International Journal of Advances in Medicine
Rajkumar H et al. Int J Adv Med. 2026 May;13(3):146-149
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20261092

Case Report

Tacrolimus-associated hemichorea in a post–renal transplant patient: a case report

Haripriya Rajkumar[1]*, Animesh Kar[2], Ocean[2]

[1]Department of Critical Care Medicine, NH-Rabindranath Tagore International Institute of Cardiac Sciences, Mukundapur, Kolkata, West Bengal, India
[2]Department of Neurology, NH-Rabindranath Tagore International Institute of Cardiac Sciences, Mukundapur, Kolkata, West Bengal, India

Received: 02 March 2026
Revised: 13 March 2026
Accepted: 03 April 2026

***Correspondence:**
Dr. Haripriya Rajkumar,
E-mail: hariprabha000@gmail.com

ABSTRACT

Tacrolimus is a calcineurin inhibitor widely used as maintenance immunosuppression after renal transplantation. Although neurotoxicity is a well-recognized complication of tacrolimus therapy, manifesting as tremor, seizures, posterior reversible encephalopathy syndrome, and encephalopathy, the occurrence of choreiform movements—particularly hemichorea—is exceptionally rare. We report a 32-year-old male who developed acute-onset unilateral chorea one month after renal transplantation while receiving tacrolimus and prednisolone. Laboratory evaluation revealed a markedly elevated tacrolimus trough level, while magnetic resonance imaging (MRI) of the brain demonstrated an old right capsulo-ganglionic hemorrhage, providing a structural vulnerability. The patient improved promptly after reduction of tacrolimus dosing and symptomatic therapy. This case underscores the need to consider tacrolimus-induced neurotoxicity in the differential diagnosis of new-onset movement disorders in transplant recipients.

Keywords: Tacrolimus, Drug induced movement disorder, Hemichorea

INTRODUCTION

Tacrolimus, a macrolide lactone derived from *Streptomyces tsukubaensis*, is an essential component of immunosuppressive therapy following solid organ transplantation.[1] As a potent calcineurin inhibitor, tacrolimus prevents T-cell activation and proliferation by blocking the dephosphorylation of nuclear factor of activated T cells (NFAT).[2] However, despite its efficacy in preventing graft rejection, the drug is associated with a variety of systemic adverse effects, including nephrotoxicity, hypertension, glucose intolerance, and neurotoxicity.[3]

Neurological side effects are dose-dependent and occur in 10–32% of patients receiving tacrolimus, although severe manifestations are less common.[4] These include tremor, headache, seizures, visual disturbances, ataxia, agitation, and posterior reversible encephalopathy syndrome (PRES).[4,5] Movement disorders, particularly chorea or hemichorea, are distinctly unusual and sparsely documented in the literature.[6] Drug-induced hemichorea itself is uncommon, with reported cases linked to agents such as antiepileptics, antidepressants, oral contraceptives, and non-benzodiazepine sedatives.[7] In renal transplant patients, movement disorders may be multifactorial, arising from metabolic disturbances, opportunistic infections, or structural brain lesions.[4] However, tacrolimus-induced pure hemichorea, especially presenting early post-transplant, is extremely rare.[8] Understanding this entity is crucial, as early recognition and dose modification can lead to rapid and complete reversal of symptoms.[4]

We present a case of tacrolimus-induced hemichorea in a young renal transplant recipient with supratherapeutic

tacrolimus levels and structurally vulnerable basal ganglia, emphasizing the neurotoxic potential of tacrolimus and the importance of monitoring for neurologic complications.[9]

CASE REPORT

A 32-year-old male, with a known history of hypertension and end-stage renal disease secondary to presumed chronic glomerulonephritis, underwent a live-donor renal transplantation. The perioperative period was uneventful, and graft function stabilized within the expected trajectory. He was discharged on a standard immunosuppressive regimen consisting of tacrolimus 2.5 mg twice daily, prednisolone 20 mg once daily, and mycophenolate mofetil 500mg twice daily. Over the next four weeks, he maintained satisfactory urine output and stable serum creatinine levels, and he adhered strictly to his medication schedule. No episodes of rejection, infection, gastrointestinal intolerance, or neurological symptoms were reported in the immediate post-transplant period. Approximately one month after transplantation, the patient presented to the emergency department with a sudden onset of involuntary movements affecting his left upper limb. According to his family, the movements began insidiously earlier that morning but progressed over several hours to become persistent and disabling. He described the movements as "jerky, dance-like, and uncontrollable," interfering with his ability to hold objects, perform daily activities, or keep the arm still (Figure 1). There was no involvement of the face, tongue, or lower limbs, and no associated weakness, numbness, or paresthesia. He denied headache, dizziness, abnormal behavior, seizures, speech difficulty, or gait imbalance. There was no history of fever, recent infections, missed immunosuppressant doses, or exposure to neurotoxic agents. His sleep–wake cycle and mental status remained normal. The patient had not experienced similar episodes in the past.

Figure 1: Involuntary jerky dance-like movement of left upper arm during unbuttoning in (a) sitting, and (b) standing posture.

On examination, he was alert, oriented, and cooperative. His vital signs were stable, with blood pressure 138/82 mmHg, pulse 88/min, respiratory rate 18/min, afebrile, and oxygen saturation 98% on room air. Cardiovascular and respiratory examinations were unremarkable. Renal graft tenderness or bruit was absent.

Neurological examination revealed prominent choreiform movements involving the proximal and distal muscles of the left upper limb. The movements were irregular, arrhythmic, and semi-purposeful, consistent with chorea. They increased significantly with voluntary actions such as reaching or maintaining posture—suggesting action-induced amplification—but subsided partially at rest and disappeared during sleep, as reported by the family. There was no dystonia, myoclonus, tremor, or ballismus. Cranial nerves were intact. Motor strength, tone, and deep tendon reflexes were normal in all limbs except for the interference caused by involuntary movements in the left arm. Sensory examination, cerebellar function testing, coordination, and gait analysis were normal. No signs of meningeal irritation were present. Given the acute onset of unilateral hyperkinetic movement in a post-transplant patient, an expanded metabolic and hematologic panel was ordered to evaluate for potential triggers including tacrolimus toxicity, metabolic derangements, infection, or cerebrovascular events. Laboratory investigations revealed significantly elevated random blood glucose values and an HbA1c of 10.2%, suggestive of poorly controlled steroid- and tacrolimus-associated hyperglycemia. However, arterial blood gas analysis was normal, and urine ketones were negative, ruling out diabetic ketoacidosis or hyperosmolar states.

Serum tacrolimus trough level was found to be markedly elevated at 22.95 ng/ml (therapeutic range: 5–20 ng/ml), raising immediate suspicion for tacrolimus-induced neurotoxicity. Serum electrolytes, including sodium, potassium, calcium, phosphorus, and magnesium, were within normal limits. Complete blood count and liver function tests were unremarkable. Serum creatinine remained stable at 1.1 mg/dl, indicating preserved graft function.

Given the focal nature of symptoms, a magnetic resonance imaging (MRI) scan of the brain was performed to exclude acute ischemia, hemorrhage, mass lesions, or demyelinating pathology. MRI revealed (Figure 2) an old right capsuloganglionic bleed with associated gliosis and encephalomalacic changes, consistent with a chronic insult. No acute infarct, no diffusion restriction, no new hemorrhage, and no characteristic T1 hyperintensity of the striatum (as seen in diabetic striatopathy) were noted. These findings supported the possibility that the chronic basal ganglia lesion served as a structural substrate predisposing the patient to hyperkinetic manifestations when exposed to tacrolimus toxicity.

Based on the clinical profile, abnormal tacrolimus level, and absence of acute structural pathology, a diagnosis of tacrolimus-induced hemichorea was made, with uncontrolled hyperglycemia considered a contributing but not primary factor.

Management focused on modifying the immunosuppressive regimen, controlling symptoms, and stabilizing metabolic parameters. Tacrolimus dose was reduced from 2.5 mg to 2 mg twice daily, and the prednisolone dose was lowered from 20 mg to 15 mg daily. Mycophenolate mofetil was continued unchanged. Glycemic control was intensified with basal–bolus insulin therapy, later transitioned to oral hypoglycemic agents once glucose levels stabilized. For symptomatic relief of involuntary movements, haloperidol and tetrabenazine were initiated at low doses.

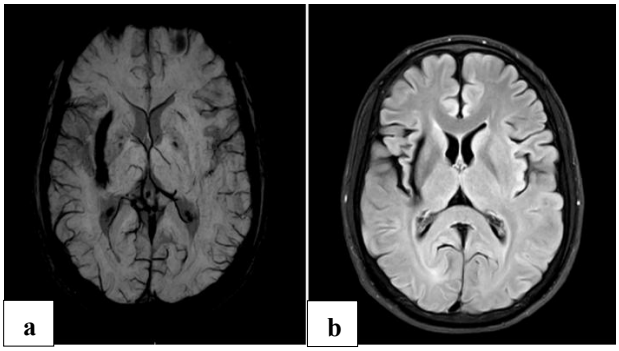

Figure 2: MRI brain showing (a) blooming hypo-intensity in SWI image, and (b) evidence of gliosis and volume loss in FLAIR image in right capsule ganglionic area suggesting a sequelae of old intracerebral hemorrhage.

The patient showed steady improvement beginning on the third hospital day, with a marked decrease in choreiform activity. By the fifth day, involuntary movements were significantly reduced, allowing him to perform routine tasks with minimal difficulty. A repeat serum tacrolimus trough level measured on day five had decreased to 13.48 ng/mL, within an acceptable therapeutic range. No extrapyramidal side effects from haloperidol were noted. The patient was discharged on the seventh hospital day with almost complete resolution of hemichorea. He was instructed on strict glucose monitoring, adherence to the revised immunosuppressive regimen, and early reporting of any neurological symptoms. A follow-up after two weeks confirmed sustained improvement, stable graft function, and adequate glycemic control.

DISCUSSION

Tacrolimus-induced neurotoxicity represents one of the most clinically significant complications encountered in solid-organ transplant recipients, with a spectrum of manifestations ranging from mild tremors to life-threatening encephalopathy. Although tacrolimus neurotoxicity occurs in approximately 10–32% of transplant patients, reports of choreiform movement disorders remain exceedingly uncommon, making this case particularly noteworthy. The calcineurin-inhibiting action of tacrolimus disrupts intracellular calcium signaling pathways that are essential for synaptic stability and neurotransmission within the basal ganglia, a region especially vulnerable to metabolic and excitatory imbalance. Elevated tacrolimus levels have been shown to correlate strongly with the occurrence of neurological dysfunction, even though it is well recognized that neurotoxicity can occasionally occur despite "therapeutic" trough levels, underscoring individual variation in drug sensitivity and blood–brain barrier permeability.[10,11]

In the present case, the patient's tacrolimus trough level was clearly supratherapeutic, a finding frequently associated with more severe neurological manifestations and consistent with earlier reports linking high drug concentrations with movement disorders, including chorea and dystonia.[12] The clinical presentation of abrupt-onset unilateral chorea is most compatible with dysfunction of the contralateral striatum, which plays a central role in regulating motor inhibition. Although hyperglycemia has been well documented as a cause of hemichorea–hemiballismus, particularly in older individuals with nonketotic hyperglycemia, typical radiographic findings include T1 hyperintensity of the basal ganglia and marked metabolic disturbance, features absent in our patient.[16] Moreover, the unusually rapid resolution of symptoms following reduction of tacrolimus strongly supports drug-induced neurotoxicity as the primary mechanism rather than metabolic imbalance.

The MRI finding of a chronic right capsuloganglionic hemorrhage further adds complexity by highlighting the concept of a "vulnerable striatum." Structural lesions within the basal ganglia, even when clinically silent, may lower the threshold for developing dyskinetic manifestations when exposed to neurotoxic or metabolic stressors.[14,15] In such individuals, impaired neuronal reserve and compromised local autoregulation render the striatum especially susceptible to additional insults, including those mediated by calcineurin inhibitors. This interplay between chronic anatomical vulnerability and acute chemical toxicity provides a compelling explanation for the unilateral nature of the patient's symptoms.

Tacrolimus acts not only through calcineurin inhibition but also through mechanisms such as cerebral vasoconstriction, endothelial dysfunction, mitochondrial impairment, and altered GABAergic and glutamatergic neurotransmission, all of which can culminate in basal ganglia hyperexcitability.[10,11] These mechanisms resemble those described in other drug-induced movement disorders, including those associated with sertraline, zolpidem, or gabapentin, where interference with dopaminergic or inhibitory pathways precipitates reversible chorea.[13,18,19] The present case aligns with previously reported drug-induced hemichorea syndromes, particularly regarding reversibility after withdrawal or reduction of the offending agent.

Another consideration in transplant medicine is the high prevalence of concurrent metabolic abnormalities—namely hyperglycemia—related to corticosteroid therapy and tacrolimus itself. Although hyperglycemia was present in this patient, its contribution appears secondary. This interpretation is consistent with reports emphasizing that glycemic derangements may exacerbate or unmask

underlying neuronal susceptibility but rarely account for such an acute presentation in isolation unless radiological markers of diabetic striatopathy are present.[16]

Importantly, early identification of tacrolimus neurotoxicity is essential because timely dose adjustment can yield complete neurological recovery and prevent progression to more severe complications such as posterior reversible encephalopathy syndrome (PRES) or persistent movement disorders. Neurological complications are a major contributor to morbidity following renal transplantation, and clinicians must remain vigilant, particularly during the initial months post-transplant, when tacrolimus dosing is highest and pharmacokinetic instability is common.[17]

This case underscores the critical importance of therapeutic drug monitoring, awareness of atypical neurological presentations, and an appreciation of the interplay between structural brain vulnerability and calcineurin inhibitor toxicity. The dramatic improvement following tacrolimus dose reduction not only reinforces its causative role but also highlights the reversibility of this rare but significant complication.

CONCLUSION

Tacrolimus-induced hemichorea is an uncommon but clinically important complication of calcineurin inhibitor therapy in renal transplant patients. This case demonstrates that even modest elevations in tacrolimus levels can precipitate basal ganglia dysfunction, particularly in individuals with pre-existing structural vulnerabilities. Hyperglycemia acted as a contributory factor but was not the primary cause. Prompt recognition, adjustment of immunosuppression, and metabolic optimization can lead to full recovery. Clinicians should maintain a high index of suspicion for tacrolimus neurotoxicity when transplant recipients present with new-onset movement disorders.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Kino T, Hatanaka H, Hashimoto M, Nishiyama M, Goto T, Okuhara M, et al. FK-506, a novel immunosuppressant isolated from a Streptomyces. I. Fermentation, isolation, and physico-chemical and biological characteristics. J Antibiot (Tokyo). 1987;40(9):1249-55.
2. Liu J, Farmer JD Jr, Lane WS, Friedman J, Weissman I, Schreiber SL. Calcineurin is a common target of cyclophilin–cyclosporin A and FKBP–FK506 complexes. Cell. 1991;66(4):807-15.
3. Staatz CE, Tett SE. Clinical pharmacokinetics and pharmacodynamics of tacrolimus in solid organ transplantation. Clin Pharmacokinet. 2004;43(10):623-53.
4. Wijdicks EFM. Neurotoxicity of immunosuppressive drugs. Liver Transpl. 2001;7(11):937-42.
5. Bartynski WS. Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol. 2008;29(6):1036-42.
6. Cardoso F. Chorea: a journey through history, genetics, and clinical aspects. Lancet Neurol. 2010;9(9):887-98.
7. Alpay K, Ertas M, Orhan EK, Ustay DK, Lienemann A, Baykan B. Chorea associated with various drugs: a review. Neurologist. 2012;18(3):145-50.
8. Kahveci F, Kendirli T, Gurbanov A, Botan E, Koloğlu M, Bektaş Ö, et al. Tacrolimus toxicity-related chorea in an infant after liver transplantation. Ann Clin Case Rep. 2022;37(3):477-9.
9. Jin B, Kim GY, Cheon SM. Tacrolimus-induced neurotoxicity from bipolar disorder to status epilepticus under therapeutic serum levels: a case report. BMC Neurol. 2021;21:448.
10. Wijdicks EFM. Neurotoxicity of calcineurin inhibitors. N Engl J Med. 2003;349(22):2230-9.
11. Bartynski WS, Zeigler ZR, Shadduck RK, Lister J. Tacrolimus-associated cerebral vasculopathy and neurotoxicity. AJNR Am J Neuroradiol. 2001;22(8):1472-7.
12. Lee SY, Lim BC, Kim HS, Chae JH. Tacrolimus-associated movement disorder in a pediatric liver transplant recipient. Pediatr Neurol. 2010;43(4):277-80.
13. Watari T, Tokuda Y. Drug-induced hemichorea. BMJ Case Rep. 2015;2015:bcr2014208872.
14. Piccolo I, Defanti CA, Soliveri P, Volonté MA, Cislaghi G, Girotti F. Cause and course in a series of patients with sporadic chorea. J Neurol. 2003;250(4):429-35.
15. Ghika-Schmid F, Ghika J, Regli F, Bogousslavsky J. Hyperkinetic movement disorders during and after acute stroke: the Lausanne Stroke Registry. J Neurol Sci. 1997;146(2):109-16.
16. Lee EJ, Choi JY, Lee SH. Hemichorea-hemiballism in uncontrolled hyperglycemia: MR imaging findings. Neurology. 2002;58(11):1711-4.
17. Tüzün E, Kantarcı F, Gürsoy A, Baykan B, Akman-Demir G. Neurologic complications of solid organ transplantation. J Neurol Sci. 2013;333(1–2):9-20.
18. Jain KK. Drug-induced movement disorders. In: Jain KK, editor. Drug-Induced Neurological Disorders. Seattle: Hogrefe & Huber. 2001;171-209.
19. Oki M, Kano O, Kuwabara S. Chorea associated with immunosuppressive drugs: clinical features and implications. Tremor Other Hyperkinet Mov. 2017;7:527.

Cite this article as: Rajkumar H, Kar A, Ocean. Tacrolimus-associated hemichorea in a post–renal transplant patient: a case report. Int J Adv Med 2026;13:146-9.