



# New-onset Psychosis in an Immunosuppressed Patient With Kidney Transplantation: An Educational Case Report

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## Abstract

**Rationale:** New-onset psychosis in an immunosuppressed patient post-kidney transplantation (KT) is a diagnostic challenge. A broad differential diagnosis merits consideration; however, an approach to this differential diagnosis remains to be outlined in the literature. Also, when and how to modify the maintenance immunosuppressive regimen remains a significant area of controversy.

**Presenting concerns:** A 23-year-old male, known for X-linked Alport syndrome for which he had undergone KT 1 year prior, presented with a 1-week history of disorganized speech, bizarre behavior, religious delusions, and visual hallucinations.

**Diagnoses:** After ruling out infectious, metabolic, autoimmune, and structural causes, immunosuppressant medications were changed from tacrolimus to cyclosporine. The patient did not improve after this change, and a second opinion consultation with a transplant psychiatrist led to a diagnosis of primary first-episode psychosis, later refined to bipolar disorder type I.

**Interventions:** The patient was started on risperidone, which led to a significant improvement in his symptoms.

**Outcomes:** Twelve months after discharge, his mood and behavior had returned to baseline on aripiprazole, bupropion, and citalopram. However, he developed acute allograft rejection, prompting a change from cyclosporine back to tacrolimus, with stability of his mental state and graft function.

**Teaching points:** This report offers learners an extensive and organized differential diagnosis to the work up of psychosis post kidney transplantation. A complete history, with input from collateral sources, and a systematic approach to the differential diagnosis, are crucial and should not be overshadowed by the risk of immunosuppressant-related neurotoxicity. We underscore the importance of multi-disciplinary management and comprehensive psychosocial assessment and re-assessment to refine the diagnosis. We also report the successful re-introduction of tacrolimus once the diagnosis of a primary psychiatric disorder is confirmed. Finally, we offer a simplified approach that can aid in distinguishing between a primary psychiatric diagnosis versus tacrolimus-associated psychosis.

## Abrégé

**Contexte:** Le diagnostic d'un premier épisode psychotique chez un patient immunosupprimé en raison d'une transplantation rénale (TR) est complexe; un diagnostic différentiel doit alors être envisagé. L'approche à adopter pour établir un diagnostic différentiel reste à définir de façon plus précise dans la littérature. De plus, une controverse subsiste quant au moment et à la manière de procéder pour changer le traitement immunosuppresseur.

**Présentation du cas:** Nous présentons le cas d'un patient de 23 ans atteint du syndrome d'Alport lié à l'X, lequel avait mené à une TR un an auparavant. Depuis une semaine, le sujet manifestait des comportements inhabituels, avait un discours incohérent, était pris de délires religieux et souffrait d'hallucinations visuelles.

**Diagnostic:** Après avoir écarté les causes infectieuses, métaboliques, auto-immunes et structurelles, le traitement immunosuppresseur de tacrolimus a été changé pour la cyclosporine. L'état du patient ne s'étant pas amélioré après le changement de médication, un second avis a été demandé. Une consultation avec un psychiatre spécialisé en transplantation a permis de diagnostiquer un premier épisode psychotique et ultérieurement, un trouble bipolaire de type I.

**Interventions:** L'administration de risperidone a grandement amélioré les symptômes du patient.

**Résultats:** La prise d'aripiprazole, de bupropion et de citalopram avait rétabli l'humeur et le comportement du patient douze mois après son congé de l'hôpital. Le développement d'une réaction aiguë de rejet de l'allogreffe a toutefois entraîné



le remplacement immédiat de la cyclosporine pour le tacrolimus. L'état mental du patient et la fonction du greffon se sont stabilisés.

**Enseignements tirés:** La présentation de ce cas offre aux apprenants un diagnostic différentiel complet et organisé pour l'étude de la psychose chez les patients greffés. Une évaluation approfondie de la condition clinique, avec l'apport de sources parallèles, et une approche systématique du diagnostic différentiel sont essentielles et ne devraient jamais être éclipsées par le risque de neurotoxicité associé aux traitements immunosuppresseurs. Nous insistons sur l'importance d'une prise en charge multidisciplinaire, d'une évaluation psychosociale complète et d'une ré-évaluation pour préciser le diagnostic. Nous rapportons également la réintroduction réussie du tacrolimus une fois le diagnostic du premier épisode psychotique confirmé. Enfin, nous fournissons une approche simplifiée pour aider à distinguer un premier épisode psychotique d'une psychose associée à la prise de tacrolimus.

## Keywords

kidney transplantation, psychosis, immunosuppression, tacrolimus, diagnostic approach, educational case report, new-onset psychosis, psychiatric disorders

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## What was known before

- In an immunosuppressed patient presenting with new-onset psychosis, a broad differential diagnosis merits consideration; however, an approach to this differential diagnosis remains to be outlined in the literature.
- When and how to modify the maintenance immunosuppressive regimen also remains a significant area of controversy and whether some of these agents can be re-introduced has never been reported.

## What this adds

- A complete, reliable history, with input from collateral sources, and a systematic approach to differential diagnosis are crucial in assessing new-onset psychosis after solid organ transplantation.
- Multi-disciplinary management of these patients is indispensable.
- A simplified approach to consider when distinguishing between a primary psychiatric diagnosis versus tacrolimus-associated psychosis is presented
- Tacrolimus can be successfully re-introduced in those who are diagnosed to have primary bipolar disorder.

## Introduction

New-onset psychosis in a patient with a history of solid organ transplantation is a challenging clinical conundrum

with a broad differential diagnosis. Primary and secondary psychiatric disorders and non-psychiatric and transplant-specific causes of psychosis merit consideration. These include infectious, toxic, immunological, metabolic, structural, vascular, and drug-related etiologies. Diagnosis relies on an extensive workup to establish an acute, chronic, and temporal presentation, in which the history and collateral reports play a crucial role; diagnosis often takes months to refine.

Following this, therapeutic considerations, and in particular when and how to modify the maintenance immunosuppressive regimen, remain controversial.<sup>1-3</sup> Calcineurin inhibitors (CNI) are known to cause neurotoxicity, and symptoms ranging from confusion, seizures, posterior reversible encephalopathy syndrome, and acute-onset psychosis have been reported.<sup>1-9</sup> Steroid-based regimens can contribute to neuropsychiatric changes, with symptoms ranging from mild affective, cognitive, or behavioral symptoms to florid psychotic episodes.<sup>10</sup> There is little guidance in the literature on how best to manage these patients.

New-onset psychosis in an immunosuppressed patient is sometimes hastily linked to CNIs, and transplant physicians often struggle to balance the risk of rejection with therapeutic considerations and recommendations from other consulting services. This report describes a case of new-onset psychosis in a young patient after kidney transplantation (KT) and aims to underscore important diagnostic considerations in the breadth and extent of relevant clinical work-up. We propose an approach to the differential diagnosis and

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work-up of this challenging clinical presentation and report the successful re-introduction of tacrolimus once the diagnosis of a primary psychiatric disorder was rendered.

## Presenting Concerns

A 23-year-old male with a history of a KT presented to the emergency room with a 1-week history of bizarre behavior, disorganized speech, and visual hallucinations. There was no history of fever, chills, headache, respiratory symptoms, nausea, vomiting, focal neurological deficits, seizures, urinary symptoms, or known sick contacts.

## Clinical Findings

### *Medical, Family, and Psychosocial History*

The patient's past medical history was notable for hypertension and X-linked Alport syndrome leading to end-stage renal disease. DNA sequencing had confirmed this diagnosis. The patient was known for a transition mutation at exon 7 of the *COL4A5* gene on chromosome X.<sup>11</sup> His grandmother, mother, and 2 aunts were known carriers, and 2 male cousins were affected by the mutation; one required a KT. Family history was otherwise notable for psychiatric disease of unknown type.

Fourteen months before presentation, the patient underwent an uncomplicated living donor KT and the graft was a mismatch at 6/6 HLA antigens. His crossmatch was negative and he received anti-thymocyte globulin and steroid induction, followed by a rapid steroid taper. Notably, there was a mismatch in Epstein-Barr virus serostatus (donor positive, recipient negative). Post-transplant, he did very well, and his creatinine stabilized around 100  $\mu\text{mol/L}$ . His maintenance immunosuppression regimen was tacrolimus with a target trough of 6 to 8 ng/mL and mycophenolic acid 720 mg twice per day. Eventually, 5 mg of prednisone was added due to persistent neutropenia and inability to tolerate full dose of the anti-metabolite. He was known to be adherent to therapy, medical follow-ups and clinic visits and was high functioning from a psychosocial perspective. He lived with his brother, was entirely independent, worked full-time, and had a strong support network. There was no known history of substance abuse or illicit drug use.

### *Physical Examination and Diagnostic Testing*

On presentation, the patient was found to be alert, oriented to person, time, and place, but was agitated, easily distracted, and carried out repetitive movements, such as folding a blanket. He was afebrile and hemodynamically stable. His neck was supple, and there were no signs of a respiratory infection on the head and neck exam. His cardiac, respiratory, and abdominal exams were unremarkable. A neurological exam showed no deficits.

Given the acute course of his symptoms, their lack of specificity, and the high likelihood of an underlying organic etiology in the context of chronic immunosuppression, the patient's presentation was initially deemed atypical for neurodegenerative or psychiatric illness. A preliminary diagnosis of unspecified encephalopathy was made, with possible causes including infectious, inflammatory, autoimmune, metabolic, toxic, and structural etiologies. Immunosuppressant-related neurotoxicity, secondary to his tacrolimus or prednisone, was also considered.

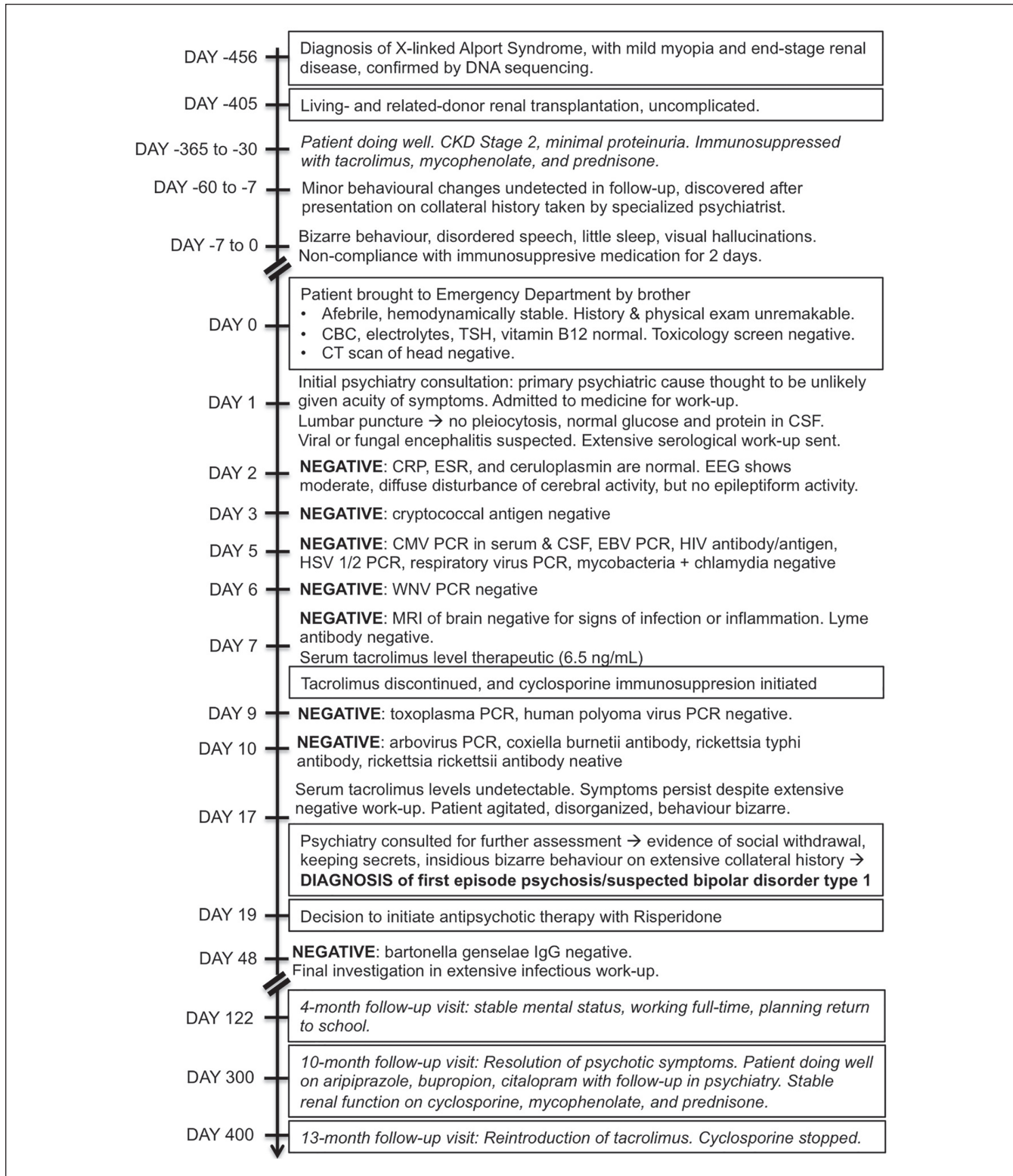
### *Diagnostic Focus and Assessment*

A timeline of events pertaining to this case is shown in Figure 1, and a complete differential diagnosis, with clinical investigations relevant to each etiology, is detailed in Table 1. Infectious workup, toxicology screen, brain imaging, and cerebrospinal fluid analysis were all within normal limits. Immunological and autoimmune causes for the presentation were less likely. His serum tacrolimus trough was therapeutic. With input from a general consult liaison psychiatrist, the patient's psychosis was diagnosed as iatrogenic, secondary to immunosuppressive medication. The transplant team subsequently changed his CNI regimen to cyclosporine with a target 2-hr level of 400 to 600 ng/mL, and mycophenolic acid and prednisone were continued.

This change did not lead to a resolution of his psychotic symptoms; rather, the patient's paranoia was exacerbated, as he became concerned his immunosuppressive therapy was poisonous. A few days later, a second psychiatric consultation with a dedicated solid-organ transplant liaison psychiatrist was obtained, and the patient's case was reassessed. Clinical interview did not reveal any subtle nor gross neurocognitive findings, only typical symptoms of mania: tangential thinking, pressure of speech, psychomotor agitation, and distractibility. This supported by extensive chart review, further collateral history, a family interview, and a detailed review of family psychiatric history led to a change in diagnosis to primary first-episode psychosis. Multiple family members were discovered to have a psychiatric illness. Of note, it emerged that the patient's behavioral changes predated his presentation by a period of several months.

### *Therapeutic Intervention*

A shared decision to start antipsychotic treatment with risperidone 0.5 mg twice daily was made with the patient, his family, and the treating teams. Only in reason of the patient's and his family's paranoia about tacrolimus, his cyclosporine was continued. The patient was initially transferred to a psychiatry ward, where he was found to tolerate the treatment and respond favorably. His symptoms improved considerably, and he was discharged with plans for outpatient follow-up with psychiatry. In the context of a history of a manic episode with psychotic features and prior depressive episodes,



**Figure 1.** Timeline of symptoms, investigations, and therapeutic decisions.

Note. New presentations, diagnoses, therapies, and procedures are enclosed in boxes. Negative investigations are designated by the label "NEGATIVE." CBC = complete blood count; CMV = cytomegalovirus; CRP = C-reactive protein; CSP = cerebrospinal fluid; CT = computed tomography; DNA = deoxyribonucleic acid; EBV = Epstein-Barr virus; EEG = electroencephalogram; ESR = erythrocyte sedimentation rate; HIV = human immunodeficiency virus; HSV = herpes simplex virus; MRI = magnetic resonance imaging; PCR = polymerase chain reaction; TSH = thyroid stimulating hormone; WNV = West Nile virus.

**Table 1.** Differential Diagnosis of New-onset Psychosis in an Immunosuppressed Patient Post-renal Transplantation, With Targeted Clinical Investigations.

Etiology	Targeted clinical investigations	Present case
<i>Infection, viral encephalitis</i>		
Arbovirus	CSF cell count + Arbovirus serology	No pleiocytosis, serology negative
Cytomegalovirus	CSF cell count + CMV PCR	No pleiocytosis, PCR negative
Enterovirus	CSF cell count + Enterovirus PCR	No pleiocytosis, PCR negative
Epstein-Barr virus	CSF cell count + EBV CSF PCR	No pleiocytosis, PCR negative
Hepatitis B and C	CSF cell count + Hepatitis serologies	No pleiocytosis, serology negative
Herpes simplex virus 1/2	CSF cell count + HSV PCR	No pleiocytosis, PCR negative
Human immunodeficiency virus	CSF cell count + HIV serology	No pleiocytosis, PCR negative
Human polyoma virus	CSF cell count + PCR, serology, or urine cytology	No pleiocytosis, PCR negative
West Nile virus	CSF cell count + WNV serology	No pleiocytosis, serology negative
Zika virus	CSF cell count + Zika serology or PCR	No pleiocytosis, PCR negative
<i>Infection, bacterial encephalitis</i>		
Typical bacterial causes	CSF cell count + CSF culture	No pleiocytosis, normal glucose, culture negative
Bartonella henselae	CSF cell count + B. henselae serology	No pleiocytosis, normal glucose, serology negative
Borrelia species (Lyme disease)	CSF cell count + Lyme serology	No pleiocytosis, normal glucose, serology negative
Rickettsia species	CSF cell count + Rickettsia serology	No pleiocytosis, normal glucose, serology negative
Syphilis	CSF cell count + Syphilis serologies	No pleiocytosis, normal glucose, serology negative
<i>Infection, parasitic and fungal encephalitis</i>		
Cryptococcus	CSF cell count + Cryptococcal antigen serology	No pleiocytosis, serology negative
Toxoplasmosis	CSF cell count + Toxoplasma serology	No pleiocytosis, serology negative
<i>Infectious causes, other</i>		
Delirium secondary to respiratory infections	Sputum analysis/culture, nasopharyngeal aspirate PCR, chest X-ray	Sputum analysis/culture negative, PCR negative, chest X-ray negative
Delirium secondary to sepsis	Blood cultures	Blood cultures negative
Delirium secondary to urinary infections	Urinalysis, urine culture	Urinalysis negative, Urine culture negative
<i>Primary psychiatric disease</i>	Psychiatric evaluation + Mental status exam	Consistent with first-episode psychosis after exclusion of organic causes
Bipolar disorder, major depression with psychotic features, schizophrenia, schizophreniform disorder, brief psychotic disorder, schizoaffective disorder		
<i>Neurocognitive disease</i>		
Major or mild neurocognitive disorder (Alzheimer's, Lewy body, Frontotemporal, Vascular), Huntington's disease	Collateral history, Cognitive testing, Head CT/MRI	Head CT and MRI negative
<i>Intoxication, poisoning</i>		
Alcohol, barbiturates, benzodiazepines, cannabinoids, hallucinogens, opioids, stimulants	Urine toxicology screen	Urine toxicology screen negative
Lead poisoning	Blood levels	Not applicable
<i>Structural and vascular</i>		
Hypoxic-ischemic encephalopathy	Head CT or MRI	Head CT and MRI negative
Non-convulsive seizure disorder	EEG	EEG negative for epileptiform activity

Table 1. (continued)

Etiology	Targeted clinical investigations	Present case
Post-transplant lymphoproliferative disorder Space-occupying lesion (tumor, cysts) <i>Medication-induced</i>	EBV PCR, LDH, Head CT/MRI Head CT or MRI	EBV PCR negative, LDH normal, Head CT/MRI negative Head CT and MRI negative
Tacrolimus	Serum tacrolimus levels, clinical assessment, diagnosis of exclusion	Levels in text; eventually undetectable
Cyclosporine	Serum cyclosporine levels, clinical assessment, diagnosis of exclusion	Not applicable
Prednisone	Clinical assessment, diagnosis of exclusion	Not applicable
OKT3 monoclonal antibody-induced	Clinical assessment, diagnosis of exclusion	No known exposure to offending agents
Other drugs (anabolic steroids, analgesics, anticholinergics, antidepressants, antiepileptics, antimalarials, anti-parkinsonian, antivirals, cardiovascular medication, interferons)	History to determine exact medication history; work-up varies depending on drug.	
<i>Immunological and autoimmune</i>		
Anti-NDMA receptor encephalitis	NDMAR antibodies in CSF	NDMAR antibodies negative
Encephalopathy of acute rejection	Head CT	Head CT negative
Paraneoplastic encephalitis	Head CT/MRI, EEG, CSF analysis for known paraneoplastic antibodies	Head imaging and EEG negative. CSF negative for known paraneoplastic antibodies
Systemic lupus erythematosus	Antinuclear antibodies, antiphospholipid antibodies, C3 and C4 complement levels, acute- phase reactants CBC, liver enzymes, serum ceruloplasmin	Negative for antinuclear antibodies, antiphospholipid antibodies. Normal C3 and C4 complement levels, Normal acute-phase reactants levels. liver enzymes normal, serum ceruloplasmin normal
Wilson's disease		
<i>Endocrine/metabolic</i>		
Adrenal disease	If insufficiency suspected: Morning cortisol, Plasma ACTH, Dexamethasone suppression test If Cushing's syndrome suspected: Late-night salivary cortisol, 24-hr urinary-free cortisol excretion, Dexamethasone suppression test	Morning cortisol normal
Electrolyte abnormalities	Serum electrolytes	Serum electrolytes normal
Hepatic encephalopathy	Liver enzymes	Liver enzymes normal
Parathyroid disease	Parathyroid hormone levels	Parathyroid hormone level normal
Porphyrias (especially acute intermittent porphyria)	Porphobilinogen urine test	Not applicable
Thyroid disease	Thyroid hormone levels	Thyroid hormone level normal
Uremic encephalopathy	Blood urea	Blood urea normal
Vitamin B12 deficiency	Serum vitamin B12 level	Serum B12 level normal
Wernicke encephalopathy	Thiamine	Thiamine normal

Note. CBC = complete blood count; CSF = cerebrospinal fluid; CMV = cytomegalovirus; CT = computed tomography; EBV = Epstein-Barr virus; EEG = electroencephalogram; HIV = human immunodeficiency virus; HSV = herpes simplex virus; MRI = magnetic resonance imaging; PCR = polymerase chain reaction; WNV = West Nile virus.

his diagnosis was refined to bipolar disorder type I, which was well controlled on aripiprazole, bupropion, and citalopram.

## Follow-up and Outcomes

About 10 months after his discharge, the patient's mental state remained stable in close follow-up with his treating psychiatrist, transplant team, and social worker. He successfully reintegrated back into the workforce, and partook in a vacation with his family. While his graft function remained stable, a protocol screen revealed a new donor-specific antibody. A for-indication allograft biopsy was done, showing glomerulitis and staining positive for C4d. A diagnosis of antibody-mediated rejection was made. Treatment included rituximab, steroids, intravenous immunoglobulins, and plasmapheresis. After careful discussion with the patient, his family, and his psychiatrist, tacrolimus was re-introduced into his maintenance regimen and cyclosporine was stopped. Six months after this change, his mental state and graft function are stable. His creatinine was at his baseline of approximately a 100  $\mu\text{mol/L}$  and he has no proteinuria.

## Discussion

Mental health disorders are highly prevalent among those with end-stage renal disease and in those who receive transplantation. Depression affects up to 60% of solid-organ transplant recipients, and 20% develop mood and anxiety disorders within the first year after KT.<sup>12</sup> Depression is an independent risk factor for mortality in patients post KT.<sup>13</sup> Data regarding the risks of psychosis or mania in KT recipients are conflicting.<sup>14-16</sup> Few guidelines exist on the evaluation and management of new-onset psychiatric diseases after transplantation. Our case highlights the diagnostic challenges this presentation raises and summarizes management considerations. We propose relevant investigations for each diagnostic possibility and highlight the value of multi-disciplinary care in these patients. Most importantly, we report that tacrolimus was successfully re-introduced once the diagnosis of a primary psychiatric disorder was rendered and the patient was on a stable psychiatric regimen.

Modifying maintenance immunosuppressive regimen in KT recipients presenting with new-onset psychiatric conditions remains an area of controversy in transplant medicine. Neurotoxicity is a well-described side effect of CNIs, yet the underlying mechanism remains poorly understood. Calcineurin inhibitors may alter the intracellular environment of astrocytes, or have cytotoxic effects on brain endothelium.<sup>4-7</sup> Neurotoxicity is more common with tacrolimus than with cyclosporine and is generally dose-dependent, reversible, and mild.<sup>4,5,17</sup> Presentation of CNI related psychosis is usually acute and is reported to occur within days to weeks of initiating it.<sup>3</sup> In some cases, it has been reported to

occur a few months after tacrolimus use.<sup>1,2</sup> Others have reported that it can occur years after KT but was associated with trough tacrolimus blood concentrations that were higher than normal for the patient.<sup>8,18,19</sup>

In patients with suspicion of CNI-induced psychosis, several approaches have been suggested to manage immunosuppression. When associated with an elevated trough level ( $>10 \text{ ng/mL}$ ), some report decreasing serum levels can resolve symptoms of psychosis.<sup>3,18,19</sup> However, psychosis even at lower levels has been reported and discontinuing tacrolimus and switching to cyclosporine, everolimus, or sirolimus was reported to lead to resolution of symptoms.<sup>1,2,8,19</sup> In most cases, after the CNI is decreased or switched to another agent, resolution of psychiatric symptoms occurs within days to weeks, and in some cases, clinicians were able to completely taper off the anti-psychiatric medications.<sup>1-3,8,18,19</sup>

Tacrolimus, however, is the cornerstone of maintenance immunosuppression and one has to balance out the risks of acute rejection. As with our patient's case, others have reported a higher risk of acute rejection with tacrolimus cessation.<sup>1</sup> In one case, a patient developed 2 discrete episodes of psychosis, both of which occurred while on treatment with tacrolimus with a target trough of 3 to 8  $\text{ng/mL}$ , with complete recovery of her mental state between episodes when tacrolimus therapy was withdrawn.<sup>1</sup> The authors tried to re-introduce tacrolimus following an episode of acute rejection but the patient presented with a psychotic episode necessitating the use of everolimus instead. In another case, a young patient presented with 2 different episodes of manic and psychotic symptoms both in the presence of elevated tacrolimus levels.<sup>19</sup> Tacrolimus was stopped and cyclosporine started but the authors do not allude into the details of why this switch was made rather than targeting a lower target trough.

We report the successful re-introduction of tacrolimus after the episode of acute rejection, as the cause of our patient's presentation was not due to the CNI. The broad range of symptoms and time courses with which CNI-related neurotoxicity can present makes it difficult to distinguish from other causes. Primary psychoses, the top differential diagnosis in this case, generally presents more insidiously, and patients exhibit distractibility, usually without neurocognitive symptoms. We present a simple approach that may guide a clinician in distinguishing between these 2 diagnoses (Table 2). We underscore the value of drawing these distinctions in a complete history with collateral information. While a high index of suspicion of CNI-related neurotoxicity is needed, the possibility of this adverse reaction should not limit the work-up undertaken or overshadow other diagnoses on the differential as highlighted in Table 1. An immunosuppressed patient post-KT has an increased risk of infection, cardiovascular disease, malignancy, and psychiatric disease. Consequently, a systematic assessment of changes in their mental status,

**Table 2.** A Simplified Approach to Consider When Distinguishing Between a Primary Psychiatric Disorder and Tacrolimus-Associated Psychosis.

	Primary psychiatric disorder	Tacrolimus-associated psychosis
Onset	Variable and usually an insidious presentation	Usually within days to weeks but years later has been reported with trough levels higher than normal for the patient
Tacrolimus trough	Within normal range	Higher or higher than normal for the patient
Resolution of symptoms after tacrolimus discontinued	No resolution	Within days to weeks
Re-introduction of tacrolimus	No change in clinical condition	Associated with recurrence of symptoms

including a full history and possible involvement of a specialized psychiatrist, is indispensable. Findings from a thorough clinical history and specialized psychiatric assessment will guide clinical reasoning, allowing for important distinctions between primary and secondary causes of psychosis, transplant-specific and other causes, as well as acute, subacute, and chronic presentations, which can aid in narrowing the differential diagnosis.

In conclusions, the present report offers learners an extensive and organized differential diagnosis and a diagnostic approach to patients with a history of solid organ transplantation presenting with new-onset psychosis. Also, this is the first case report describing the successful re-introduction of tacrolimus in an immunosuppressed patient who presented with a primary psychiatric disorder.

### Ethics Approval and Consent to Participate

The patient provided informed consent for the write-up of this case report.

### Consent for Publication

Not applicable

### Availability of Data and Materials

Not applicable

### Declaration of Conflicting Interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Dr Sandal has received an education grant from Amgen to increase living donor kidney transplantation. The other authors have no relevant disclosures.

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