Femoral Neuropathy in Renal Transplantation

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Abstract: Acute femoral neuropathy after renal transplantation is an uncommon and rarely recognized complication. Recovery of the nerve is usual. Although rare, five cases have come to our attention in the past twenty years. A detailed clinical and electrophysiological analysis with a six month follow-up is presented. A review of sixteen other reported cases is also provided. The possible pathophysiology including direct compression and nerve ischemia, is discussed. We believe that nerve ischemia, possibly caused by a steal phenomenon, occurs in all cases following the anastomosis of the graft renal artery to the internal iliac artery, with a superimposed component of compression in some cases. The severity of ischemia probably determines the degree of recovery.

Résumé: La neuropathie fémorale suite à la transplantation rénale. La neuropathie fémorale aiguë après une transplantation rénale est une complication rare et peu reconnue. La récupération nerveuse est de règle. Bien que rare, nous en avons identifié cinq cas dans les vingt dernières années. Nous présentons une analyse clinique et électorphysiologique détaillée avec un suivi de six mois. Nous présentons également une revue de seize autres cas rapportés dans la littérature. Nous discutons de la physiopathologie possible, dont la compression directe et l'ischémie nerveuse. Nous croyons que l'ischémie, possiblement causée par un phénomène de spoliation survient chez tous les cas à la suite de l'anastomose de l'artère rénale du greffon à l'artère iliaque interne, avec une composante de compression surajoutée dans certains cas. La sévérité de l'ischémie détermine probablement le degré de récupération.

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The occurrence of femoral neuropathy in patients with renal disease complicated by diabetes is well recognized. This usually takes the form of a so called "lumbar plexopathy", involving several other nerves in the lumbar plexus. In contrast, there are only a few published reports of femoral neuropathy following renal transplantation.[1-9] We report five cases of acute femoral neuropathy occurring ipsilateral to and immediately after renal transplantation. The clinical and the electro-physiologic features and the possible mechanisms of involvement are discussed.

Methods

A total of 654 renal transplants were carried out between the years 1972 and 1992 at St. Michael's Hospital, Toronto, Ontario. The transplant database and charts were reviewed retrospec-tively. Five cases of postoperative "femoral neuropathy" had been recorded in the entire database during this interval. All patients had been examined by a consultant neurologist.

The MRC criteria for grading severity of weakness and the subsequent recovery are summarized in Table 1.[10]

Post-operatively, the patients had routine biochemical tests done to assess renal function. All patients had a pelvic ultrasound examination on developing the neurological syndrome, to rule out intrapelvic hemorrhage.

Electrophysiological examination was performed three to four weeks after presentation. This consisted of bilateral sensory nerve conduction studies of the median, ulnar, tibial and sural nerves, and motor conduction studies of the median, ulnar and peroneal nerves. F wave latencies as well as H responses were examined in the peroneal and tibial nerves respectively. Electromyographic examination was done on both quadriceps, as well as tibialis anterior and gastrocnemius muscles ipsilateral to the affected lower extremity. The presence of spontaneous activity in the form of positive waves and fibrillation potentials, as well as large amplitude potentials was defined as being compatible with denervation.[11]

Results

Patient Summaries

Patient 1

This 40-year-old hypertensive male, with diabetic glomerulopathy, underwent an uncomplicated right-sided cadaveric renal transplant. Two days post-operatively, he complained of right leg heaviness. The only neurological finding was weakness in right hip flexor (Grade 2) and knee extensor (Grade 3), with an absent right knee jerk and numbness over the anterior thigh. A mild increase in pin-prick threshold was noted up to the ankles. Electrophysiological testing confirmed a mild distal sensory neuropathy. Acute denervation was seen only in the right quadriceps muscle. At 6 months, there were no sensory abnormalities with motor function in both affected muscles recovering to Grade 4.

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Table 1.[10] Diagnostic Criteria.

Grading of Muscle Power	
Grade 0 = No muscle contraction	(0%)
Grade 1 = Flicker	(0-10% normal)
Grade 2 = Partial movement with no gravity	(11-25% normal)
Grade 3 = Complete movement against gravity	(26-50% normal)
Grade 4 = Complete movement against variable resistance	(51-75% normal)
Grade 5 = Normal power	(100% normal)

Rating of Recovery		
Poor	=	No recovery
Fair	=	25-50% recovery
Good	=	50-75% recovery
Excellent	=	75-100% recovery

Patient 2

This 39-year-old male, with a history of hyperlipidemia and proliferative glomerulonephritis, received a cadaveric renal transplant in the left iliac fossa without any intraoperative complications. Attempting to ambulate on the third post-operative day, left lower extremity weakness was noted. Neurological examination confirmed an isolated left hip flexor (Grade 2) and knee extensor (Grade 1) weakness with an absent knee jerk and numbness along the antero-medial thigh. Electrophysiology showed complete absence of motor units in the left quariceps muscle with occasional positive waves. By 6 months, there was complete motor (Grade 5) and sensory recovery in the affected territory.

Patient 3

This 32-year-old hypertensive female, with chronic glomerulonephritis underwent uncomplicated cadaveric renal transplantation in the left iliac fossa. One day post-operatively, weakness was observed in the left lower extremity. Clinically, only the left hip flexor (Grade 3) and knee extensor (Grade 2) were weak, with an absent knee jerk and sensory loss over the antero-medial thigh and medial calf. These findings were confirmed on electrophysiology with the left quadriceps denervated. At 6 months, left hip flexor (Grade 5) and knee extensor (Grade 4) power and sensation had virtually recovered. She died of septic shock, 8 months after the operation.

Patient 4

A 40-year-old hypertensive, diabetic male with polycystic kidney disease had an uncomplicated left sided cadaveric renal transplant. Two days post-operatively, he was unable to ambulate. Examination revealed marked weakness of the left hip flexor (Grade 1) and knee extensor (Grade 0). The left knee jerk was absent with sensory loss over the antero-medial thigh and the entire medial calf. Electrophysiological study showed no motor units in the left quadriceps with definite positive waves. A slight sensory conduction delay was seen in both sural and tibial nerves. Six month follow-up showed persistently impaired motor function of the left hip flexor (Grade 3) and knee extensor (Grade 2), with wasting of the left quadriceps.

Patient 5

This 18-year-old female with chronic glomerulonephritis received a cadaveric left iliac fossa renal transplant. Difficulty ambulating was noted thirty-six hours later. Examination showed weakness in the left hip flexor (Grade 4) and knee extensor (Grade 3) with loss of the knee reflex and numbness along the anterior thigh. Electrophysiological testing showed normal nerve conductions with signs of denervation in the left quadriceps muscle. Six months recovery was complete in the hip flexor and knee extensor (Grade 5).

Summary

The clinical data are summarized in Table 2. The patient ages ranged between 18 and 40. There were three males and two females. The etiology of renal dysfunction was variable and two patients were diabetic. All patients were on hemodialysis and had been well dialysed pre-operatively. All of the transplanted kidneys were placed in the iliac fossa on the side ipsilateral to the femoral neuropathy. In every patient, the paralysis occurred immediately following the operation with difficulty on ambulation. Physical examination revealed unilateral hip flexion and knee extension (quadriceps) weakness, an absent knee jerk and numbness over the anterior aspect of the thigh, always on the side of the transplant. There were no other neurological abnormalities.

A mild, distal, symmetrical, sensory and sensory-motor neuropathy was detected in two patients, both of whom were diabetic. Spontaneous activity in the form of positive waves and/or fibrillation potentials was seen in the quadriceps muscle in all cases, consistent with denervation. Peroneal F waves and tibial H responses were normal in all cases.

The details of the follow up are summarized in Table 3. In our five cases of post renal transplantation femoral neuropathy, recovery was excellent in three, good in one and poor in another at six month follow-up. One patient died eight months after the transplant from septic shock. There was no apparent relationship between the success of the transplanted kidney and recovery from the femoral nerve palsy. Three of our five patients achieved excellent renal function, with concommitant femoral nerve recovery being excellent in only one and good in the other two. Patient four had excellent renal function but poor femoral nerve function.

There was no clinical or electrophysiological evidence favoring a diagnosis of a lumbar plexopathy. The absence of involvement of any other nerves and preserved F and H responses supported a diagnosis of an isolated femoral neuropathy.

Discussion

Femoral neuropathy has been described as a complication of diabetes mellitus, compression due to haematoma from anticoagulation,[12-15] pelvic surgery[16-20] radiotherapy[21] and malignancy.[22] One case of post renal transplantation femoral neuropathy due to direct compression from a retroperitoneal hematoma has also been reported.[1]

Only sixteen cases of femoral neuropathy have been reported after renal transplantation, in the combined adult and pediatric literature.[2-8] None of the above-mentioned mechanisms were relevent in these cases. Details as to the grading of the severity of the paresis are not consistently available in any of the studies. In the most recent study by Meech et al. no electromyographic evaluation was done.[8]

Recovery was excellent in seven, good in three, fair in four and poor in only two patients, although the extent of follow-up was inconsistent.[2-8] No mention is made of associated diabetes mellitus in all except one paper where this clearly excluded.[8] Although no definite conclusions can be drawn regarding recovery of the neuropathy and transplant function those with better renal function appeared to have a faster recovery.[2-8]

A complete summary of all reported cases with the available details is provided in Table 4.

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Table 2. Case Summaries (1972 - 1992) – Clinical.

Patient #	Age/ (Sex)	Renal Disease	Associated Disease	Onset Post-Op.	Findings	EMG & NCS (3 Weeks Post Op.)
1 (1992)	40 (M)	Glomerulopathy	Hypertension Diabetes	2 Days	R. Hip Flexor (Gr. 2) R. Quads (Gr. 3) Absent Knee Jerk Numb Anterior Thigh	Absent Peroneal & Sural Sensory Denervation in R. Quads.
2 (1982)	39 (M)	Proliferative G.N.	Hyperlipidemia	3 Days	L. Hip Flexor (Gr. 2) L. Quads (Gr. 1) Absent Knee Jerk Numb Ant-Med. Thigh	Normal Conductions No Motor Units & Occasional (+)VE Waves in L. Quads.
3 (1977)	32 (F)	Chronic G.N.	Hypertension	1 Day	L. Hip Flexor (Gr. 3) L. Quads. (Gr. 2) Absent Knee Jerk Numb Ant-Med. Thigh & Medial Calf	Normal Conductions No Motor Units & Degenervation in L. Quads.
4 (1973)	40 (M)	Polycystic Kidney	Hypertension Diabetes	2 Days	L. Hip Flexor (Gr. 1) L. Quads. (Gr. 0) Absent Knee Jerk Numb Ant-Med. Thigh & Medial Calf	Mild Sensory Delay No Motor Units & Denervation in L. Quads.
5 (1973)	18 (F)	G.N.	Nil	36 Hours	L. Hip Flexor (Gr. 4) L. Quads (Gr. 3) Absent Knee Jerk No Sensory Loss	Normal Conductions Early Denervation L. Quads

G.N. = Glomerulonephritis Quads. = Quadriceps

Table 3. Case Summaries (1972 - 1992) – Six Month Follow-up.

Patient # Outcome	1 (1992)	2 (1982)	3 (1977)	4 (1973)	5 (1973)
Function	Good	Excellent	Excellent	Poor	Excellent
Hip Flexor Knee Extensor	Gr. 4 Gr. 4	Gr. 5 Gr. 5	Gr. 5 Gr. 4	Gr. 3 Gr. 2	Gr. 5 Gr. 5
Sensation Recovery	Normal Full	Normal Full	Normal Full	Abnormal Partial	Normal Full
Transplant Function	Excellent	Excellent	Rejection Deceased 8 Months Post-Op.	Excellent	Fair Single Rejection

Anatomy

The femoral nerve originates from the anterior rami of the three lumbar roots L2, L3 and L4 forming in the psoas muscle itself. The branch to the psoas muscle may originate here while the femoral nerve is still a part of the lumbar plexus or after the nerve is fully constituted, although some controversy exists. After emerging through the psoas, it lies in the iliopsoas groove for the distal one-third of its intrapelvic course. The nerve then passes deep to the iliopsoas fascia emerging under the inguinal ligament, postero-lateral to the external iliac artery.[23] It then divides into the anterior and posterior divisions, approximately 1½" below the inguinal ligament. The anterior division gives rise to the medial and intermediate cutaneous nerves of the thigh and muscular branches to sartorius and pectinius muscles. The posterior division supplies the quadriceps femoris muscle and then continues on along the medial border of the calf as the saphenous nerve, a sensory nerve.[23]

The iliac ramus of the iliolumbar artery (a branch of the internal iliac artery) supplies the intrapelvic portion of the femoral nerve, accompanying it longitudinally with occasional supply from the lumbar rami.[24] The proximal and distal portions of the femoral nerve receive vascularization from the local branches. This makes the middle portion of the femoral nerve the most vulnerable section as it lies in the third intrapelvic region, just proximal to the inguinal ligament.

Operative Procedure

An incision is made in the appropriate lower quadrant curvi-

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Table 4. Summary of Reported Post-Transplanation Femoral Neuropathy Cases (1976 - 1992).

Patient #	Author/ Year	Age/ (Sex)	Renal Disease	Onset	Follow Up	Outcome	Transplant Function
1	Vaziri (2,4) (1976)	33 (F)	Chronic G.N.	Immed (*)	8 Months	Fair	Early Rejection
2	(1981)	30 (F)	Lupus Nephritis	Immed (*)	8 Weeks	Fair	Fair
3		22 (F)	Chronic G.N.	Immed (*)	7 Months	Poor	Poor
4		51 (M)	Nephrosclerosis	Immed (*)	10 Days	Excellent	Excellent
5	Pontin (3) (1978)	45 (M)	Chronic G.N.	Immed (*)	18 Months	Motor Poor Sensory Fair	Good
6		42 (F)	Unknown	Immed (*)	4 Months	Motor Fair Sensory Good	Good
7	Yazbeck (5) (1985)	18 (M)	Reflux Glomerulopathy	Immed (*)	1 Month	Good	Failed
8		16 (M)	Focal Glomerular Sclerosis	1 Day	2 Months	Motor Excellent Sensory Fair	Failed
9	Kumar (6) (1991)	18 (M)	Unknown	3 Days	6 Months	Motor/Sensory Excellent	Excellent
10	Meech (8) (1990)	58 (M)	Polycystic Kidney	Immed (*)	6 Weeks - 9 Months	Excellent	Good
11		52 (M)	Polycystic Kidney	Immed (*)	6 Weeks - 9 Months	Excellent	Good
12		30 (M)	Focal Glomerular Sclerosis	Immed (*)	6 Weeks - 9 Months	Excellent	Rejection
13		31 (F)	G.N.	Immed (*)	6 Weeks - 9 Months	Good	Rejection
14	Vogels (7) (1987)	5 (M)	Focal Glomerular Sclerosis	6 Days	5 Months	Motor Excellent Sensory (?)	Excellent
15		16 (M)	Renal Dysplasia	3 Days	12 Months	Good	Excellent
16		12-7/12 (M)	Cystinosis	Immed (*)	8 Months	Fair	Excellent

G.N. = Glomerulonephritis Immed (*) = Period Unspecified

linearly from the symphisis pubis to the anterior superior iliac spine.[25-27] After dissection through the rectus abdominis, external and internal obliques and transversus abdominis muscles, the peritoneum is retracted medially. Self retaining retractors are used for this purpose with two medial and lateral blades. The iliac vessels are then exposed and the overlying tissue is carefully separated so as to preserve the lymphatics. Once freed, either the external or internal iliac vessels can be used for anastomosis. Venous end-to-end anastomosis is performed by placing proximal and distal vascular clamps. Similarly, arterial anastomosis is begun by placing proximal clamps on the internal iliac artery. Then, an end-to-end anastomosis is performed with a subsequent endarterectomy of the distal interal iliac vessel. The venous and arterial clamps are then released.

The kidney is placed in the iliac fossa. The femoral nerve and lumbar plexus are never directly exposed during the entire procedure but the middle portion of the nerve lies near the inferomedial blade, as it emerges from the psoas.

Pathophysiology

There are several possible mechanisms of involvement of the femoral nerve in patients who undergo renal transplantation. Hefty et al.[9] have reported acute lumbosacral plexopathy in diabetic women after renal transplantation. The mechanism that is invoked is one of vascular predisposition with subsequent ischemia of the lumbosacral plexus from internal iliac artery ligation. No explanation is given as to why, in their series, male patients with diabetes who had undergone the same procedure failed to develop a similar complication.[9] The lumbar plexus has a rich anastomotic vascular supply especially from the inferior mesenteric and vesical arteries. On the contrary, the middle and distal intrapelvic portions of the femoral nerve are dependent on the integrity of the internal iliac artery for their vascular supply.[12,14] The renal artery of the donor kidney is anastomosed to this internal iliac artery.

Although direct compression or compromise of vascular structures probably does not occur, the possibility of significant localised "steal" does exist. Proximal end-end anastomosis of the renal artery to the internal iliac artery can shunt blood away from the vasa nervorum. Ligation of the distal portion of the internal iliac artery will only add further insult. The presence of additional factors such as diabetes and the existence of local atherosclerosis clearly make the femoral nerve susceptible to ischemic injury.

The most common mechanism invoked[1-9] is one of instrument-induced injury during the operative procedure. Although

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the femoral nerve is not exposed usually during the procedure, damage from stretching, diathermy and direct downward compression from retractors are possible etiologic factors. The surgical site itself is actually well above the psoas and quite distant from the course of the femoral nerve. Hence the former two causes are unlikely. Nerve compression caused by prolonged application of self-retaining retractors is a feasible explanation. Although the inferior and medial blades of the retractor are in proximity to the middle portion of the femoral nerve, care is taken to avoid such direct entrapment. This is the most vulnerable portion of the femoral nerve. Therefore, if compression were to in fact occur it would likely produce ischemia giving rise to the neuropathy.

Milder injuries, that is those that recover rapidly, might be neuropraxic due solely to retraction and mild transient ischemia. On the other hand, more severe and prolonged ischemia from vascular interruption may result in axonal loss and a slow, incomplete recovery. Haematoma formation as a cause of femoral neuropathy was ruled out in our patients by radiologic investigations and lack of the appropriate history of use of anticoagulants.

Femoral neuropathy as a complication of renal transplantation has an incidence of less than 1% (0.77%) in our series. This is an infrequent but important problem. The anastomosis of the renal artery of the graft to the internal iliac artery with vascular compromise due to a steal phenomenon is a plausible explanation.

Mechanical compression from surgical blades is improbable. Such a mechanism may be relevant only if superimposed on a nerve that may be suffering vascular compromise. Extra precaution should be taken in patients with diabetes mellitus, as well as advanced atherosclerosis.

The outcome of the neuropathy is favourable in most cases within six months and does not appear to be related to the function of the transplanted kidney.

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