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Case Series

Page Kidney In Kidney Transplantation: A Case Series

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Introduction

The phenomenon called Page Kidney (PK) refers to the compression of the renal parenchyma. It is frequently the result of a subcapsular hematoma or a mass that leads to the activation of Renin-Angiotensin-Aldosterone System (*RAAS*) and resulting in arterial hypertension [1]. There have been described more than 150 cases of PK [2], however, in Kidney Transplantation (KT) the cases described do not reach 40. We present four cases diagnosed in our center.

Cases

Patient #1

A 70-year-old male with hypertension, ischemic cardiac diseaseand with a chronic kidney disease due to membranous nephropathy, received a KT from a controlled cardiac death donor. He received as a induction therapy basiliximab, and as a maintenance immunosuppression tacrolimus, mycophenolate mofetil, and prednisone. The following days after KT, the patient presented delayed graft function (creatinine (Cr) 575 µ mol/L) with need of hemodialysis and he also presented refractory hypertension (persisting arterial pressure over 140/90mmHg although treatment with 3 or more antihypertensive drugs). An ultrasound was performed in the following 24 hours, showing a subcapsular hematoma of 89x26mm in the graft (Figure 1A) with elevated parenchyma resistances (0.86-0.96). An abdominal scan revealed a generalized renal hypoperfusion without involvement of the renal vessels, ruling out active bleeding. It was orientated as a subcapsular hematoma with parenchymal compression probably originated at the point of preimplantation renal biopsy. We decided together with Urology Department to follow a conservative strategy. After 12 days, the urine output increased, kidney function ameliorated progressively allowing discontinuation of dialysis and we proceeded with discharge. After 3 months, in the outpatient consult, the patient achieved levels of serum creatinine of 177 µ mol/L, a reduction of the subcapsular hematoma (69x27mm) (Figure 1B) and a well-controlled hypertension with 3 antihypertensive drugs (doxazosin, amlodipine and losartan).

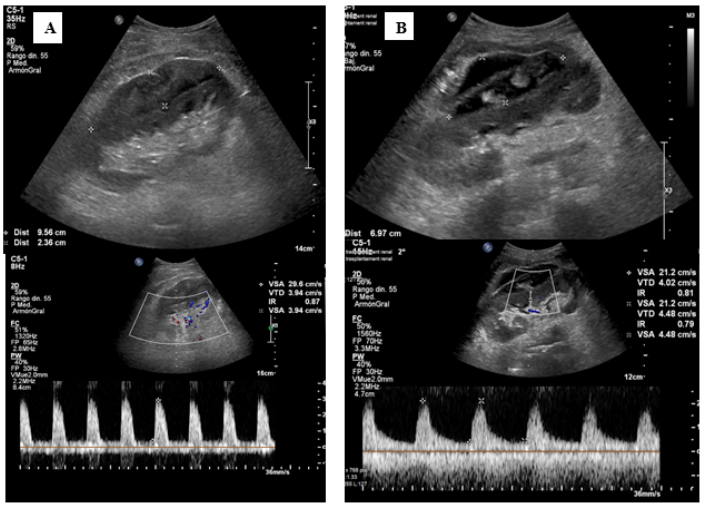

Figure 1: Patient #1 Doppler- ultrasounds. **A)** At diagnosis: hematoma subcapsular of 95.6x25.8mm (superior image) and Doppler showing resistance indices of 0.87 (inferior image). **B)** At 3 months: hematoma subcapsular de 69.7x27.6 mm.

Patient #2

A 69-years-old female with a chronic kidney disease due to immune-complexes mediated membranoproliferative glomerulonephritis, well-controlled hypertension, moderated aortic stenosis and chronic hepatitis C treated and cured, received a KT. She achieved Cr of 140 µ mol/L in the outpatient follow-up. Eight years after KT, she presented to the emergency room with acute pain in the kidney graft area and hypertension of 188/99mmHg. She denied any history of trauma or other symptoms. Laboratory evaluation showed a serum creatinine of 336µmol/L and blood loss resulting in a decrease in hemoglobin concentration of -1.3g/L. An ultrasound showed a subcapsular hematoma of 70x48mm, resistances of 1 and absence of diastolic flux. Conservative strategy was decided. Seventy-two hours after this, the patient developed oligoanuria. An abdomen scan was performed revealing an extense hematoma (70x48mm) and decortication involving almost the entire cortex with signs of hypoperfusion (Figure 2A). The patient was started on hemodialysis. Forty days after dialysis initiation, in the outpatient visit, the patient noticed an increase of urine output. Laboratory tests confirmed renal function improve with serum creatinine 130µ mol/L allowing hemodialysis discontinuation. Three months after the diagnosis, an ultrasound showed a decrease of the hematoma size (9.2mm). However, the patient had persistent arterial hypertension controlled by treatment with four drugs (doxazosin, lecarnidipine, losartan, and furosemide).

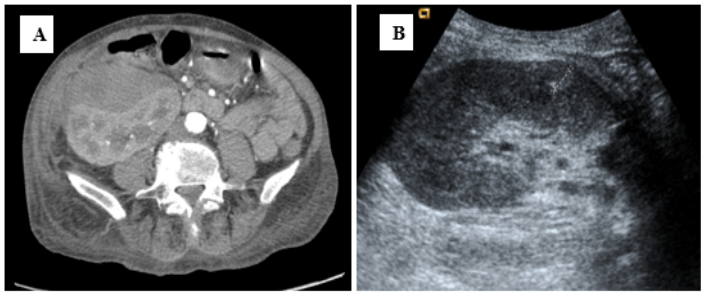

Figure 2: Patient #2 CT scan and Ultrasound. **A)** CT scan showing hematoma (70x48mm), that involves and compresses kidney cortical. **B)** Kidney Ultrasound after 3 months showing partial resolution (9mm).

Patient #3

A 59-year-old male with a chronic kidney disease due to focal segmental glomerulosclerosis, hypertension and chronic hepatitis C treated and cured, received a left KT. Although there were no intraoperative complications, in the first 24 hours the patient presented a haemorrhagic shock resulting in a decrease in hemoglobin concentration of -4g/L. An abdominal scan was performed showing a subcapsular hematoma of 13mm in the graft and decortication of the middle-lower third of the anterior part of lower renal pole, with several active arterial bleeding points. This retroperitoneal and perirenal hematoma of 80 x 70 x 85 mm was probably secondary to a probable rupture of the renal subcapsular hematoma. The patient required six red cell concentrates. His hemodynamic situation was stabilized but the patient remained with delayed graft function with anuria during twelve days. We decided together with Urology Department to follow a conservative strategy without any intervention. During the follow-up, we performed three doppler ultrasounds that showed a progressive diminution of the hematoma size (from 17cc four days after the surgery to 10 cc twenty-three days after the surgery) (Figure 3). Levels of blood pressure were high with high doses of antihypertensive treatment during the first three weeks, but later the recipient presented good control by treatment with three drugs. The urine output increased progressively and kidney function ameliorated achieving levels of serum creatinine of 497 µ mol/L the day of the Hospital discharge (30 days after KT). The last serum creatinine in outpatient control 6 weeks after KT was of 267 µ mol/L.

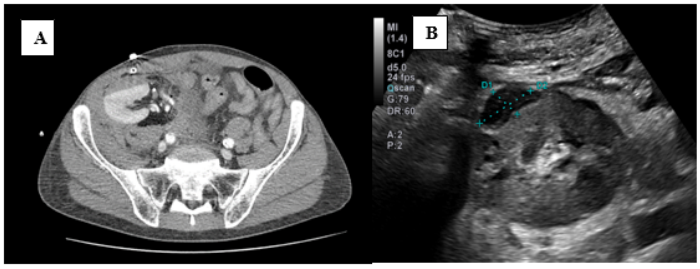

Figure 3: Patient #3 imaging studies. **A)** Axial computed tomography with contrast after KT. **B)** Control ultrasound 3 weeks after surgery.

Patient #4

A 70-year-old male with hypertension, ischemic cardiac disease, stroke and chronic kidney disease due to diabetic nephropathy, that initiated haemodialysis when he was 67 years-old, received a KT. There were no intraoperative complications. His induction immunosuppression was basiliximab, and maintenance immunosuppression was tacrolimus, mycophenolate mofetil, and prednisone. After nine days of hospitzalization, he was discharged with serum creatinine levels of 170 µ mol/L. After one month, just one week after having removed ureteral catheter, the patient came to Emergency Department with heart failure, decreased urine output and renal disfunction with a serum creatinine of 588 µ mol/L. He needed a hemodialysis session. An ultrasound was performed showing a grade II pyelocaliceal ectasia and a drainage by position a nephrostomy was tried. As a complication of the puncture, the patient developed a PK due to a subcapsular hematoma of 30mm (Figure 4). Conservative strategy was decided and the recipient needed to maintain hemodialysis three times a week. After one month of the iatrogenic bleeding, the patient increased diuresis and hemodialysis was not necessary even more. An ultrasound with doppler showed a decrease of the hematoma size (7mm), maintaining resistive indices mildly increased ranging from 0.86 to 0.88 throughout the transplant kidney. Kidney function presented a slow improvement with serum levels of creatinine of 160 µ mol/L in the last outpatient visit, three months after KT.

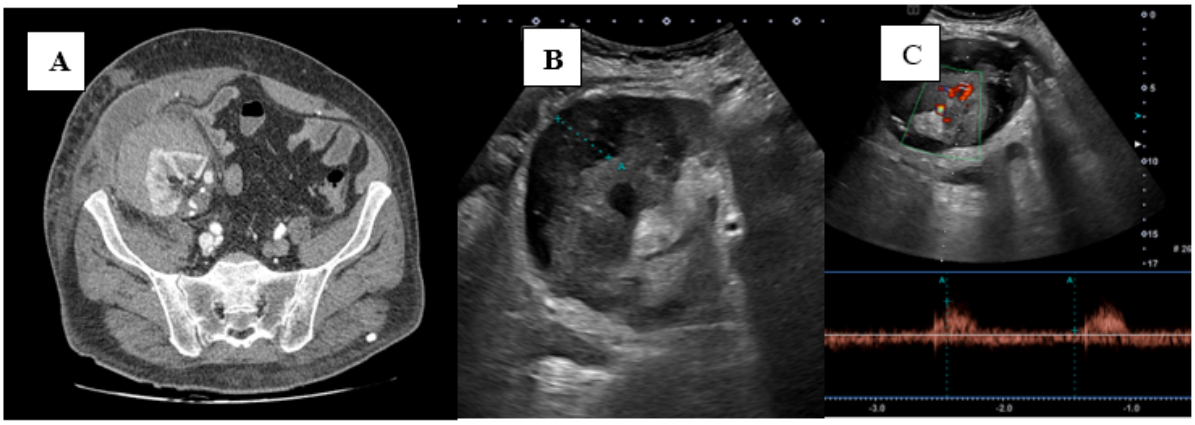

Figure 4: Patient #4 imaging studies at diagnosis. **A)** Axial computed tomography with contrast after KT **B)** Ultrasound. **C)** Doppler.

Discussion

Page kidney phenomenon or "Page Kidney"was first described in 1939 by Dr. Irvine Page [1,3]. He was able to induce a hypertensive response after compressing canine kidneys by wrapping them in cellophane. Hypoperfusion and microvascular ischemia activate the RAAS developing arterial hypertension. Although this activation can be quantified by measuring plasma renin, the measurement of which was not possible in these four cases. The typical presentation is hypertension and pain with or without kidney dysfunction. In KT, this phenomenon may lead to terminal kidney disease. Multiple causes have been described: hematomas (due to trauma, intervention, spontaneous…), neoplasms, cysts, lymphoceles, renal pathology… [4,5]. Possible clinical presentations and most frequent causes that have already been described in the literature are summarized in Table 1.

		N (%)
CAUSES	Iatrogenic: • Kidney Biopsy • Arterial stent • Ureteral stent • Nephrostomy	24 (65) 21 (57) 1 (3) 1 (3) 1 (3)
	Traumatism	4 (11)
	Complications: • Bleeding • Lymphocele	6 (16) 4 (11) 2 (5)
	Spontaneous	3 (8)
CLINICAL PRESENTATION	Hypertension	23(62)
	Pain	18 (49)
	Anuria	15 (41)
	Oliguria	13 (35)
	Hematuria	5(14)
	Hypotension	4(11)
	Nausea	2(6)

Table 1: Summary table of main causes and clinical presentations in the 37 cases published in the literature.

The diagnosis can be made by Doppler-ultrasound (observing absent or reverse venous flow and increased resistance indices together with an intra or perirenal space occupying lesion), Computed Tomography (CT) scan or selective arteriography [6] showing hypoperfusion, compression or ischemia of kidney parenchyma. In our four cases, Doppler-ultrasound and later CT scan, which showed subscapular hematomas and revealed renal ischemia, made the diagnosis. In the published cases, the average size of the hematomas that leads to PK was of 57 ± 13mm. There are different therapeutic approaches in KT: some clinicians wait that the compression resolves spontaneously (reabsorption or evacuation of a hematoma) [7-9], others indicate intervention, specially in case of larger collections or those that increase in size (surgical drainage or nephrectomy). In all of our four cases, conservative management was chosen, however, in the literature, the authors showed more interventionist attitudes (32/36 cases) (Table 2). Regarding the results, complete resolution of PK was found in only 66% of the cases with conservative treatment [7-9] compared to 89.2% of the cases with interventional management (28 capsulotomies [4,10-30], 3 drainages [9,31-34] and 1 nephrectomy [35]) (Table 2). Blood pressure was corrected after interventionism in nine cases that reported this result. Sixty-three percent of the procedures were performed immediately after diagnosis with a 95.2% success rate. In the cases in which the procedure was delayed (15%), the success rate was lower, with 80% of cases with a complete renal response. No capsulotomy-related complications were described in the reviewed cases. In our case series all cases resolved spontaneously.

Author	Age	Gender	Time after KT	Cause	Arterial pressure (mmHg)		Creatinine(µ mol/L)			Diagnostic	Intervention	Time before intervention	Results
					At diagnostic	After treatment	Before event	At diagnostic	After intervention				
Cromie et al 1976 [16]	35	M	10d	Post KT bleeding	194/100	140/80	124		124	Kidney scan, Doppler	Capsulotomy	2d	CR
Figueroa et al 1988 [17]	40	F	11m	Biopsy	184/104		168	415	203	Arteriography, CT scan	Capsulotomy	30h	CR
Yussim et al 1988 [33]	40	F	5m	Post KT scar	190/110	140/80	141	575	221	Kidney scan	Capsulotomy	Not known	RP

Kliewer et al 1991 [35]	56	F	2s	Biopsy					IRC	Doppler CT scan	Nephrec-tomy	Not known	GL
Dempsey et al 1993 [19]	19	F	2a	Biopsy				619	194	Doppler	Capsu-lotomy	Imme-diate	CR
Ben Hamida et al 1993 [36]	32	M	7m	Heparin induced bleed-ing						Doppler	Conser-vative		CR
Nguyen et al 1994 [20]	26	M	12h	Post KT bleed-ing	170/95	112/52	1282	893	177	Kidney scan	Capsu-lotomy	Imme-diate	CR
Machida et al 1996 [7]	32	M	4m	Biopsy	170/100	190/115	168	283	133	Doppler CT scan, Kidney scan	Conser-vador		CR
Vanwal-leghem et al 1997 [31]	59	F	2a	Lym-phocele	160/90	120/70	168	151		MRI	Drainage	Not known	CR
Rea et al 2000 [10]	34	M	3a	Biopsy			245	447	248	Doppler, CT scan	Capsu-lotomy	Imme-diate	CR
Gibney et al 2005 [21]	32	M	1a	Biopsy	190/89	150/85	124	592	124	Doppler	Capsu-lotomy	Imme-diate	CR
Patel et al 2007 [24]	69	M	7a	Biopsy	180/100	142/84	133	248	141	Doppler	Capsu-lotomy	Imme-diate	CR
Chung et al 2008 [25]	27	F	11d	Biopsy			689		131	Doppler, CT scan	Capsu-lotomy	Imme-diate	CR
Chung et al 2008 [25]	39	F	7d	Biopsy	195/105		265		82	Doppler, CT scan	Capsu-lotomy	Imme-diate	CR

Chung et al 2008 [25]	35	M	4d	Biopsy	180/100		498	588	IRC	Doppler, CT scan	Capsulotomy	Immediate	PR
Chung et al 2008 [25]	33	F	9m	Biopsy	200		89	243	75	Doppler, CT scan	Capsulotomy	Immediate	CR
Heffernan et al 2008 [26]	64	M	4m	Biopsy	160/70	115/70	123	388	160	Doppler	Capsulotomy	Immediate	CR
Kamar et al 2009 [8]	47	M	1a	Biopsy	170/110		159	283	177	Doppler	Conservative		CR
Kamar et al 2009 [8]	59	M	1a	Biopsy	160/90		106	380	124	Doppler	Conservative		CR
Amezquita et al 2009 [27]	60	M	1m	Nephrostomy	Normal	Normal	Normal	760	Normal	Doppler, CT scan	Capsulotomy	Not known	CR
Posadas et al 2010 [28]	55	M	3m	Biopsy	200/100	130/60	62	292	71	Doppler	Capsulotomy	Immediate	CR
Butt et al 2010 [1]	61	F	24d	Spontaneous	162/667		106	522	88	CT scan	Capsulotomy	Immediate	CR
Maurya et al 2011 [29]	30	M	7d	Biopsy			106	520	177	Doppler, CT scan	Capsulotomy	Immediate	CR
Okechukwu et al 2011 [30]	32	M	8d	UreteralStent			176.8	424	124	Doppler	Capsulotomy	Immediate	CR
Gandhi et al 2012 [22]	46	M	17a	Spontaneous	185/110		170	605	180	Doppler	Capsulotomy	Immediate	CR
Hamidian et al 2013 [9]	19	M	5s	ArterialStent			170	512	164	Doppler	Drainage	6h	CR

Adjei-Gyamfi et al 2014 [11]	12	M	7s	Biopsy			71	526	61	Doppler, CT scan	Capsulotomy	Immediate	CR
Adjei-Gyamfi et al 2014 [11]	18	F	1a	Biopsy			114	325	109	Doppler	Capsulotomy	Immediate	CR
Sedigh et al 2015 [12]	67	M	13a	Traumatism	110/70		62	241	72	Doppler	Capsulotomy	12h	CR
Kapoor et al 2016 [32]	42	F	Not known	Biopsy	210/110	130/70	292	371	274	CT scan, Doppler	Drainage	Not known	CR
Kumar et al 2017 [23]	66	M	4a	Traumatism	196/90		83	453	99	Doppler	Capsulotomy	Immediate	CR
Takahashi et al 2017 [34]	67	M	12a	Traumatism	163/54		176.8	477	IRC	CT scan, Doppler	Capsulotomy	3d	GL
Ay et al 2017 [13]	50	M	1d	Post KT bleeding			400	400	165	Doppler	Capsulotomy	Immediate	CR
McFadden et al 2018 [14]	63	M	6m	Biopsy	177/102	120/88	116	394	158	Doppler	Capsulotomy	Immediate	CR
Zvavanjanja et a 2018 [18]	42	M	5m	Biopsy	161/96		194	1317	194	Doppler	Capsulotomy	Immediate	CR
Hori et al 2018 [15]	66	M	2d	Post KT bleeding	145/80			530	150	Doppler	Capsulotomy	Not known	CR

Table 2: Summary of KT PK cases described in the English literature.

Conclusions

Page kidney is a rare disease in kidney transplantation with characteristic clinical features. Although different therapeutic schemes have been published in the literature with more frequent interventionist approaches, in our experience, conservative management obtained favorable results, avoiding the risks associated with interventional management.

Acknowledgment

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