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ACUTE PANCREATITIS ASSOCIATED WITH TACROLIMUS IN A LUNG TRANSPLANT RECIPIENT: A CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

Background: Tacrolimus is a commonly used immunosuppressant with well-defined side effects, including hypertriglyceridemia and hyperglycaemia. However, acute pancreatitis is still not widely recognized as an adverse event related to tacrolimus.

Case presentation: A 60-year-old male was admitted to the intensive care unit with symptoms and signs of acute pancreatitis. He underwent lung transplantation 4 years ago and since then received tacrolimus, mycophenolate mofetil (MMF) and prednisone. His initial laboratory tests revealed elevated serum amylase and lipase, high inflammatory parameters, markedly elevated triglycerides and hyperglycemia with diabetic ketoacidosis. He was treated with continuous insulin infusion, electrolyte correction, crystalloid titration and antimicrobial therapy. During the fasting period, immunosuppression was maintained with parenteral methylprednisolone and tacrolimus. Gradually, oral tacrolimus, MMF and full enteral nutrition were introduced. A control computed tomography scan showed pancreatic body necrosis with a large pseudocyst. The patient was discharged on a strict diet, fenofibrate and short- and long-acting insulin. Immunosuppressive therapy with tacrolimus, MMF and prednisone was continued. He was hospitalized several times within a year due to complications of severe necrotizing pancreatitis.

Conclusion: Acute pancreatitis associated with the use of tacrolimus is rare. Factors contributing to its occurrence are longer exposure period, toxic levels of the drug and concurrent metabolic derangements, predominantly hypertriglyceridemia. In this case report we present a patient after lung transplantation on tacrolimus therapy who developed severe acute necrotizing pancreatitis along with hypertriglyceridemia and diabetes mellitus. We also conducted a literature search and found 13 other cases of acute pancreatitis presumably related to tacrolimus therapy.

KEYWORDS

Tacrolimus, lung transplant, acute pancreatitis, hypertriglyceridemia, diabetes mellitus

LEARNING POINTS

- Tacrolimus related complications including metabolic changes are commonly observed.
- Development of acute pancreatitis is one of the rare complications of tacrolimus therapy, seldomly reported in transplant recipients.
- The definitive therapeutic approach is still to be determined.

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INTRODUCTION

Tacrolimus is a selective calcineurin inhibitor used as an immunosuppressant after hematopoietic and solid organ transplantation. Some of its well-known side effects are the increased risk of infections and malignancy, myelotoxicity, nephrotoxicity, hepatotoxicity, hypertension, hypertriglyceridemia and diabetes mellitus[1]. Of note, a certain number of patients with tacrolimus induced acute pancreatitis has been described.

In this report we present a case of acute necrotizing pancreatitis with severe hypertriglyceridemia and new onset diabetes mellitus with diabetic ketoacidosis in a lung transplant patient treated with tacrolimus.

CASE DESCRIPTION

A 60-year-old man was admitted to the intensive care unit with symptoms of nausea, vomiting, epigastric pain, fever and dyspnoea. Four years prior, he underwent bilateral lung transplant due to end-stage chronic obstructive pulmonary disease and since then was taking immunosuppressive therapy consisting of tacrolimus (1.5 mg BID), mycophenolate mofetil (500 mg BID) and prednisone (5 mg OD). During regular pulmonologist checkup 2 weeks prior to this event, his blood glucose was elevated (17.7 mmol/l) and a computed tomography (CT) scan showed liver steatosis, but the only treatment introduced was dietary modification.

Upon admission, he was conscious, febrile (38°C), tachypnoeic (respiratory rate 28 breaths per minute), tachycardic (120 bpm) and hypertensive (150/85 mmHg). On physical examination patient was adipose (body mass index 40.7 kg/m^2) with diminished breath sounds over the left lung base, pain on palpation in the epigastrium and right paraumbilical area with silent peristalsis. Initial laboratory revealed white blood cell count 25.1 ×10^9/l, hemoglobin 143 g/l, platelet count 271×10^9/l, aspartate aminotransferase 34 IU/l, alanine aminotransferase 34 IU/l, lactate dehydrogenase 610 IU/l, gamma-glutamyl transferase 182 IU/l, alkaline phosphatase 120 IU/l, blood urea nitrogen 7.7 mmol/l, creatinine 109 µmol/l, total bilirubin 10 µmol/l, serum amylase 524 IU/l and lipase 290 IU/l, ionized calcium 1.13 mmol/l, phosphate 0.5 mmol/l, sodium 128 mmol/l, potassium 6.1 mmol/l, C-reactive protein 236.4 mg/l, procalcitonin 8.24 µg/l. Lipid profile showed markedly elevated triglycerides (32.2 mmol/l) with high total cholesterol (10.1 mmol/l). The patient also had hyperglycaemia (blood glucose 28.2 mmol/l) and diabetic ketoacidosis (pH 7.2, base excess -17.8, blood ketones 4.8 mmol/l and positive urinary ketones). His HbA1c was elevated (12.7%), while C-peptide levels were normal. Abdominal ultrasound excluded gallstones and the patient history was negative for alcohol consumption. Initial CT scan showed oedematous pancreatic parenchyma with a hypodense area in the head, extensive peripancreatic oedema and collections of dense liquid with expansion into the mesenteric fatty tissue and along the area of the left anterior renal fascia. There were no signs of abdominal compartment syndrome.

Treatment consisted of continuous insulin infusion and crystalloids per protocol, electrolyte correction and wide spectrum antimicrobial therapy (meropenem) along with other intensive care measures. In consultation with transplant pulmonologist, during the fasting period, immunosuppression was maintained with parenteral methylprednisolone (20 mg) while tacrolimus (1 mg) was administered in a continuous infusion with serum levels regularly monitored. On the third day, enteral mycophenolate mofetil (MMF) (500 mg OD) was introduced, along with water and initial food intake. On the sixth day, full enteral nutrition, oral tacrolimus and pneumocystis pneumonia prophylaxis were started. Tacrolimus levels were within the therapeutic range throughout the whole stay (4.1 – 5.4 mcg/l, ref. value 4 – 15 using liquid chromatography-tandem mass spectrometry).

A repeat CT scan showed pancreatic body necrosis and a 6 cm large pseudocyst along the small curvature of the stomach.

Subsequently, the patient was clinically well, without ultrasound or clinical signs of local complications. Blood glucose levels were adequately regulated and triglyceride levels almost normalized (1.8 mmol/l). The patient was discharged on a strict diabetic, pancreatic, hypolipemic diet, fenofibrate (215 mg) along with short- and long-acting insulin. Immunosuppressive therapy was modified after consultations with his pulmonologist to tacrolimus 2 mg BID, MMF 500 mg BID and prednisone 10 mg OD.

Shortly after, the patient was again hospitalized due to progression of pancreatic necrosis and pseudocyst without signs of infection. Moreover, the patient was subsequently hospitalized several times within a year, due to complications of severe necrotizing pancreatitis including chronic splenic and portal vein thrombosis, pancreatic body necrosis and multiple pseudocysts which eventually needed endoscopic drainage. On the last pulmonologist follow-up, the patient was still receiving tacrolimus (2 mg BID), MMF (500 mg BID) and prednisone (5 mg OD).

DISCUSSION

Acute pancreatitis is a common disease usually caused by alcohol, gallstones and elevated triglycerides. Drug induced acute pancreatitis (DIAP) is a rare condition generally associated with a good prognosis and low mortality[2]. Since diagnosis can be challenging, Saini et al. introduced a revised classification of drugs with causal relation to acute pancreatitis based on the evidence strength[3]. Mallory and Kern established criteria for definitive association of drugs with pancreatitis including: appearance of pancreatitis during treatment with the drug in question; disappearance upon withdrawal of the drug; exclusion of other causes; and relapse upon rechallenge[4]. The Naranjo score can also be used for determining causal probability[5]. Nevertheless, recent review demonstrated that diagnosis of DIAP is not standardized across the literature and causal relationship to culprit drug relies on case reports [6,7]. Moreover,

complexity of treatment for certain conditions, such as transplant patients, makes identification of the causative drug extremely difficult due to polypharmacy and high risk of organ rejection if a suspected drug is discontinued.

A pharmacovigilance study using the JADER database listed 105 reports of tacrolimus induced pancreatitis[8]. Yang et al. conducted a pharmacovigilance study which listed 221 cases of calcineurin inhibitors induced acute pancreatitis showing usual occurrence in males median age of 43 years with mortality rate of 31.67%[9].

We conducted an English-language literature search and found 13 other cases of acute pancreatitis presumably caused by tacrolimus, characteristics of which are shown in *Table 1* (appendix). Our review demonstrated a slight male predominance, with a wider age gap due to 3 paediatric cases. Transplanted organs included kidneys (7 cases), hematopoietic transplantation (3 cases), with heart, liver and lungs 1 case each. This, to our knowledge, makes our case the second described in lung transplant recipients.

While DIAP typically occurs within 4 weeks of therapy initiation[9], our review showed a time range from 8 days to 7 years. This may indicate the cumulative toxic effect of tacrolimus and its metabolites. Mechanisms of tacrolimus induced pancreatitis are still insufficiently investigated, preclinical studies suggesting tacrolimus driven endocrine and exocrine pancreatic dysfunction[10]. In five cases the serum levels of tacrolimus were toxic (>25 ng/ml) and in three were over the upper limit (>15 ng/ml) but not considered toxic yet. This is suggestive of toxic levels of tacrolimus contributing to the development of acute pancreatitis.

Two patients were receiving tacrolimus as monotherapy, while in others tacrolimus was combined with other immunosuppressants. As for other drugs implicated in causing DIAP, our patient received low dose prednisone, mycophenolate mofetil, pantoprazole and co-trimoxazole.

Regarding other possible aetiologies, our patient had marked hypertriglyceridemia, which is a known cause of acute pancreatitis, especially in ranges >11.1 mmol/l. Risk factors for hypertriglyceridemia in our case included obesity, corticosteroid use and unrecognized diabetes mellitus with concurrent ketoacidosis. Tacrolimus alone has adverse metabolic effects and can cause hypertriglyceridemia partially due to decrease in lipoprotein lipase activity and plasma concentration[11] and new onset diabetes mellitus by affecting the pancreatic β-cells, thereby decreasing insulin secretion[12,13].

We believe that tacrolimus itself (possible causality by using the Naranjo score[5]) and its marked metabolic derangements were two main mechanisms intertwined in developing acute pancreatitis in our patient.

The Atlanta classification defines pancreatitis severity as mild, moderate or severe, based on the presence of local and systemic complications and organ failure[14]. In our review, 3 patients clearly had severe acute pancreatitis. There were 4 fatal outcomes, only one being linked to recurrent acute pancreatitis with pancreatic abscess and sepsis, while others occurred later due to other conditions. In some reports it was not possible to determine the type of pancreatitis based on available information, but there were 2 cases of necrotizing pancreatitis with complications of pancreatic pseudocyst and hematoma similar to our patient.

Regarding the initial approach, in 9 cases tacrolimus was acutely discontinued from therapy and mostly switched to parenteral cyclosporine. In others, tacrolimus was continued in lower doses. Our patient continued receiving tacrolimus parenterally with levels monitored and kept at low normal. As studies suggest, in thoracic transplant tacrolimus appears to be superior to cyclosporine regarding incidence of bronchiolitis obliterans syndrome and chronic lung allograft dysfunction[15]. Overall, tacrolimus was part of the maintenance immunosuppressive therapy after the episode of acute pancreatitis in 6 cases (excluding ours). Upon rechallenge, pancreatitis recurred in three patients. As these patients cannot be left without any immunosuppression, it appears reasonable to consider discontinuation of tacrolimus and switch to another immunosuppressant if the causal relationship with acute pancreatitis is strongly suspected acknowledging benefits of each calcineurin inhibitor in terms of organ survival.

CONCLUSION

Although acute pancreatitis is still not widely recognized as an adverse reaction to tacrolimus, clinicians should be alert to this possibility in patients with typical symptoms as the disease can have varying outcomes and potentially fatal consequences. Factors that may contribute to its occurrence are prolonged exposure period, toxic levels of the drug and concurrent hypertriglyceridemia. Further studies are needed to determine the casual relationship and therapeutic approach as existing conclusions are based on individual case reports.

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Table 1. Results of English -language literature search for cases of acute pancreatitis presumably caused by tacrolimus.

Reference	Sex	Age (years)	Time to AP	Transplant	Maintenance immune suppression	Tac level (ng/ml)	HTgc	HGlc	DKA	AP type/ severity	Complications	Immune suppression modification	Tac rechallenge	AP recurrence	Outcome	Time and cause of death
Sastry et al. (2004)	M	9	19 d	BMT	Tac, MMF	5.4	No	No	No	IEP/ NA	NA	Tac ex, CsA in	No	No	Recover	NA
Uemoto et al. (1993)	NA	<18	14 m	Liver	Tac	NA	NA	NA	NA	NA/ NA	NA	Tac reduced	No	NA	NA	NA
Mazumder et al. (2022)	M	17	52 d	Kidney	Tac, MMF, Gcs	29.3	Yes	Yes	Yes	IEP/ moderate	No	Tac ex, CsA in	No	No	Recover	NA
Ogunseinde et al. (2003)	F	52	2 y	Kidney	Tac, AZA	16.1	NA	Yes	No	NP/ severe	Pancreatic pseudocyst	Tac & AZA ex, MMF in	Yes	Yes	Death	5 m later, sepsis, pancreatic abscess
Xu et al. (2019)	M	45	67 d	Kidney	Tac, MMF, GCs	>30	No	Yes	Yes	IEP/ moderate	No	Tac & GCs ex, CsA in	No	No	Recover	NA
Liu et al. (2021)	M	24	10 d	Kidney	Tac, MMF, GCs	>30	No	No	No	IEP/ moderate	No	Tac & MMF ex, CsA in	Yes	Yes	Recover	NA
Ding et al. (2022)	F	38	20 d	Kidney	Tac, MMF, GCs	>30	No	No	No	IEP/ moderate	No	Tac ex, CsA in	No	No	Recover	NA
AlQahtani et al. (2019)	M	41	5 y	Kidney	Tac, MMF, GCs	12.8	NA	NA	NA	NA/ mild	NA	Tac reduced	No	No	Recover	NA
Peng et al. (2025)	M	31	8 d	Kidney	Tac, MMF, GCs	23.8	No	No	No	NP/ severe	Pancreatic hematoma	Tac reduced	Yes	Yes	Recover	NA
Nieto et al. (2000)	F	28	15 d	UCBT	Tac, GCs	17.6	NA	Yes	No	NA/ severe	ARDS, AKI	Tac ex	No	No	Death	85 d later, brain haemorrhage
Tsutsumi et al. (2006)	M	58	46 d	NST	Tac, GCs	NA	NA	Yes	No	NA/ NA	Leukoencephalopathy, cerebral haemorrhage	Tac & GCs ex, CsA in	Yes	No	Death	170 d later, pneumonia
Im et al. (2013)	F	22	7 m	Heart	Tac	>30	NA	Yes	Yes	IEP/ NA	NA	Tac ex for 2 d	Yes	No	Recover	NA
Khan et al. (2006.)	M	65	7 y	Lung	Tac, AZA, GCs	NA	NA	NA	NA	NA/ mild	PTLD	Tac reduced, AZA ex, GCs in	Yes	No	Death	2 m later, unknown
This case	M	60	4 y	Lung	Tac, MMF, GCs	5.2	Yes	Yes	Yes	NP/ moderate	Pancreatic pseudocyst	Tac reduced, MMF ex for 2d	Yes	No	Recover	NA

Abbreviations: AKI, acute kidney injury; AP, acute pancreatitis; ARDS, acute respiratory distress syndrome; AZA, azathioprine; BMT, bone marrow transplantation; CsA, cyclosporine; d, days; DKA, diabetic ketoacidosis; ex, excluded; F, female; GCs, glucocorticoids; HGlc, hyperglycaemia; HTgc, hypertriglyceridemia; in, included; IEP, interstitial oedematous pancreatitis; M, male; m, month; MMF, mycophenolate mofetil; NA, not applicable; NP, necrotizing pancreatitis; NST, nonmyeloablative stem cell transplantation; PTLD, post transplantation lymphoproliferative disease; Tac, tacrolimus; UCBT, umbilical cord blood transplantation.

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