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Case Report

Guillain-Barré syndrome after living-related renal transplantation: a case report and literature review

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ABSTRACT

Guillain-Barré syndrome (GBS) is a rare but potentially life-threatening neurological complication after kidney transplantation. Diagnosis may be challenging because its presentation can mimic calcineurin-inhibitor neurotoxicity, metabolic disturbances, or infection-related neuromuscular disorders. A 34-year-old man with a living-related renal transplant performed in 2020 presented with a two-day history of progressive ascending weakness, distal paresthesia, and an inability to ambulate following a self-limited diarrheal illness. A neurological examination revealed symmetric flaccid quadriparesis and generalized areflexia. Respiratory deterioration necessitated ventilatory support, while renal allograft function remained stable throughout his hospitalization. Cerebrospinal fluid analysis demonstrated albuminocytologic dissociation, and nerve-conduction studies showed prolonged distal and F-wave latencies with reduced conduction velocities. These findings were consistent with acute inflammatory demyelinating polyradiculoneuropathy. Cytomegalovirus DNA polymerase chain reaction testing was negative. The patient received intravenous immunoglobulin (2 g/kg over 5 days), ventilatory support, and intensive rehabilitation. Progressive neurological recovery began within the first week of treatment. He was successfully weaned from ventilatory support and regained independent ambulation. At the 3-month follow-up, his motor strength had completely recovered, with preserved renal allograft function and no neurological sequelae. GBS should be considered in kidney transplant recipients presenting with acute progressive weakness, even in the absence of active cytomegalovirus infection or graft dysfunction. Early cerebrospinal fluid evaluation, electrophysiological testing, and prompt administration of intravenous immunoglobulin can result in complete neurological recovery while preserving allograft function.

Keywords: Guillain-Barré syndrome, Kidney transplantation Cytomegalovirus, Intravenous immunoglobulin, Acute inflammatory demyelinating polyradiculoneuropathy.

INTRODUCTION

Guillain-Barré syndrome (GBS) is a rare but potentially life-threatening acute immune-mediated polyradiculo-neuropathy that can progress rapidly to generalized weakness, autonomic instability, and respiratory failure.[1] In kidney transplant recipients, recognition is particularly challenging because new weakness may be misattributed to calcineurin-inhibitor neurotoxicity, electrolyte abnormalities, infection-related myopathy, cerebro-vascular events, or critical illness neuropathy.[2-5] In the post-renal transplant setting, cytomegalovirus is the most frequently reported trigger, although Campylobacter jejuni infection, Epstein-Barr virus, antithymocyte globulin exposure, calcineurin inhibitors, and paraneoplastic disease have also been described.[2-14] Diagnosis relies on clinical suspicion supported by cerebrospinal fluid and electrophysiological findings, but these may be normal early in the illness; therefore, prompt recognition is essential because intravenous immunoglobulin and plasma exchange remain the only proven disease-specific therapies.[1]

We report a kidney transplant recipient who developed rapidly progressive motor-predominant weakness following an antecedent diarrheal illness, with preserved allograft function and a favorable response to intravenous immunoglobulin. This case adds to the limited literature on post-transplant GBS and highlights the importance of considering immune-mediated neuropathy early when acute weakness develops after transplantation, even when graft function is stable.[2,4,5,7,9-14]

CASE REPORT

A 34-year-old man underwent living-related kidney transplantation in 2020 for end-stage kidney disease secondary to chronic glomerulonephritis. The donor was his 52-year-old mother. Both donor and recipient were cytomegalovirus (CMV) IgG positive and IgM negative, with an HLA match of 3/6. The patient was maintained on tacrolimus, mycophenolate mofetil, and corticosteroids, with stable allograft function and a baseline serum creatinine of approximately 1.2 mg/dl. The baseline clinical characteristics of the donor and recipient are summarized in Figure 1.

The patient presented to the Department of Nephrology at Shankus Hospital, Mehsana, Gujarat, India, with a 2-day history of progressive lower-limb weakness that initially manifested as difficulty rising from a chair, climbing stairs, and walking. The weakness subsequently ascended and was accompanied by distal paresthesia involving the feet and hands, resulting in loss of independent ambulation. Neurological symptoms were preceded by a self-limited diarrheal illness approximately three weeks earlier. There was no history of fever, rash, recent vaccination, cranial nerve symptoms, bowel or bladder dysfunction, or exposure to neurotoxic medications beyond his maintenance immunosuppressive regimen. On admission, he was afebrile, hemodynamically stable, and fully oriented. Neurological examination revealed symmetric flaccid weakness predominantly affecting the lower limbs, with Medical Research Council (MRC) grades of 2/5 in the lower extremities and 4/5 in the upper extremities. Deep tendon reflexes were absent in all four limbs. Sensory examination demonstrated reduced light-touch and pinprick sensation in a stocking-glove distribution. Cranial nerve examination was normal, and there were no cerebellar or meningeal signs. Over the ensuing 48 hours, weakness progressed rapidly, leading to respiratory compromise requiring ventilatory support in the intensive care unit. The serial neurological findings are summarized in Table 1. Initial laboratory investigations demonstrated mild anemia and leukocytosis, with preserved platelet counts and stable renal allograft function throughout the clinical course.

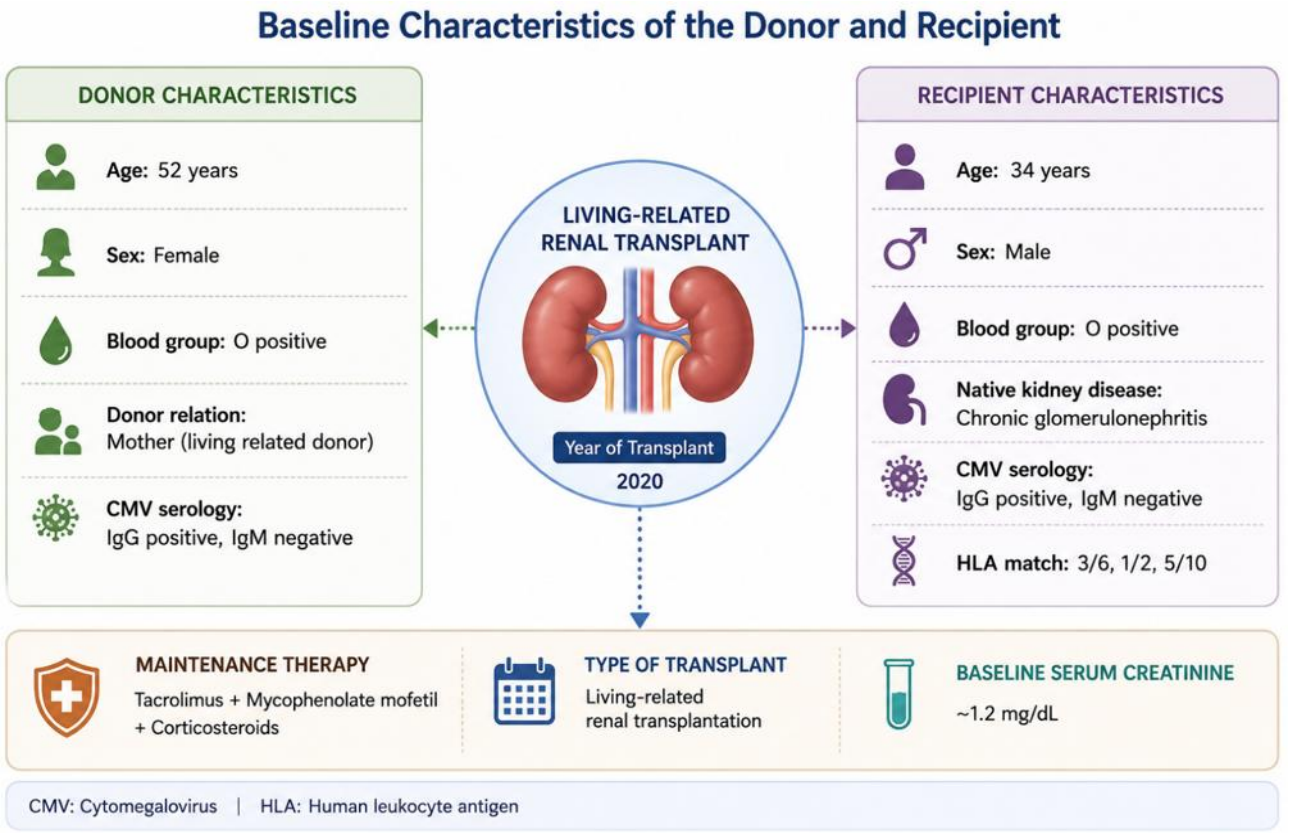

Figure 1. Baseline characteristics of the donor and recipient.
Infographic summarizing demographic, transplant-related, immunological, and clinical characteristics of the living-related kidney transplant recipient and donor.

Serum electrolytes, calcium, magnesium, and creatine phosphokinase levels were within normal limits and did not support an alternative metabolic or myopathic etiology. CMV DNA polymerase chain reaction testing was negative. Serial laboratory results are shown in Table 2.

Table 1. Serial neurological examination findings

Examination domains	Day 1 (admission)	Day 3 (peak disability)	Day 12 (discharge)	3-month follow-up
Mental status	Alert and oriented	Alert, anxious	Alert and oriented	Alert and oriented
Cranial nerves	Intact	Intact	Intact	Intact
Motor system - upper limbs	Power 4/5 bilaterally	Power 3/5 bilaterally	Power 4+/5 bilaterally	Power 5/5 bilaterally
Motor system - lower limbs	Power 2/5 proximally and distally	Power 1/5 throughout	Power 3/5 throughout	Power 5/5 throughout
Sensory system	Reduced light touch and pinprick in stocking-glove distribution	Markedly reduced sensation to mid-forearm and mid-thigh	Mild residual paresthesia in fingertips and toes	Minimal residual paresthesia in fingertips only
Reflexes	Areflexic in all limbs	Areflexic	Hyporeflexic (1+)	Normoreflexic (2+)
Coordination	Not testable due to weakness	Not testable	Mild dysmetria on finger-nose testing	Normal coordination
Gait	Not ambulatory	Bedbound, requiring ventilatory support	Able to walk with walker	Normal gait

Table 2. Serial laboratory investigations

Tests	Reference range	Day 1 (Admission)	Day 5	Day 12 (Discharge)
Hemoglobin (g/dl)	13.5-17.5	11.5	11.4	11.8
White blood cell count (/mm3)	4,000-10,000	10,420	9,800	8,900
Platelets (/mm^3)	150,000-400,000	333,000	320,000	310,000
Serum creatinine (mg/dl)	0.6-1.3	1.2	1.2	1.2
Sodium (mEq/l)	135-145	136.7	138	139
Potassium (mEq/l)	3.5-5.0	5.5	4.8	4.5
Calcium (mg/dl)	8.5-10.5	9.0	9.1	9.2
Magnesium (mg/dl)	1.7-2.4	1.76	1.8	1.9
CPK (U/l)	20-200	37	35	34
CMV DNA PCR	Negative	Undetectable	Undetectable	Undetectable
CSF protein (mg/dl)	15-45	86.2	-	-
CSF cells (/mm3)	<5	5	-	-

Table 3. Nerve Conduction Study Results (Day 4)

Nerve	Stimulation site	Distal latency (ms)	Amplitude (mV)	Conduction velocity (m/s)	F-wave latency (ms)	Interpretation
Median	Wrist, elbow	6.2 (N<4.4)	4.2 (N>4.0)	35 (N>50)	68 (N<31)	Severe demyelinating abnormality
Ulnar	Wrist, below elbow, above elbow	6.8 (N<3.3)	6.2 (N>6.0)	32 (N>50)	72 (N<32)	Severe demyelinating abnormality
Peroneal	Ankle, fibular head	8.5 (N<5.8)	3.2 (N>2.0)	28 (N>41)	78 (N<56)	Severe demyelinating abnormality
Tibial	Ankle, popliteal fossa	9.1 (N<6.1)	4.3 (N>4.0)	26 (N>41)	82 (N<58)	Severe demyelinating abnormality

Values in parentheses indicate normal reference ranges. The electrophysiological pattern is most consistent with a predominantly demyelinating polyradiculoneuropathy

Cerebrospinal fluid analysis revealed albuminocytologic dissociation, with elevated protein concentration and minimal cellularity. The nerve-conduction study demonstrated markedly prolonged distal motor and F-wave latencies with significantly reduced conduction velocities and relatively preserved compound muscle action potential amplitudes, consistent with a predominantly demyelinating sensorimotor polyradiculoneuropathy compatible with acute inflammatory demyelinating polyradiculoneuropathy (AIDP). Detailed electrophysiological findings are presented in Table 3. The differential diagnosis included Guillain-Barré syndrome, tacrolimus-associated neurotoxicity, infectious polyradiculoneuropathy, critical illness neuropathy, acute myositis, and structural spinal pathology. Based on the characteristic clinical presentation, cerebrospinal fluid findings, and electrophysiological abnormalities, a diagnosis of Guillain-Barré syndrome was established. The patient received intravenous immunoglobulin at a total dose of 2 g/kg administered over 5 days, together with ventilatory support, intensive physiotherapy, and comprehensive supportive care. Maintenance immunosuppressive therapy was continued with close monitoring of graft function. Neurological recovery became evident within the first week of treatment. The patient was successfully weaned from ventilatory support, regained the ability to sit and stand with assistance, and subsequently achieved ambulation with a walker before discharge. At 3-month follow-up, neurological examination demonstrated complete recovery, with normal muscle strength, intact cranial nerve function, restoration of independent gait, and no residual sensory deficits. Renal allograft function remained stable throughout follow-up. The patient's clinical course, diagnostic evaluation, treatment, and recovery are summarized in Figure 2.

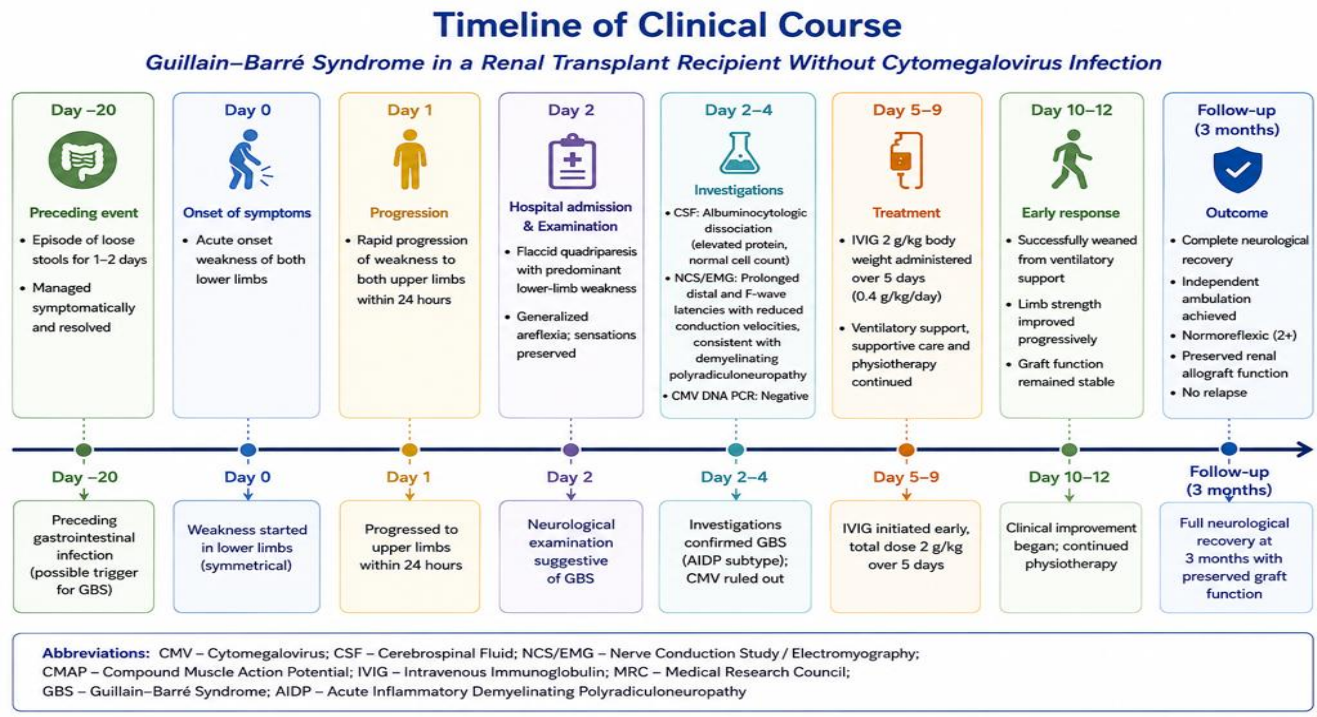

Figure 2. Clinical timeline of Guillain-Barré syndrome after kidney transplantation.

Timeline illustrating symptom onset, neurological progression, diagnostic evaluation, intravenous immunoglobulin therapy, ventilatory support, and complete neurological recovery with preserved renal allograft function at 3-month follow-up.

DISCUSSION

GBS is an uncommon but potentially life-threatening neurological complication after kidney transplantation. Although most episodes of weakness in transplant recipients are attributable to metabolic abnormalities, medication-related neurotoxicity, infection, or cerebrovascular disease, GBS should remain an important diagnostic consideration because delayed recognition may result in respiratory failure and prolonged disability.[1,2] The present case highlights the diagnostic challenges associated with acute neuromuscular weakness in transplant recipients and demonstrates that timely diagnosis and treatment can result in complete neurological recovery without compromising allograft function.

Several clinical features strongly supported the diagnosis of GBS in our patient. The illness was preceded by a self-limited diarrheal episode, followed by rapidly progressive symmetrical ascending weakness, distal paresthesia, generalized areflexia, and subsequent respiratory

compromise. Cerebrospinal fluid analysis demonstrated albuminocytologic dissociation, while nerve-conduction studies revealed prolonged distal motor latencies, reduced conduction velocities, and prolonged F-wave latencies, findings characteristic of a demyelinating polyradiculoneuropathy.[1] Collectively, these findings were most consistent with acute inflammatory demyelinating polyradiculoneuropathy (AIDP), the commonest electrophysiological subtype of GBS reported worldwide.[1]

Table 4. Representative published reports of Guillain-Barré syndrome after renal transplantation

Year/Author [Ref.]	Patient characteristics	Trigger / associated factor	Key clinical findings	Treatment	Outcome
Present case	34-year-old man; living-related renal transplant recipient	Antecedent diarrheal illness; CMV PCR negative	AIDP with albuminocytologic dissociation and demyelinating nerve-conduction abnormalities	IVIG (2 g/kg over 5 days), ventilatory support, and rehabilitation	Complete neurological recovery with preserved allograft function at 3 months
Lopes et al[14]	8-year-old pediatric kidney transplant recipient	CMV infection	CMV-associated GBS with progressive weakness	Antiviral therapy + IVIG	Complete neurological recovery
Tavakoli et al [5]	Adult kidney transplant recipient	Rabbit antithymocyte globulin exposure	Progressive ascending weakness following ATG administration	IVIG	Significant neurological improvement
Zakrocka et al[13]	47-year-old man; second kidney transplant recipient	Allograft renal cell carcinoma	Paraneoplastic GBS	Malignancy-directed therapy and supportive care	Neurological improvement after treatment of malignancy
Merzkani et al [12]	Living-related renal transplant recipient	Primary CMV infection with viremia	AIDP with albuminocytologic dissociation	Valganciclovir + plasma exchange + IVIG	Good neurological recovery
Shaban et al [11]	Kidney transplant recipient	Late-onset CMV disease	GBS associated with active CMV infection	IVIG + antiviral therapy	Good recovery with preserved graft function
Jakes et al [4]	Renal transplant recipient	Tacrolimus/basiliximab exposure considered	Demyelinating neuropathy presenting a diagnostic challenge	IVIG and immunosuppressive regimen review	Complete recovery
Masajtis-Zagajewska et al[10]	Kidney transplant recipient	Epstein–Barr virus infection	GBS occurring during EBV infection	Supportive therapy	Recovered
García-Álvarez et al [3]	Kidney transplant recipient	No specific precipitating factor identified	GBS developing after renal transplantation	Immunomodulatory and supportive therapy	Recovered
Keithi-Reddy et al [9]	Cadaveric renal allograft recipient	CMV disease	GBS associated with active CMV infection	Antiviral therapy and GBS-directed management	Clinical improvement
Murali et al [8]	Immunocompromised patient with previous kidney transplantation	Coccidioidomycosis infection	GBS associated with systemic fungal infection	Antifungal therapy and supportive care	Recovered

Abbreviations: AIDP: Acute inflammatory demyelinating polyradiculoneuropathy, ATG: Antithymocyte globulin, CMV: Cytomegalovirus, EBV: Epstein-Barr virus, GBS: Guillain-Barré syndrome, IVIG: Intravenous immunoglobulin.

The differential diagnosis of acute weakness after kidney transplantation is broad. Calcineurin inhibitors, particularly tacrolimus, may cause tremor, encephalopathy, seizures, posterior reversible encephalopathy syndrome, and, less frequently, peripheral neuropathy.[2,4,5] However, the combination of ascending weakness, generalized areflexia, albuminocytologic

dissociation, and characteristic electrophysiological abnormalities strongly favored GBS in the present case.

Critical illness neuropathy was considered less likely because neurological symptoms preceded intensive care admission and evolved before respiratory failure developed. Similarly, acute myositis was not supported by normal creatine phosphokinase levels, while structural spinal pathology was unlikely in the absence of sphincter dysfunction or upper motor neuron signs.

Previous reports have identified cytomegalovirus (CMV) infection as the most frequently reported trigger of GBS after kidney transplantation.[2,6,9,11,12,14] Other reported associations include Campylobacter jejuni infection, Epstein-Barr virus infection, antithymocyte globulin exposure, calcineurin-inhibitor therapy, and paraneoplastic syndromes.[3,5,7,10,13] Representative cases from the literature are summarized in Table 4.

These observations suggest that post-transplant GBS represents a final common immune-mediated pathway triggered by infectious, pharmacological, or neoplastic stimuli in susceptible individuals. Consequently, evaluation should extend beyond confirmation of GBS and include a systematic search for potentially reversible precipitating factors.

In the present case, the antecedent diarrheal illness raised the possibility of Campylobacter-associated molecular mimicry, a well-recognized mechanism in GBS pathogenesis.[1,7] Although microbiological confirmation was unavailable, the clinical history was compatible with a post-infectious immune-mediated neuropathy.

Importantly, CMV DNA polymerase chain reaction remained negative throughout the evaluation, reducing the likelihood of CMV-associated polyradiculopathy. Unlike several previously reported transplant-associated cases, our patient developed GBS in the absence of active CMV disease or graft dysfunction, emphasizing that GBS may occur even in clinically stable transplant recipients.

Early treatment is essential because neurological deterioration may be rapid and respiratory failure can develop within days of symptom onset.[1] Current international guidelines recommend intravenous immunoglobulin (IVIG) or plasma exchange as first-line therapy, with comparable efficacy when administered early in the disease course.[1] Our patient received IVIG promptly after diagnosis, followed by ventilatory support and intensive rehabilitation.

As illustrated in Figure 2, neurological recovery began within the first week of treatment, permitting successful liberation from ventilatory support and progressive restoration of motor function. Complete neurological recovery was achieved at 3-month follow-up, with preserved renal allograft function and no evidence of relapse.

This case underscores several important clinical lessons. Acute symmetrical weakness with areflexia in a kidney transplant recipient should prompt urgent neurological evaluation regardless of graft status.

Cerebrospinal fluid analysis and electrophysiological studies remain central to diagnosis, while concurrent investigation for CMV infection and other potential triggers is essential. Most importantly, initiation of IVIG should not be delayed when clinical suspicion for GBS is high, as early treatment is associated with improved neurological outcomes.

CONCLUSION

GBS should be considered in the differential diagnosis of acute progressive weakness after kidney transplantation. This case adds to the limited literature on post-transplant GBS and demonstrates that early recognition, exclusion of reversible triggers, and prompt administration of IVIG can result in complete neurological recovery while preserving renal allograft function.

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