballer appeared in 1955.[3] He suffered from a blunt trauma in the lumbar region leading to perinephric collection of blood and subsequent hypertension. Since then many case reports of page kidney due to various etiologies has been described in the literature. Usually, hypertension is the presenting feature with the development of page kidney but sometimes it may present as renal insufficiency in cases of single functioning kidney. It may present as pseudo rejection in case of renal allograft biopsy.[4]

Page kidney can present as episode of new onset hypertension or a significant rise in blood pressure in previously hypertensive patients after the event.[5] This phenomenon is usually transient and blood pressure returns to baseline as soon as the external pressure is relieved such as in our patient. In few cases however blood pressure may not normalise.[4,5]

Anatomically, kidney is in the retroperitoneal space with no significant protection. It is surrounded by two envelopes. The first is Gerota's facia, it is a large space and large hematoma is required to fill up this space and compress the kidney. The second is the kidney capsule which is a potential space and a little collection of blood in this space can lead to renal compression.[5]

The causes of page kidney are mainly collection of blood due to trauma, intervention and spontaneous bleeding resulting from coagulopathy. Other causes include the external compression due to hydrocele, large simple cyst and urinomas etc. [5,6]

USG guided per cutaneous renal biopsy (PRB) is an important diagnostic tool in both native and allograft kidneys. It helps in the diagnosis and management and is a relatively safe procedure with very low risk. There is 1.2% chance that USG guided PRB may lead to such complication that may require blood transfusion, angiography or nephrectomy.[1] Page kidney has been described after US guided renal biopsy both in native kidney and transplanted allograft biopsies.[7,8] Perirenal hematoma is observed more often than subcapsular hematoma.[7]

Diagnosis is readily made with USG, being a non-invasive test. However it can miss early and small subcapsular hematomas being highly operator dependent, such as in our case there was no hematoma at 6 hours post biopsy. Doppler USG can also help by showing the reduced perfusion of kidney due to external compression. Non-contrast CT scan is a non-invasive, more sensitive and able to identify even small hematomas whereas MRI may be helpful in assessing the age of hematoma and patency of the renal vasculature.[4] Other investigations like renal DTPA or DMSA nuclear scan can support the diagnosis by showing the decreased uptake or perfusion respectively.[4] Renal arteriography can be used in selected cases where there is continuous bleeding to identify the site and possible coil embolization.[9]

The treatment of Page kidney is mainly control of hypertension and bleeding.[10] ACE inhibitors and ARBs (angiotensin receptor blockers) can effectively counter the RAAS due to Page kidney and help to control hypertension efficiently. An expectant or interventional approach can be followed. Spontaneous resolution of peri renal hematoma has been reported generally in a three weeks' time.[5] In our patient, the hematoma resolved significantly by 12th day before discharge along with controlled blood pressure. For expanding hematomas with or without difficulty to control hypertension, intervention is necessary.[11] Interventional approach involves drainage of hematoma via percutaneous, laparoscopic or open surgical approach depending upon the location, size and expertise of the surgeon.[5,9] In rare cases nephrectomy may be required.[12] Long standing hematomas may organize into pseudo capsules or may lead to permanent renal parenchymal damage.

In cases of oligo/ anuria in an allograft kidney or single functioning kidney, active surgical intervention is required to relieve the compression to salvage the kidney.

Conclusion:

Page kidney is a rare complication of kidney biopsy presenting as a sudden increase in blood pressure post biopsy, and high index of suspicion will lead to early diagnoses and management. Majority of such cases will resolve spontaneously with conservative management and rarely require active intervention.

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