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RESEARCH ARTICLE

POST RENAL TRANSPLANT ATN COMPLICATED RENAL TEMPONADING FROM MARKED SUBCAPSULAR HEMATOMA

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ABSTRACT

Subcapsular hematoma after kidney transplant may result in kidney ischemia and graft loss. In this report, we present a 38 years old male patient who had post-transplant ATN complicated a subcapsular hematoma just after surgery. He had end stage renal disease secondary to chronic glomerulonephritis (hypertension, proteinuria and bilateral atrophic kidneys in abdominal ultra sound (US). He had a living-donor kidney transplant from hissister. At perfusion, there was smalldecapsulatedarea in the lower pole of the transplanted kidney. Capsulotomy was not performed due to possible risks and transplant surgery was completed. After declamping, appropriate renal perfusion and urine output were observed. Early few hours after surgery, decreasedin urine output was observed. Renal Doppler ultrasonography showed decreased diastolic flow and perigraft hematoma about 200ccand resistive index (RI) 0.9 initially that increase to 1 in serial successive Dopplers. The surgical drain is hemorrhagic leading to marked decrease in hemoglobin level that necessitatedto transfuse four units of filtered irradiated RBCS. The plan by surgeon to conserve,so renal biopsy was done revealed moderate ATN. He underwent re-exploration on day 11 due to infected hemorrhagic , huge amount drain fluid (culture and sensitivity from it MARSA), that revealed most of the renal parenchyma was compressed with marked stretching capsule due to subscapular hematoma, the plan to release it by slight dissection of capsule at different directions but not to evacuate it completely for possible bleeding. After surgery, urinary flow increased, renal Doppler ultrasonography findings and renal function gradually improved.

INTRODUCTION

Page kidney phenomenon is the occurrence of kidney hypo perfusion and ischemia due to pressure on the kidney by a sub scapular hematoma. Hypo perfusion and micro vascular ischemia in the kidney may stimulate the renin-angiotensin-aldosterone system and cause hypertension (Nurettin *et al*, 2015). It usually presents with acute pain, oliguria, and hypertension. In some cases the patient develops a significant decline in renal function; this is due to hypo perfusion of the kidney, hence decreased renal filtration ability (Rodrickand Aaron Sean 2018). There is controversy about the treatment of sub capsular hematoma. There are papers that report spontaneous resolution of sub capsular hematomas (Salgadoetal. 2015). Antihypertensive treatment, percutaneous drainage; surgical decortication, laparoscopic intervention, and nephrectomy are the other treatment options (Çiftçi and Wolf 2013).

Case report: Male Patient 38 years old had end stage renal disease and was maintained on regular hemodialysis for four months through temporary right jugular catheter, the primary cause of end stage renal disease was chronic glomerulonephritis. He was prepared for living related renal transplant from his sister, both of blood groups A1, 3mismatches 1:1:1, PRA negative and cross match negative by lymphcytotoxicity and flowcytometery. Immunosuppression , basiliximab (simulect) induction 20mg IV D0 and D4, maintenance immunosuppression, mycophenolate sodium (MPA), 720mg /12hrs starting on day -3, tacrolimus 0.75mg/12hrs starting on day -1 and methylprednisolone 250mg IV was given on D-1, D0 morning and 500mg IV before declampingintraoperatively then tapering dose later on was done till 20mg prednisolone D30. There was smooth intraoperative course but at perfusion table, there was smalldecapsulated area of the lower pole of the transplanted kidney. Capsulotomy was not performed due to possible risks and transplant surgery was completed. Although initially after declamping, appropriate renal perfusion and urine output were observed, there was decreasedin urine output early after surgery. Clinically patient was euvolaemic, hemodynamically stable, no urinary catheter obstruction. suspecting post-operative delayed graft function, so discontinuation of tacrolimus was done, IV Lasix infusion to maintain diuresis providing that keeping the patient slightly on the hydrated side,

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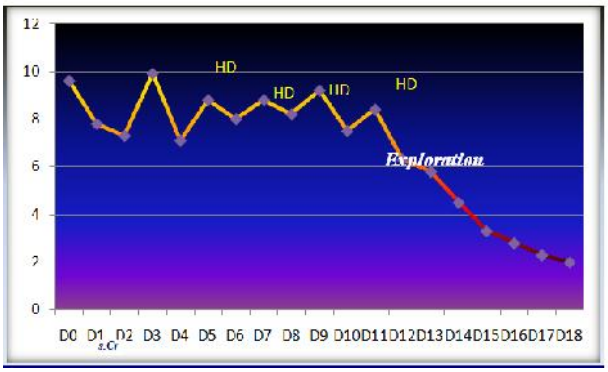
urgent Doppler US on transplanted kidney revealed perfuse kidney with patent renal artery and vein, RI initially 0.9 and there was perigraft collection 200cc, that was stable in serial successive dopplers US with increase RI to 1 (no diastolic flow). The drain fluid was hemorrhagic infected and of large volume, necessitating transfusion of 4 units of filtered irradiated RBCs. D2 patient exhibited low grade fever although he was on (cefoperazone & sulbactam 1.5gm/12hrs), so it was replaced by meronem 500mg/12 hrs andpan-cultures were sent. Result revealed positive culture of MARSA from drain fluid that was sensitive to meronem and aprozolid 600mg /12hrs, that were given as combined therapy. Renal biopsy was done on D3 revealed moderate ATN, C4d negative otherwise normal. He received 4 sessions of hemodialysis during his illness based on clinical and biochemical evaluation. Tacrolimus was reassumed to maintain its level 6-8 ng/ml to avoid rejection on top of ATN.

On day 11 he was re-explored after discussion with surgeon, patient sdrain fluidwas huge amount and persisted to be hemorrhagic, infected (2nd drain culture and sensitivity on meronem revealed MARSA with addition of 2nd antibiotics aprozolid600mg twice daily). There was no improvement of renal function although there was improvement of Doppler's RI. On re-exploration there was massive subcapsular hematoma encircle most of the parenchyma of the transplanted kidney, the capsule over it was markedly stretched, the plan to fractionate the overstretchedcapsule to relieve it. Avoid to evacuate hematoma completely as it carried the risk of severe bleeding. Post-operative urine output was significantly improved together with gradual tapering of s, creatinine and drain fluid was gradually deceased and infection was cleared by continuation of the abovetwo antibiotics for 2 weeks. On discharge s. creatinine 2mg/dl. 1st visit follow up 1 week post discharge s, creatininewas 1.3mg/dl, US Doppler RIwas 0.6, initially, there was small perigraf collection that was cleared in subsequent Doppler follow up.

DISCUSSION

Page kidney phenomenon was first described in an experimental model in the 1930s (Page, 1939). Cromie *et al.*, 1976 described it as renal allograft pseudorejection; after living-donor renal transplant, the patient had refractory hypertension, perinephric hematoma that was observed by renal ultrasonography, and blood pressure was normalized after intervention to treat the hematoma. Hypertension is the characteristic finding in the classic Page kidney phenomenon from external pressure to kidney parenchyma results in reninangiotensin- aldosterone activation (Ginza *et al.*, 1995).Various imaging modalities have been used to diagnose Page kidney. Ultrasound has the advantage of being cheap, easy to perform and non-invasive; but because it is highly operator dependent, it can miss small compressive sub-capsular hematomas. Doppler evaluation also may give valuable clues toward the diagnosis of Page Kidney: it signals a rapid and high increase in renal resistive index. CT of the abdomen is the preferred modality because it is a non-invasive, readily accessible test, which can detect even very small hematomas (Wu *et al.*, 2015). Magnetic resonance imaging (MRI) may be helpful in assessing the age of the hematomas and patency of renal blood vessels. Our patient had moderate hypertension, mild tenderness over graft postoperatively, together with perinephric hematoma that was stationary



This figure (1): reveals delayed graft function with fluctuation of s. creatinine early post transplantation necessitating 4 sessions of hemodialysis, till re-exploration was performedon D11 there is gradual improvement of renal function, s, creatinine 2mg/dl on discharge

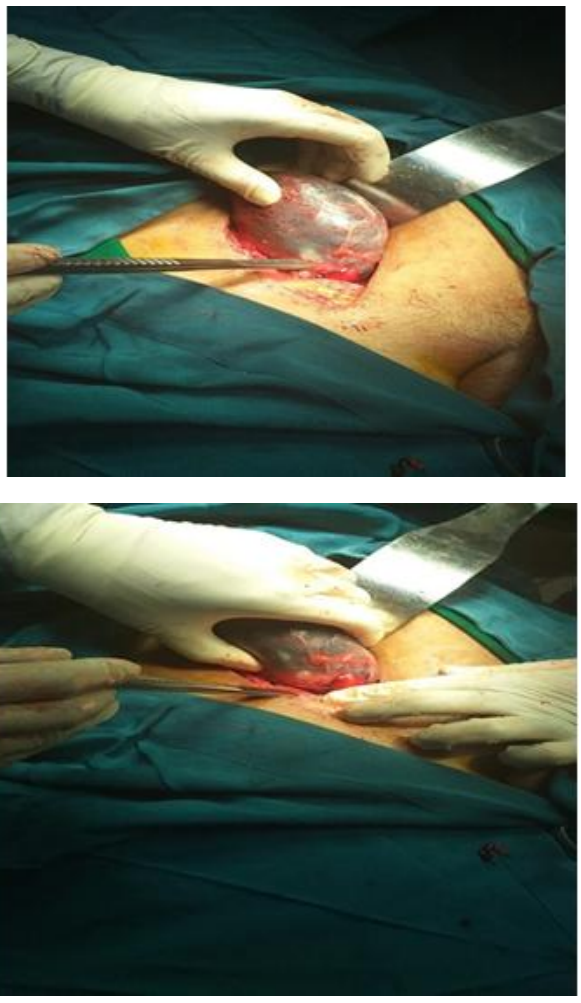


Figure (2): Marked sub-capsular hematoma surrounded more than 80% of the transplanted kidney with excellent renal pulsation after intraoperative re-exploration.

postoperative in serial doppler's US, so there was a possibility of subcapsular hematoma, but surgeon opinion after discussion with him wasto conserve. Page kidney usually presents with acute pain, oliguria, and hypertension in the background of a recent cause for subcapsular space occupying lesion (Chung *et al.*, 2008). In some cases the patient develops a significant decline in renal function, as in our patient. This is due to hypoperfusion of the kidney, hence decreased renal filtration ability.

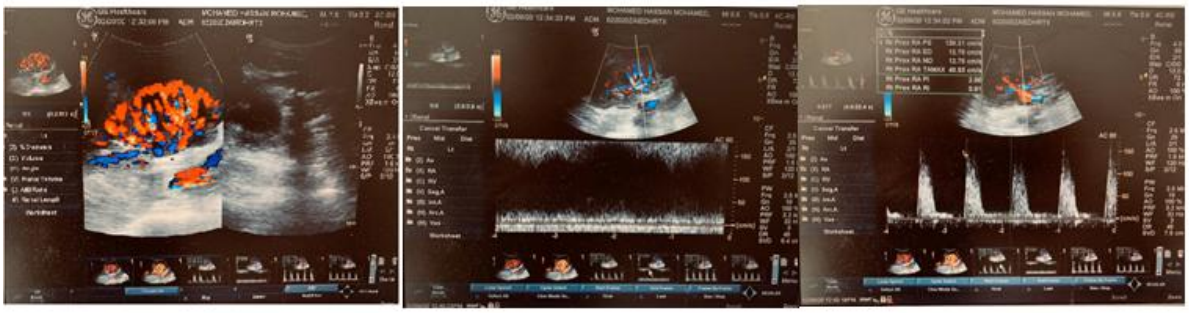


Figure (3): Doppler US of the transplantedkidney on D0, post-transplant perigraft collection about 200 cc & RI 0.9 with adequate renal perfusion

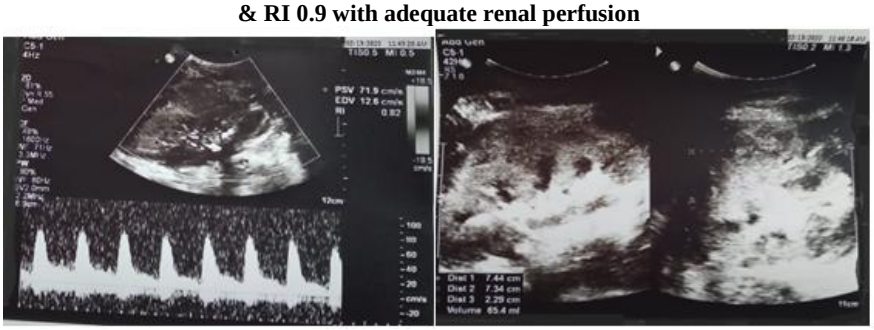


Figure (4): Doppler US of the transplanted kidney 2 days after re-exploration, (D13)perigraft collection about 180- 200 cc organized around the graft & RI 0. 82

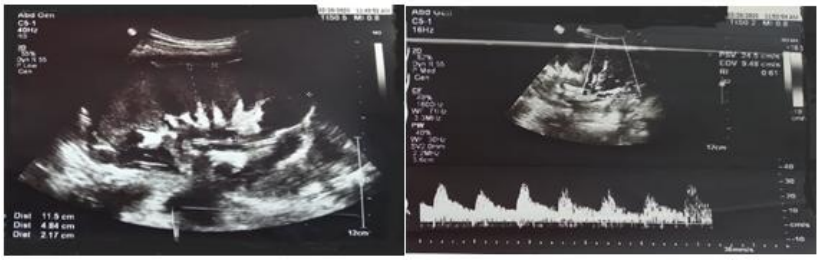


Figure (5): Doppler US of the transplantedkidney on discharge, (D20), perigraft collection about 20 cc sepetated anterior to the graft & RI 0. 80

A number of isolated page kidney cases after transplant kidney biopsies have been reported in literature (Chung *et al*., 2008), (Posadas *et al*., 2010) (Maurya *et al*., 2011). A large retrospective study of 518 renal transplant biopsies, noted only 4 patients (0.8%) to develop biopsy-induced subcapsular hematoma resulting in page kidney (Chung *et al*., 2008),. In our patient renal biopsy was done in D3, pre and post biopsy dopplers were revealedperinephric hematoma that was stable at 200cc, high RI 1 so, we cannot consider a biopsy as a cause of subcapsular hematoma. Biopsy result revealed moderate ATN. Early recognition of acute page kidney is important to prevent progressive pressure induced ischemic organ damage. There is currently no evidence-based guideline for the management of page kidney. Initial attempts should be made to stabilize the patients conservatively. However, given the delicacies surrounding transplant kidneys, there is a relatively low threshold for operative intervention to preserve the transplant kidney. Although small, asymptomatic subcapsular hematomas resolve spontaneously and are conservatively managed; larger subcapsular hematomas may necessitate more focused intervention. Unbearable pain and renal compression and ischemia are indications for immediate intervention (Rodrickand Aaron Sean 2018). Our patient initially treated conservatively , transfused four units of filtered irradiated RBCS to maintain Hb more than 10 , receive 4 session of HD to improve the general condition of the patient together with antibiotic cover for treatment of drain fluid infection, meronem is given 500 mg /12 hrs for 14 days guided by frequent CRP titre measures. Control blood pressure with target level to be not more than 140/90, and strict fluid balance. Regarding maintenance immunosuppression, the patient continued on steroid, MPA 720mg /12hrs together with reassuming of tacromiluson day 5 to maintain tacrolimus level 6-8ng/ml. The 2nd dose of basalixomab 20mg was given on D4. Some centers delay the use of calcineurin inhibitors (CNI) in patients with delayed graft function (DGF), as CNIs may contribute to renal ischemia. Sirolimus delays the recovery from ATN, and is usually not a valid alternative. The majority of transplant centers advocate the use of depletional induction therapy with polyclonal anti-thymocyte globulin when DGF is anticipated (Cardarelli, 2019). Polyclonal antithymocyte globulin was not used in our patient who had active infection from drain side. Even with the above measures there was no improvement of renal function, for that together with drain fluid volume significantly increased, it was hemorrhagic and infected (2nd culture and sensitivity that was revealed MARSA), onmeronem so linezolid was added for 14 days in addition to meronem, CT abdomen was done for more documentation that revealed perinephric collection encircle the transplanted kidney. We planned to re-explore the patient on D11. Intraoperatiely the transplanted kidney showed massive subcapsular hematoma encircle most of the transplanted kidney with tight stretched capsule over it. The renal bed was completely clean with no oozing around.

Surgical evacuation should be considered the intervention of choice, especially in rapidly progressing hematomas, or when vital signs deteriorate**(**Bans *et al*., 2010).Removal of the renal capsule and the constricting fibrous capsule has been shown to be curative (Moriarty *et al*., 1997). Chung *et al*. 2008 had successful treatment of hematoma by evacuation in all cases of page kidney in a retrospective study of 519 patients after renal biopsy. Our patient was not exposed for these modalities as he was hemodynamically stable, subcapsular hematoma stable and was not progressive together with its temponading action on renal parenchyma that may be lost with complete evacuation after removal of the capsule. So the plan to release the temponading effect of overstretched capsule by making small fissuring of it at different points. Post-operative urine output significantly improved together with gradual tapering of s, creatinine and drain fluid gradually decreased and infection is cleared by continuation of two antibiotics for 2 weeks. On discharge s. creatinine was 2mg/dl. One week follow up post discharge s, creatinine 1.3mg/dl, US Doppler RI was0.6,initially it revealed small perigraf collection that was cleared in subsequent Doppler US follow up.

Conclusion

Page effect to be suspected when we notice an increase in serum creatinine, diuresis contraction, arterial hypertension and doppler US evidence of renal resistive index increase with peri-graft effusion, without signs of acute rejection. CT scan may be helpful to detect even very small hematomas that require urgent surgical drainage in order to obtain rapid recovery of renal function.

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