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Tigecycline-Induced Severe Hypertriglyceridemia in a Patient with Chronic Osteomyelitis Due to Carbapenem-Resistant *Klebsiella pneumoniae*: A Case Report and Literature Review

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Abstract

Background: Tigecycline (Tygacil®) is a glycylcycline antibiotic increasingly used off-label at high doses for multidrug-resistant infections, such as those caused by carbapenem-resistant Enterobacterales (CRE). Gastrointestinal adverse effects, hepatotoxicity, coagulopathy, and acute pancreatitis are recognized, whereas severe hypertriglyceridemia as a standalone metabolic adverse effect of prolonged tigecycline therapy has rarely been documented.

Case Presentation: We report a 68-year-old man with treated dyslipidemia and chronic post-traumatic osteomyelitis of the right tibia caused by carbapenem-resistant *Klebsiella pneumoniae* who received high-dose tigecycline (200 mg loading dose, followed by mostly 100 mg IV every 12 hours) for approximately 14 weeks (February 8 to May 16, 2025), followed by perioperative doses during repeated 1-day admissions for wound lavage until full dosing was restarted on June 17. Atorvastatin/ezetimibe was stopped for 5 weeks because of rising liver enzyme levels and was then resumed. In mid-May, near the end of the main tigecycline course, the laboratory reported lipemic serum. Despite fenofibrate, gemfibrozil, and rosuvastatin, serum triglycerides (TG) reached 2,219 mg/dL on June 19 during readmission for flap surgery, with a serum albumin level of 1.8 g/dL. The patient had no abdominal pain, and lipase (71 U/L) and amylase (181 U/L) levels were below three times the upper limit of normal. After lipid-lowering therapy was intensified with icosapent ethyl 4 g/day, gemfibrozil, and atorvastatin, and tigecycline was reduced to 50 mg every 12 hours and then stopped on June 28, TG levels fell by 62% to 843 mg/dL within 9 days. The Naranjo score was 5 (probable). Because no baseline lipid panel was available, TG levels were not quantified before June 19, hypoalbuminemia coexisted, and the dose reduction coincided with intensified lipid-lowering therapy, we regard the association as possible to probable.

Conclusion: This case highlights severe hypertriglyceridemia as an under-recognized metabolic adverse effect that may occur during prolonged tigecycline treatment, especially at high doses in patients with pre-existing dyslipidemia. A baseline lipid panel and periodic TG monitoring are advisable during prolonged therapy, with early lipid-lowering treatment when needed to reduce the risk of acute pancreatitis. Our review of the literature identified 25 published patients with tigecycline-associated pancreatitis in 20 case reports or series, as well as two pooled reviews and several cohort studies. TG values were rarely reported and, when reported, were normal or mildly elevated. We did not identify a published adult case of isolated severe hypertriglyceridemia temporally associated with tigecycline.

Keywords

Tigecycline, Tygacil, Hypertriglyceridemia, Adverse Drug Reaction, Carbapenem-Resistant Enterobacterales, *Klebsiella pneumoniae*, Osteomyelitis, Lipid Monitoring, Naranjo Scale

Introduction

Tigecycline (Tygacil®) is the first glycylcycline antibiotic approved by the U.S. Food and Drug Administration (FDA) and exhibits broad-spectrum activity against Gram-positive, Gram-negative, and anaerobic pathogens [1]. As a minocycline derivative, tigecycline binds to the bacterial 30S ribosomal subunit with approximately 5-fold greater affinity than minocycline, thereby blocking aminoacyl-tRNA entry and inhibiting protein synthesis [1]. Its structural modification, an N,N-dimethylglycylamido side chain, confers resistance to common tetracycline efflux and ribosomal protection mechanisms [1].

The standard dosing schedule includes a 100 mg intravenous loading dose, followed by 50 mg intravenously every 12 hours [1]. For severe infections caused by multidrug-resistant organisms, higher doses, such as a 200 mg loading dose followed by 100 mg every 12 hours, are often used. A systematic review and meta-analysis support this approach, showing better clinical outcomes without a major increase in adverse effects [2], and high doses were well tolerated in critically ill patients [3]. Tigecycline is eliminated mainly through the hepatobiliary and fecal routes (59%); it has a long elimination half-life of approximately 36 hours and a large volume of distribution of 7-9 L/kg [1,4].

Common adverse effects include nausea (29.5%), vomiting (19.7%), and diarrhea (12.7%) [1]. More serious adverse effects include hepatotoxicity with elevated liver enzymes and bilirubin, acute pancreatitis, and coagulopathy [1,5]. The proposed mechanisms of tigecycline-induced pancreatitis include a toxic metabolite, drug-induced hypertriglyceridemia, and high biliary drug concentrations [6]. It has been hypothesized, by extrapolation from the tetracycline class, that impaired protein synthesis in hepatocytes may reduce triglyceride export from the liver, leading to hepatic triglyceride accumulation and potentially to pancreatitis [7,8]; this mechanism has not been demonstrated directly in humans.

Tigecycline-associated pancreatitis has been reported in many case reports and in two pooled reviews, with onset usually within 2 weeks of starting therapy (median 6.5-7 days) [9,10]. In contrast, severe hypertriglyceridemia as the main adverse event of prolonged tigecycline therapy has seldom been reported, mainly in a pediatric cohort [11].

We describe a case of severe hypertriglyceridemia (triglycerides 2,219 mg/dL) without pancreatitis that occurred during long-term high-dose tigecycline treatment for chronic osteomyelitis caused by carbapenem-resistant *K. pneumoniae*, together with a structured review of the literature and a discussion of management.

Case Presentation

Initial Presentation and History:

A 68-year-old man presented on February 5, 2025, with an infected wound 10 days after open reduction and internal fixation of a right tibial fracture. He had received outpatient oral antibiotics (amoxicillin/clavulanate and ciprofloxacin) for 7 days without improvement. On examination, the right tibial

region demonstrated cellulitis with tenderness, erythema, and edema, raising concern for osteomyelitis.

His past medical history included hypertension (amlodipine 5 mg daily), dyslipidemia treated by his private physician (atorvastatin/ezetimibe 20/10 mg daily), benign prostatic hyperplasia (tamsulosin 0.4 mg daily), and hyperuricemia (allopurinol 100 mg daily). No known drug allergies were documented. His past surgical history included bilateral inguinal hernia repair, extracorporeal lithotripsy, and recent right tibial surgery.

Admission laboratory values revealed hemoglobin 11 g/dL, white blood cell count 8,210/µL (neutrophils 65%), platelets 496,000/µL, C-reactive protein (CRP) 79 mg/L, and creatinine 1.0 mg/dL. No lipid panel was available, either from outpatient follow-up before admission or before tigecycline was started. Wound cultures grew carbapenem-resistant *K. pneumoniae* susceptible to tigecycline, colistin, and amikacin (**Table-1**).

Table-1: Simplified Antibiotic Susceptibility Profile of Carbapenem-Resistant Klebsiella pneumoniae from the Wound

Antibiotic	Result
Amoxicillin-clavulanic acid	R
Piperacillin + Tazobactam	R
Cephalosporins	R
Fluoroquinolones	R
Colistin	S
Tigecycline	S
Ceftazidime-Avibactam	R
Imipenem/Cilastatin	I
Meropenem	R
Ertapenem	R
Bactrim	R
Amikacin	S
Aztreonam	R

Antimicrobial Management and Surgical Course:

The patient was initially started on piperacillin/tazobactam 4.5 g intravenously every 6 hours and teicoplanin 600 mg intravenously once daily. After culture results confirmed carbapenem-resistant *Klebsiella pneumoniae*, the regimen was switched on February 8 to high-dose tigecycline: a 200 mg intravenous loading dose followed by 100 mg intravenously every 12 hours.

On February 17, the patient underwent surgical debridement with hardware removal, and intraoperative cultures were obtained. On February 21, because all cultures still grew carbapenem-resistant *K. pneumoniae* after 13 days of tigecycline, amikacin 1 g intravenously every 24 hours for 1 week and imipenem/cilastatin 500 mg were added. Serial CRP values showed a progressive decline from 79 mg/L to 41 mg/L on February 13 and 7.7 mg/L on February 28.

On March 7, the antibiotic regimen was escalated with the addition of imipenem/cilastatin 1 g intravenously every 8 hours and colistin 4.5 million units intravenously every 12 hours after a loading dose because the same bacteria were detected in the cultures. However, colistin was discontinued on March 10 because of acute kidney injury (AKI), with creatinine rising from 0.74 mg/dL on March 5 to 1.5 mg/dL on March 10 and 2.5 mg/dL on March 11. Following nephrology consultation and after mild increases in liver enzyme levels, tigecycline was reduced to 50 mg intravenously every 12 hours and imipenem/cilastatin to 500 mg every 6 hours. Renal function subsequently recovered (creatinine 0.9 mg/dL on June 19).

On April 5, tigecycline was increased again to 100 mg every 12 hours and imipenem/cilastatin to 1 g every 8 hours. The patient underwent four surgical debridements between February 17 and April 10; a peripherally inserted central catheter (PICC) line was inserted on February 24, and vacuum-assisted closure was applied. Serial wound cultures consistently grew carbapenem-resistant *K. pneumoniae* (nine cultures: February 5, 17, and 27; March 4, 8, 13, 19, and 24; and April 1). The patient was discharged on April 15 on outpatient parenteral antimicrobial therapy (OPAT) with imipenem/cilastatin 1 g every 8 hours and tigecycline 100 mg every 12 hours via the PICC line, planned for 6 weeks after the first negative culture on April 4.

Tigecycline was stopped at the end of this course, around May 16. Between April and June, the patient was readmitted approximately six times for 1-day

hospitalizations for wound lavage and debridement, and tigecycline was given perioperatively during each of these stays, for example, 50 mg on May 25. The complete timeline is shown in (**Table-2** and **Fig-2**).

Development of Hypertriglyceridemia:

On March 25, the patient's lipid-lowering medication (atorvastatin/ezetimibe 20/10 mg) was discontinued because of rising liver function test values attributed to tigecycline. It was restarted by the patient's private physician around April 28, before his readmission on May 5; no lipid panel was documented at that time. By mid-May, during one of the readmissions and near the end of the tigecycline course, the laboratory reported that the patient's serum was lipemic, which was the first indication of severe hypertriglyceridemia; the numerical triglyceride (TG) value from this period could not be retrieved from the records. The outpatient physician then intensified lipid-lowering therapy: fenofibrate 200 mg twice daily was started on June 3, and by June 10, the regimen consisted of fenofibrate 200 mg twice daily, gemfibrozil 600 mg twice daily, and rosuvastatin 10 mg daily, in place of atorvastatin/ezetimibe. The combination of the two fibrates was intended to be temporary. Creatine kinase was not measured, and the patient never reported muscle pain or weakness.

On June 17, the patient was readmitted for right tibial repair and flap closure, and tigecycline was resumed at the full dose (100 mg every 12 hours) with imipenem/cilastatin 1 g every 8 hours.

Table-2: Timeline of Tigecycline Dosing, Lipid-Lowering Therapy, and Triglyceride Values

Date (2025)	Tigecycline and key events	Lipid-lowering therapy	TG (mg/dL)
Before Feb 5	Not exposed	Atorvastatin/ezetimibe 20/10 mg daily (private physician)	Not available
08-Feb	200 mg IV loading dose, then 100 mg IV q12h	Atorvastatin/ezetimibe continued	Not measured
Mar 10 to 11	Colistin stopped (AKI); tigecycline reduced to 50 mg q12h	Unchanged	Not measured
25-Mar	Liver enzymes rising	Atorvastatin/ezetimibe stopped	Not measured
05-Apr	Tigecycline back to 100 mg q12h	None	Not measured
15-Apr	Discharged on OPAT: tigecycline 100 mg q12h + imipenem/cilastatin 1 g q8h	None	Not measured
About Apr 28	OPAT continued	Atorvastatin/ezetimibe restarted	Not measured
Mid-May	Readmission; laboratory reports lipemic serum	Atorvastatin/ezetimibe	Not retrievable
About May 16	OPAT completed (6 weeks after first negative culture); continuous tigecycline stopped	Atorvastatin/ezetimibe	Not measured
May 16 to Jun 16	Perioperative tigecycline at each 1-day admission for lavage/debridement (e.g. 50 mg on May 25)	See below	Not measured
03-Jun	Perioperative doses as above	Fenofibrate 200 mg BID started	Not measured
10-Jun	Perioperative doses as above	Fenofibrate 200 mg BID + gemfibrozil 600 mg BID + rosuvastatin 10 mg daily	Not measured
Jun 17 to 19	Readmission for flap closure; tigecycline resumed at 100 mg q12h; albumin 1.8 g/dL; no pancreatitis	Unchanged	2,219 (Jun 19)
22-Jun	Tigecycline reduced to 50 mg q12h	Fenofibrate and rosuvastatin stopped; gemfibrozil continued; atorvastatin 40 mg + icosapent ethyl 2 g BID started	Not measured
23-Jun			1,491
25-Jun	Lipase 52 U/L		1,183
28-Jun	Discharge; tigecycline stopped	Atorvastatin 20 mg, fenofibrate 200 mg, icosapent ethyl 1 g daily; gemfibrozil stopped	843
17-Jul	Readmission for flap procedure	Unchanged	Not measured

AKI, acute kidney injury; BID, twice daily; OPAT, outpatient parenteral antimicrobial therapy; q8h/q12h, every 8/12 hours; TG, triglycerides.

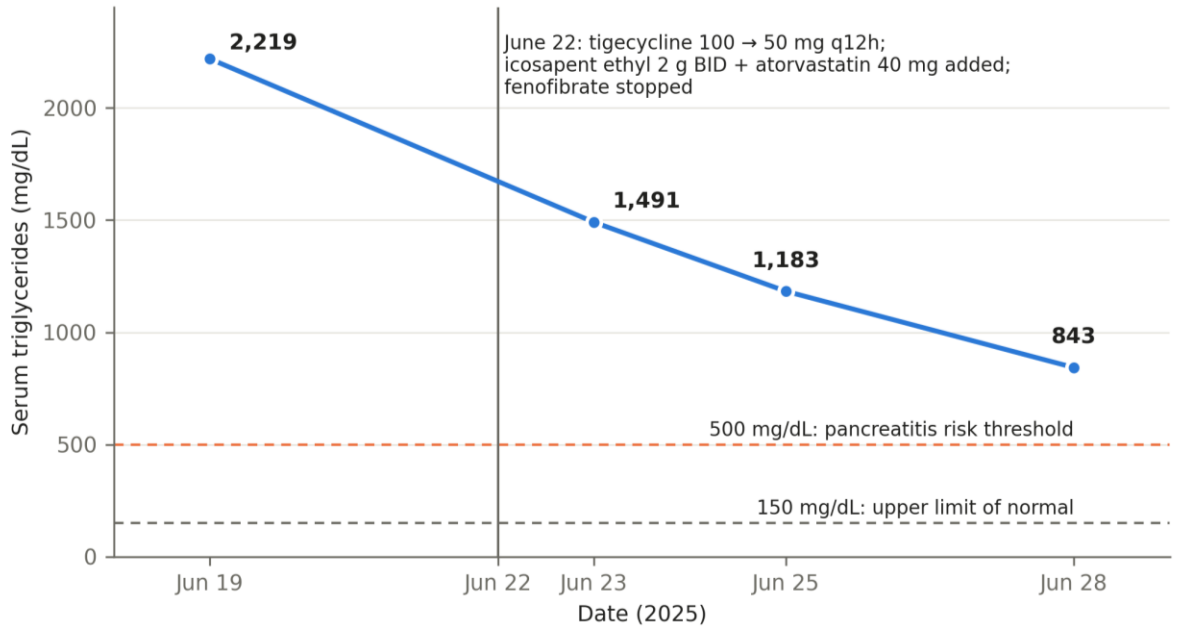


Fig-1:

Serum triglyceride levels from June 19 to June 28, 2025. After the main course ended around May 16, tigecycline was given perioperatively during 1-day admissions and restarted at full dose on June 17. On June 22 it was reduced from 100 mg to 50 mg every 12 hours, icosapent ethyl and atorvastatin were added and fenofibrate was stopped; the decline therefore reflects both interventions. Tigecycline was stopped on June 28. Dashed lines mark 500 mg/dL (pancreatitis risk threshold) and 150 mg/dL (upper limit of normal).

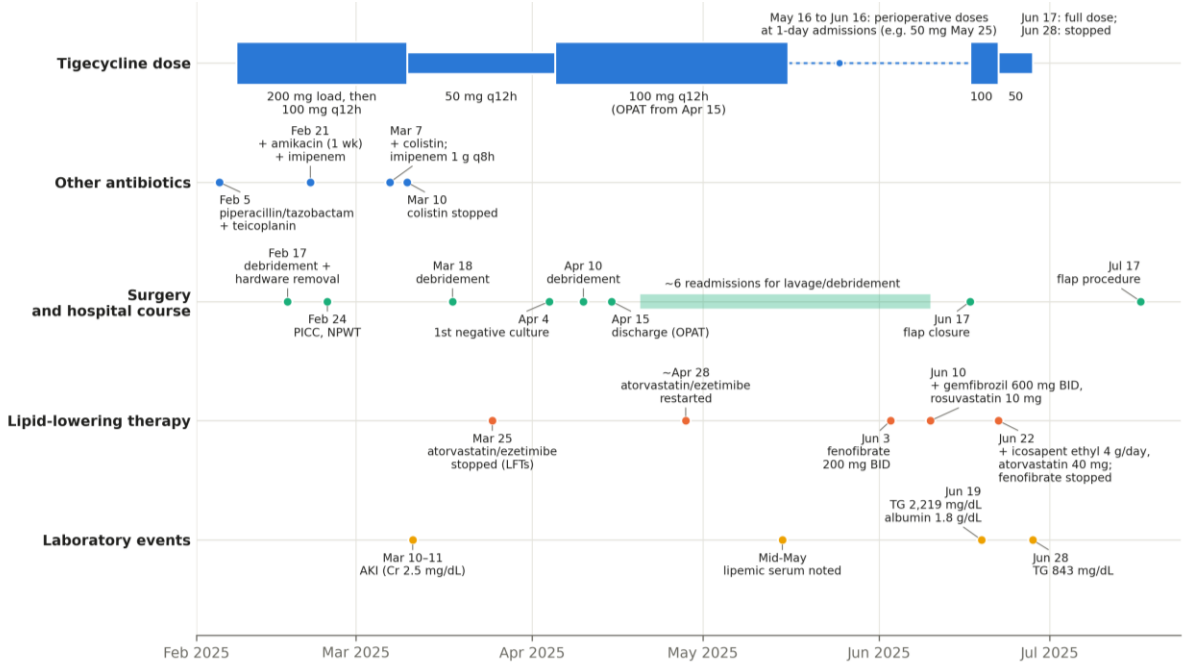


Fig-2:

Clinical timeline from February to July 2025: tigecycline dose (bar height proportional to dose; between May 16 and June 17 tigecycline was given perioperatively during 1-day admissions, e.g. 50 mg on May 25), other antibiotics, surgery and hospital course, lipid-lowering therapy and key laboratory events. AKI, acute kidney injury; Cr, creatinine; LFTs, liver function tests; NPWT, negative-pressure wound therapy; OPAT, outpatient parenteral antimicrobial therapy; PICC, peripherally inserted central catheter; TG, triglycerides.

On June 19, laboratory evaluation revealed severe hypertriglyceridemia with TG of 2,219 mg/dL and serum albumin of 1.8 g/dL. Additional results included lipase 71 U/L (reference range 13-60 U/L), amylase 181 U/L (28-100 U/L), thyroid-stimulating hormone (TSH) 0.86 mIU/L, creatinine 0.9 mg/dL, alanine aminotransferase (ALT) 43 U/L, aspartate aminotransferase (AST) 79 U/L, alkaline phosphatase 405 U/L, gamma-glutamyl transferase (GGT) 168 U/L, bilirubin within normal limits, corrected calcium 10.16 mg/dL, lactate dehydrogenase (LDH) 338 U/L, and white blood cell count 8,250/µL. The urinary albumin-to-creatinine ratio was normal, excluding nephrotic-range proteinuria. Importantly, the patient had no abdominal pain, nausea, or vomiting and no clinical evidence of acute pancreatitis; lipase was 52 U/L on June 25. He was eating orally and had not received parenteral nutrition, intravenous lipid emulsions, or propofol at any time during his course.

Following consultation, endocrinology advised optimizing lipid-lowering treatment and administering one vial of albumin intravenously three times daily. As a result, on June 22, fenofibrate and rosuvastatin were stopped, gemfibrozil 600 mg twice daily was continued, and atorvastatin 40 mg daily and icosapent ethyl 2 g twice daily (4 g/day) were started; tigecycline was reduced at the same time to 50 mg every 12 hours and was stopped at discharge on June 28. Dietary counseling, including a low-fat diet with restriction of simple carbohydrates, was provided. Insulin infusion and plasmapheresis were not used because the patient was asymptomatic, had no pancreatitis, and was not hyperglycemic.

Response to Intervention:

Triglyceride levels decreased from 2,219 mg/dL on June 19 to 1,491 mg/dL on June 23, 1,183 mg/dL on June 25, and 843 mg/dL on June 28 after tigecycline dose reduction and adjustment of lipid-lowering treatment, representing a 62% reduction from the peak within 9 days (**Fig-1**).

The patient was discharged on June 28 on atorvastatin 20 mg daily, fenofibrate 200 mg daily, rivaroxaban 10 mg daily, icosapent ethyl 1 g daily, and tamsulosin 0.4 mg daily. Gemfibrozil was discontinued to avoid its combination with a statin, and tigecycline was stopped at discharge. He was readmitted on July 17 for a further flap procedure, at which time he remained on the optimized lipid-lowering regimen without clinical pancreatitis.

Discussion

Tigecycline and Lipid Metabolism:

In this case, severe hypertriglyceridemia (TG 2,219 mg/dL) may represent an adverse effect of long-term, high-dose tigecycline treatment. Tigecycline, like other tetracycline-class drugs, binds to the 30S ribosomal subunit and inhibits bacterial protein synthesis [1]. It has been hypothesized that a similar effect on protein synthesis in hepatocytes could impair the assembly and release of triglyceride-rich lipoproteins, leading to hepatic triglyceride accumulation and hypertriglyceridemia [6,7].

Furthermore, tigecycline achieves very high biliary concentrations (median bile concentration 75.2 mg/L versus 0.112 mg/L in serum, with bile-to-serum AUC ratios of 368-537), which may contribute to hepatobiliary injury and impaired lipid metabolism [6]. These mechanisms remain hypothetical and have not been demonstrated directly in humans. In a pediatric cohort treated with a tigecycline-containing regimen for *Mycobacterium abscessus* otomastoiditis, 70% of children developed hypertriglyceridemia, and most adverse events were attributed to tigecycline [11].

Confounding and Contributing Factors:

Several factors other than tigecycline may have contributed to the severity of hypertriglyceridemia in this patient and limit causal attribution.

Pre-existing Dyslipidemia and Statin Interruption:

The patient was treated for dyslipidemia before admission, but no lipid values were available, so his baseline TG levels are unknown. Atorvastatin/ezetimibe was interrupted for approximately 5 weeks (March 25 to around April 28). Statins have only a modest TG-lowering effect [13], and severe hypertriglyceridemia was recognized after the statin had been resumed and progressed despite additional fibrate therapy, which argues against statin interruption as the main cause, although it may have contributed.

Hypoalbuminemia (1.8 g/dL):

Nephrotic-range proteinuria was excluded by a normal urinary albumin-to-creatinine ratio. The likely causes were chronic inflammation from persistent infection and repeated surgery, reduced intake during prolonged hospitalization, and possible impairment of hepatic synthesis in the setting of tigecycline-associated liver enzyme elevation. Hypoalbuminemia is associated with altered lipoprotein metabolism and may be both a marker of illness severity and an independent contributor to dyslipidemia. A direct link between hypoalbuminemia and tigecycline toxicity has not been demonstrated.

Carbapenem Exposure:

The patient received imipenem/cilastatin, not meropenem, from February 21 onward. We found no reports associating carbapenems with hypertriglyceridemia, so a contribution was considered unlikely.

Acute Kidney Injury:

The episode of March 10-11 resolved more than 3 months before the TG peak.

Nutrition and Sedation:

The patient never received parenteral nutrition, intravenous lipid emulsions, or propofol, which are common causes of drug-related hypertriglyceridemia in hospitalized patients. Thyroid function was normal, renal function had recovered, and there was no hyperglycemia.

Duration and Dose of Tigecycline:

The main course lasted approximately 14 weeks, mostly at twice the standard maintenance dose, far longer than the courses in published pancreatitis reports, in which onset usually occurred within 2 weeks (**Table-4**). Because tetracycline-class hepatotoxicity is considered dose-related, the high-dose regimen may have increased metabolic toxicity [1]. After the main course ended around May 16, exposure continued through perioperative doses during repeated 1-day admissions, and full dosing was resumed on June 17, so the patient was never free of the drug for long before the TG peak on June 19.

Clinical Significance: Absence of Pancreatitis:

Notably, the patient had no symptoms and no clinical pancreatitis despite a TG level of 2,219 mg/dL, far above the 500-1,000 mg/dL range at which the risk of pancreatitis increases [14,15]. Lipase was 71 U/L (1.2 times the upper limit of normal) on June 19 and 52 U/L on June 25, and amylase was 181 U/L (1.8 times the upper limit of normal); these values were below three times the upper limit of normal and, in the absence of abdominal pain, did not meet the diagnostic criteria for acute pancreatitis. Mild hyperamylasemia may also have non-pancreatic causes.

This suggests that long-term tigecycline therapy may cause severe hypertriglyceridemia without obvious clinical symptoms, which may be detected only incidentally, as in this case through a laboratory report of lipemic serum, or only after a serious complication occurs. This emphasizes the importance of regular lipid monitoring during prolonged tigecycline therapy.

Causality Assessment and Limitations:

The causal link between tigecycline and severe hypertriglyceridemia was evaluated using the Naranjo Adverse Drug Reaction (ADR) Probability Scale.

The Naranjo score was 5, corresponding to a probable adverse drug reaction (**Table-3**) [12]. The score depends on how item 1 is scored, and several limitations mean that the association should be regarded as possible to probable.

First, no baseline lipid panel was available, so the contribution of tigecycline relative to the patient's underlying dyslipidemia cannot be quantified. Second, TG levels were not measured between tigecycline initiation and June 19; the mid-May value could not be retrieved, so the time of onset is uncertain. Third, the dose reduction on June 22 coincided with the addition of icosapent ethyl and atorvastatin, a change in fibrate therapy, and albumin infusion, so the contributions of these interventions to the 62% TG decrease cannot be separated. Fourth, statin interruption, hypoalbuminemia, and chronic inflammation are plausible additive causes. No rechallenge was performed, and tigecycline concentrations were not measured.

Table-3: Naranjo Adverse Drug Reaction Probability Scale Applied to Tigecycline and Hypertriglyceridemia [12]

Question	Answer	Score	Justification
1. Are there previous conclusive reports on this reaction?	Yes	1	Hypertriglyceridemia in 7 of 10 children receiving tigecycline-containing regimens [11]; hypertriglyceridemia proposed as a mechanism of tetracycline-class pancreatitis [6,7]. Evidence is limited, so this item could also be scored 0.
2. Did the adverse event appear after the suspected drug was given?	Yes	2	Tigecycline started Feb 8; lipemic serum in mid-May; TG 2,219 mg/dL on Jun 19.
3. Did the reaction improve when the drug was discontinued or a specific antagonist was given?	Yes	1	TG fell 62% in 9 days after the dose reduction on Jun 22, and tigecycline was stopped on Jun 28 (confounded by simultaneous intensification of lipid-lowering therapy).
4. Did the reaction reappear when the drug was readministered?	Not done	0	No rechallenge.
5. Are there alternative causes that could on their own have caused the reaction?	Yes	−1	Pre-existing dyslipidemia, temporary statin withdrawal, hypoalbuminemia, chronic inflammation.
6. Did the reaction reappear when a placebo was given?	Not done	0	Not applicable.
7. Was the drug detected in blood in concentrations known to be toxic?	Not done	0	Tigecycline levels not measured.
8. Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?	Yes	1	Severe hypertriglyceridemia during 100 mg q12h; improvement after reduction to 50 mg q12h (confounded as above).
9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	No	0	No prior tetracycline-class exposure documented.
10. Was the adverse event confirmed by any objective evidence?	Yes	1	Lipemic serum and serial laboratory TG measurements.
Total		**5**	**Probable** (≥9 definite; 5 to 8 probable; 1 to 4 possible; ≤0 doubtful). Score 4 (possible) if item 1 is scored 0.

Table-4: Published Reports of Tigecycline-Associated Pancreatitis and Lipid Abnormalities (Chronological)

Author, year [ref]	Patient(s)	Indication	Regimen / time to onset	Lipid data	Event and outcome
Gilson, 2008 [23]	Adult man	Enterobacter cloacae osteitis	NR	NR	Severe acute pancreatitis
Lipshitz, 2009 [24]	Middle-aged woman	NR	NR	NR	Acute pancreatitis (letter)
Hung, 2009 [25]	69 y, F	Complicated skin and soft-tissue infection	Onset day 7	NR	Acute pancreatitis; Naranjo probable
Marshall, 2009 [7]	55 y, F	NR	NR	NR	Acute pancreatitis
Prot-Labarthe, 2010 [26]	Child, M	E. cloacae sepsis	NR	NR	Acute necrotizing pancreatitis
Mascarello, 2012 [27]	Adult	NR	NR	NR	Acute necrotizing pancreatitis
Marot, 2012 [6]	2 adults, M	Soft-tissue infection	Symptoms by day 5; enzymes day 5 to 6	NR	Mild pancreatitis; resolved within 1 week of withdrawal
Okon, 2013 [41]	62 FAERS reports	Various	NR	NR	Disproportionate reporting signal; higher in women
McGovern, 2014 [40]	9 of 3,788 trial patients (0.24%)	Phase 3 and 4 trials	Median duration 8 days	NR	Incidence similar to comparators (0.27%)
Hemphill, 2016 [28]	22 y, M, cystic fibrosis	M. chelonae bronchitis	Two courses	NR	Pancreatitis on both exposures (positive rechallenge); resolved

Bernas Albeniz, 2017 [29]	68 y, F	Sepsis of unknown origin	Onset within 5 days	NR	Acute pancreatitis; Naranjo probable
Akhter, 2018 [30]	Middle-aged man	Skin infection	NR	NR	Acute pancreatitis (letter)
Lin, 2018 [31]	48 y, F, KT	MDR A. baumannii	100 mg, then 50 mg q12h; onset after 15 days	No hyperlipidemia	Balthazar grade D; amylase normal within 1 week of withdrawal
Fang, 2020 [9]	Review: 22 patients, 19 studies	Various	Median onset 6.5 days (2 to 28)	NR	90% mild; enzymes normal after median 5 days
Wang, 2021 [32]	87 y, F	Urinary tract infection	100 mg, then 50 mg q12h; onset after 6 days	NR	Enzymes normal 5 days after withdrawal; later died of septic shock
Yazirli, 2021 [33]	35 y, M, KT	Perianal cellulitis; abdominal infection	Day 10; recurrence day 3 of re-exposure	NR	Two episodes (positive rechallenge); resolved after withdrawal
Chang, 2022 [34]	Child, cystic fibrosis	Pulmonary infection	Onset day 5	NR	Resolved 2 weeks after withdrawal
Fredj, 2023 [35]	5 of 303 burn ICU patients (1.65%); mean age 28 y	Sepsis	200 mg, then 100 mg q12h; mean onset 5.4 days	Hypertriglyceridemia excluded	Symptoms resolved about 4 days and enzymes 9 days (2 to 20) after withdrawal
Maringanti, 2023 [36]	27 y, F	NR	Started about 2 weeks before	Lipid panel normal	Resolved after withdrawal
Li, 2023 [37]	75 y, M	A. baumannii (blood, sputum)	Detected day 12; with furosemide	NR	Pancreatitis and hypofibrinogenemia; improved after withdrawal
Pan, 2023 [10]	Review: 34 patients, 29 articles; 46.9% transplant	Various	89.3% standard dose; median onset 7 days; 91.2% within 14 days	NR	Enzymes normal after median 5 days; 4 deaths
Tan, 2023 [42]	21 of 3,823 adults (0.56%); juveniles 1.15%	Various	Mean onset 7.2 days	NR	All mild; renal insufficiency an independent risk factor
Ren, 2023 [38]	62 y, M, chronic renal insufficiency	Pneumonia	100 mg q12h for 5 days, then 50 mg; onset day 5	NR (fatty liver on imaging)	Diagnosed by bedside ultrasound; amylase normal 3 days after withdrawal
Arafat, 2024 [8]	39 y, F, recurrent malignancy	CRE bacteremia and urinary infection	200 mg, then 100 mg q12h	TG 192 mg/dL	Improved after withdrawal
Wee, 2024 [39]	78 y, M, diabetes, CKD, known hyperlipidemia	Infected toe gangrene	50 mg q12h; onset day 6	TG NR	Amylase 10-fold; normal 4 days after withdrawal; Naranjo 7
Feng, 2025 [43]	9 of 80 KT recipients (11.25%)	Various	Mean onset 7 days	NR	4 deaths; AKI and high tacrolimus levels risk factors
Yang, 2025 [44]	5 of 10 post-KT pancreatitis cases	Various	NR	NR	Tigecycline the only drug cause identified; good outcomes
Lu, 2026 [45]	52 of 6,942 patients (0.75%)	Various	Median duration 7.5 days; median onset 4 days	NR	Risk factors: transplantation, laboratory markers, esomeprazole
Leow, 2026 [11]	10 children (mean 8 y)	M. abscessus otomastoiditis	Prolonged multidrug regimen	Hypertriglyceridemia 70%; hypoalbuminemia 90%	Mostly attributed to tigecycline; all cured
Present case	68 y, M	CRE K. pneumoniae tibial osteomyelitis	200 mg, then 100 mg q12h for about 14 weeks, then perioperative doses; lipemic serum at about 3 months; full dose again Jun 17 to 28	TG 2,219 mg/dL; albumin 1.8 g/dL	No pancreatitis; TG fell 62% in 9 days after dose reduction and lipid therapy

Management of Severe Drug-Induced Hypertriglyceridemia:

The management strategy in this case followed current evidence-based recommendations. For severe hypertriglyceridemia (≥500 mg/dL), the main objective is to lower TG levels to prevent pancreatitis [15,16].

For TG levels above 1,000 mg/dL, as in our case, guidelines recommend a fibrate as first-line drug therapy, together with a very-low-fat diet, restriction of simple carbohydrates and alcohol, and management of secondary causes, including drugs that raise TG levels [13,15]. Fibrates lower TG levels by 25-50% by increasing lipoprotein lipase activity through PPAR-α activation [14].

Prescription omega-3 fatty acids at 4 g/day (EPA plus DHA or EPA alone) lower TG levels by 30% or more by reducing VLDL synthesis, increasing β-oxidation, and decreasing hepatic lipogenesis, and are recommended by the American Heart Association as a safe and effective TG-lowering therapy [16,17]. Icosapent ethyl, a highly purified eicosapentaenoic acid ethyl ester, does not raise LDL cholesterol, unlike EPA plus DHA combinations [18]. In the REDUCE-IT trial, icosapent ethyl 2 g twice daily reduced the primary composite cardiovascular endpoint (cardiovascular death, nonfatal myocardial infarction, nonfatal stroke, coronary revascularization, or unstable angina) by 25% in statin-treated patients with TG levels of 135-499 mg/dL and established cardiovascular disease or diabetes [19]; its use here was aimed at TG lowering rather than cardiovascular prevention.

Insulin infusion and plasmapheresis are generally reserved for hypertriglyceridemia-induced pancreatitis or uncontrolled hyperglycemia and were not indicated in our asymptomatic, normoglycemic patient [15].

The combination of fenofibrate with gemfibrozil used transiently before readmission is not recommended, and gemfibrozil should be avoided with statins because it increases the risk of myopathy and rhabdomyolysis; if a fibrate is combined with a statin, fenofibrate is preferred [15]. This regimen was prescribed in the outpatient setting as a temporary measure. Creatine kinase was not measured, which is a limitation, but the patient had no muscle symptoms. During hospitalization, the regimen was rationalized, and at discharge, gemfibrozil was stopped and fenofibrate was combined with atorvastatin, in line with guideline recommendations.

In our patient, TG levels fell by 62% within 9 days after the combination of tigecycline dose reduction (from 100 mg to 50 mg twice daily) and intensified lipid-lowering therapy (icosapent ethyl 2 g twice daily, atorvastatin 40 mg, and gemfibrozil 600 mg twice daily). This response is consistent with a drug-related contribution, but because both interventions were started simultaneously, their relative effects cannot be separated, and the improvement cannot be attributed to tigecycline reduction alone.

Antibiotic Considerations in CRE Osteomyelitis:

This patient's osteomyelitis caused by carbapenem-resistant *K. pneumoniae* required prolonged antimicrobial therapy, creating a trade-off between metabolic toxicity and effective infection control. Tigecycline was chosen because the isolate was resistant to ceftazidime/avibactam and most beta-lactams, susceptible only to colistin, amikacin, and tigecycline, and intermediate to imipenem (**Table-1**), and because colistin had to be discontinued because of acute kidney injury.

In an experimental rabbit model of osteomyelitis caused by carbapenemase-producing *K. pneumoniae*, colistin alone was ineffective, whereas colistin combined with meropenem, with or without gentamicin, or with fosfomycin was bactericidal; the combination of colistin with tigecycline was antagonistic in vitro and reduced the efficacy of colistin in vivo [20]. These experimental findings cannot be directly translated into clinical treatment recommendations.

Despite these concerns, tigecycline remains an option in bone and joint infections caused by extensively drug-resistant Gram-negative bacteria when susceptibility is documented. Clinical data are limited to small case series, and a narrative review concluded that tigecycline appears to be most effective at the highest possible dose and in combination with other active agents [21].

High-dose regimens (200 mg loading dose, followed by 100 mg every 12 hours) have been associated with improved clinical outcomes in severe multidrug-resistant infections [2,3], which supported their use in this patient despite the risk of metabolic toxicity.

Combination regimens based on a polymyxin with a carbapenem, aminoglycoside, or fosfomycin are supported mainly by experimental and observational data [20,21]. In a retrospective comparison of tigecycline and colistin monotherapy for carbapenem-resistant osteomyelitis cited in the same review, tigecycline had a better safety profile and similar 12-month outcomes [21].

Newer beta-lactam/beta-lactamase inhibitor combinations, such as ceftazidime/avibactam, have been used successfully in case reports and small series of bone and joint infections, usually combined with colistin, fosfomycin, or an aminoglycoside; however, the isolate in our patient was resistant to ceftazidime/avibactam [21].

The optimal duration of therapy for CRE osteomyelitis remains uncertain. Radical debridement and removal of hardware, followed by prolonged combination antimicrobial therapy, usually for at least 6 weeks after surgical source control, are generally recommended [21,22]. Most available data come from experimental models, observational studies, and case reports rather than randomized trials, and the optimal combination, dosing, and duration remain to be defined in larger multicenter studies [21].

Literature Review

Search Strategy:

PubMed was searched from inception to September 29, 2026, using "tigecycline" combined with "hypertriglyceridemia", "hypertriglyceridaemia", "triglyceride(s)", "hyperlipidemia", "dyslipidemia", "lipid metabolism", "hyperamylasemia", and "pancreatitis". The reference lists of retrieved articles and two published reviews [9,10] were screened. We included case reports, case series, cohort studies, pooled clinical-trial analyses, and pharmacovigilance studies describing tigecycline-associated pancreatitis or lipid abnormalities in humans. We excluded reports in which tigecycline was used to treat infected pancreatitis rather than being the suspected cause, experimental studies, and articles without an English abstract. Data were extracted from full texts when available in PubMed Central and otherwise from abstracts.

Results:

Only one study explicitly documented hypertriglyceridemia during tigecycline-containing therapy: among 10 children treated for *M. abscessus* otomastoiditis, 7 (70%) developed hypertriglyceridemia and 9 (90%) developed hypoalbuminemia, and most adverse events were attributed to tigecycline [11]. We did not identify a published adult case report of isolated severe hypertriglyceridemia temporally associated with tigecycline; this statement reflects the limitations of a PubMed-based search.

Tigecycline-associated acute pancreatitis is much more extensively reported. A 2020 review identified 22 patients from 19 studies, with a median onset of 6.5 days [9], and a 2023 review identified 34 patients from 29 articles published before February 2023, 46.9% of whom were solid-organ transplant recipients; the median time to onset was 7 days, and 91.2% of cases occurred within 14 days [10]. The number of reported patients varies between reviews because of differences in search periods, databases, inclusion criteria, and language, and because of possible duplicate reporting.

Table-4 summarizes 29 publications identified by our search: 20 case reports or small series describing 25 patients [6-8,23-39], two pooled reviews, one pooled analysis of phase 3 and 4 trials, one pharmacovigilance study, and five cohort studies [40-45]. The reported incidence of pancreatitis ranged from 0.24% in clinical trials, similar to comparators [40], and 0.56-0.75% in large hospital cohorts [42,45] to 1.65% in burn intensive care patients [35] and 11.25% in kidney transplant recipients [43]. Renal insufficiency, transplantation, and tacrolimus exposure were identified as risk factors [42,43,45]. Positive rechallenge was reported in two patients [28,33]. Triglycerides were rarely reported; when they were, they were normal or only slightly elevated, with a maximum of 192 mg/dL, and hypertriglyceridemia was excluded as the cause of pancreatitis [8,31,35,36].

Our case differs from published reports in three respects: a much longer duration of exposure of approximately 14 weeks, a severe and numerically documented lipid abnormality, and the absence of pancreatitis. This toxicity may be overlooked unless TG levels are measured during long-term tigecycline therapy, and TG values should be reported systematically in future reports of tigecycline adverse events.

Conclusion

To our knowledge, no comparable adult case of severe hypertriglyceridemia (TG 2,219 mg/dL) without pancreatitis associated with extended high-dose tigecycline therapy has been published. Causality was probable according to the Naranjo scale, but the absence of baseline lipid values, the gap in monitoring, hypoalbuminemia, and the simultaneous intensification of lipid-lowering therapy mean that the association should be regarded as possible to probable. In published reports of tigecycline-associated pancreatitis, TG levels were rarely measured and, when measured, were normal or mildly elevated. This case illustrates several clinical lessons:

1. During long-term tigecycline therapy, a baseline lipid panel and routine monitoring of TG levels are advisable, especially when high-dose regimens are used.
2. Pre-existing dyslipidemia and interruption of statin therapy, for example, because of hepatotoxicity, may be risk factors for severe hypertriglyceridemia during tigecycline therapy; continuation or substitution of lipid-lowering therapy in patients with known dyslipidemia is advisable whenever liver function allows.
3. Secondary causes should be evaluated promptly, and guideline-based treatment should be initiated when TG levels exceed 500-1,000 mg/dL, while avoiding gemfibrozil in combination with statins.
4. TG values should be systematically reported in future reports of tigecycline-associated adverse events.

Consent for Publication

Written informed consent for publication of this case report and accompanying data was obtained from the patient.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors declare no conflicts of interest.

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References

[1] Greer ND. Tigecycline (Tygacil): the first in the glycylcycline class of antibiotics. Proc (Bayl Univ Med Cent). 2006 Apr;19(2):155-61. [**PMID**: 16609746]

[2] Zha L, Pan L, Guo J, French N, Villanueva EV, Tefsen B. Effectiveness and Safety of High Dose Tigecycline for the Treatment of Severe Infections: A Systematic Review and Meta-Analysis. Adv Ther. 2020 Mar;37(3):1049-64. [**PMID**: 32006240]

[3] De Pascale G, Montini L, Pennisi M, Bernini V, Maviglia R, Bello G, Spanu T, Tumbarello M, Antonelli M. High dose tigecycline in critically ill patients with severe infections due to multidrug-resistant bacteria. Crit Care. 2014 May 5;18(3):R90. [**PMID**: 24887101]

[4] Leng B, Yan G, Wang C, Shen C, Zhang W, Wang W. Dose optimisation based on pharmacokinetic/pharmacodynamic target of tigecycline. J Glob Antimicrob Resist. 2021;25:315-22.

[5] Yang N, Zheng X, Ji X, Yao H, Xu K, Zhang T, Jin L, Zhu H, Wang M. LC-MS-Based Serum Metabolomic Analysis Predicts the Risk of Tigecycline-Induced Coagulopathy in Critically Ill Patients. Drug Des Devel Ther. 2025 Sep 13;19:8237-50. [**PMID**: 40969845]

[6] Marot JC, Jonckheere S, Munyentwali H, Belkhir L, Vandercam B, Yombi JC. Tigecycline-induced acute pancreatitis: about two cases and review of the literature. Acta Clin Belg. 2012 May-Jun;67(3):229-32. [**PMID**: 22897076]

[7] Marshall SR. Tigecycline-induced pancreatitis. Hosp Pharm. 2009;44(3):239-41.

[8] Arafat H, Sadaqa D, Sabateen A. Tigecycline-induced pancreatitis in a patient with recurrent malignancy: a case report and literature review. Ann Med Surg (Lond). 2024 Apr 29;86(6):3717-20. [**PMID**: 38846851]

[9] Fang W, Yi D, Sun L, Wang C. Analysis of clinical characteristics of tigecycline-induced acute pancreatitis. J Clin Pharm Ther. 2020 Dec;45(6):1320-24. [**PMID**:

32649800]

[10] Pan J, Ye C, Zhou LZ, Li ZY, Wang J, He X, Chen SJ, Zhou GQ. The Spectrum of Tigecycline-Induced Pancreatitis in Clinical Characteristics, Diagnosis, and Management. Int J Gen Med. 2023 Jul 12;16:2971-79. [**PMID**: 37465554]

[11] Leow TYS, Bekkers S, Henriet SSV, Hol MKS, Janssen AM, Jansen TTG, Kunst HPM, Pegge SAH, Waterval JJ, van Ingen J, Van Aerde KJ. Safety and tolerability of a standardized treatment regimen for Mycobacterium abscessus otomastoiditis. Int J Pediatr Otorhinolaryngol. 2026 Feb;201:112715. [**PMID**: 41529611]

[12] Naranjo CA, Busto U, Sellers EM, Sandor P, Ruiz I, Roberts EA, Janecek E, Domecq C, Greenblatt DJ. A method for estimating the probability of adverse drug reactions. Clin Pharmacol Ther. 1981 Aug;30(2):239-45. [**PMID**: 7249508]

[13] Pulipati VP, Brinton EA, Hatipoglu B. Management of Mild-to-Moderate Hypertriglyceridemia. Endocr Pract. 2022 Nov;28(11):1187-95. [**PMID**: 35850450]

[14] Álvarez-López H, Ruiz-Gastélum E, Díaz-Aragón A. Current treatment of hypertriglyceridemia. Cardiovasc Metab Sci. 2021;32(Suppl 3):S242-46. Spanish.

[15] Berglund L, Brunzell JD, Goldberg AC, Goldberg IJ, Sacks F, Murad MH, Stalenhoef AF; Endocrine society. Evaluation and treatment of hypertriglyceridemia: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2012 Sep;97(9):2969-89. Erratum in: J Clin Endocrinol Metab. 2015 Dec;100(12):4685. Erratum in: J Clin Endocrinol Metab. 2026 Jan 21;111(2):e647. [**PMID**: 22962670]

[16] Karalis DG. A Review of Clinical Practice Guidelines for the Management of Hypertriglyceridemia: A Focus on High Dose Omega-3 Fatty Acids. Adv Ther. 2017 Feb;34(2):300-23. [**PMID**: 27981496]

[17] Skulas-Ray AC, Wilson PWF, Harris WS, Brinton EA, Kris-Etherton PM, Richter CK, Jacobson TA, Engler MB, Miller M, Robinson JG, Blum CB, Rodriguez-Leyva D, de Ferranti SD, Welty FK; American Heart Association Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Lifestyle and Cardiometabolic Health; Council on Cardiovascular Disease in the Young; Council on Cardiovascular and Stroke Nursing; and Council on Clinical Cardiology. Omega-3 Fatty Acids for the Management of Hypertriglyceridemia: A Science Advisory From the American Heart Association. Circulation. 2019 Sep 17;140(12):e673-e691. [**PMID**: 31422671]

[18] Nelson SD, Munger MA. Icosapent ethyl for treatment of elevated triglyceride levels. Ann Pharmacother. 2013 Nov;47(11):1517-23. [**PMID**: 24259598]

[19] Bhatt DL, Steg PG, Miller M, Brinton EA, Jacobson TA, Ketchum SB, Doyle RT Jr, Juliano RA, Jiao L, Granowitz C, Tardif JC, Ballantyne CM; REDUCE-IT Investigators. Cardiovascular Risk Reduction with Icosapent Ethyl for Hypertriglyceridemia. N Engl J Med. 2019 Jan 3;380(1):11-22. [**PMID**: 30415628]

[20] Crémieux AC, Dinh A, Nordmann P, Mouton W, Tattevin P, Ghout I, Jayol A, Aimer O, Gatin L, Verdier MC, Saleh-Mghir A, Laurent F. Efficacy of colistin alone and in various combinations for the treatment of experimental osteomyelitis due to carbapenemase-producing Klebsiella pneumoniae. J Antimicrob Chemother. 2019 Sep 1;74(9):2666-75. [**PMID**: 31263884]

[21] Tsilika M, Ntziora F, Giannitsioti E. Antimicrobial Treatment Options for Multidrug Resistant Gram-Negative Pathogens in Bone and Joint Infections. Pathogens. 2025 Feb 1;14(2):130. [**PMID**: 40005507]

[22] Lew DP, Waldvogel FA. Osteomyelitis. Lancet. 2004 Jul 24-30;364(9431):369-79. [**PMID**: 15276398]

[23] Gilson M, Moachon L, Jeanne L, Dumaine V, Eyrolle L, Morand P, Ben m'Rad M, Salmon D. Acute pancreatitis related to tigecycline: case report and review of the literature. Scand J Infect Dis. 2008;40(8):681-83. [**PMID**: 18979610]

[24] Lipshitz J, Kruh J, Cheung P, Cassagnol M. Tigecycline-induced pancreatitis. J Clin Gastroenterol. 2009 Jan;43(1):93. [**PMID**: 18769361]

[25] Hung WY, Kogelman L, Volpe G, Iafrati M, Davidson L. Tigecycline-induced acute pancreatitis: case report and literature review. Int J Antimicrob Agents. 2009 Nov;34(5):486-89. [**PMID**: 19540093]

[26] Prot-Labarthe S, Youdaren R, Benkerrou M, Basmaci R, Lorrot M. Pediatric acute pancreatitis related to tigecycline. Pediatr Infect Dis J. 2010 Sep;29(9):890-91. [**PMID**: 20720475]

[27] Mascarello M, Papa G, Arnez ZM, Luzzati R. Acute necrotizing pancreatitis related to tigecycline. J Antimicrob Chemother. 2012;67(5):1296-97.

[28] Hemphill MT, Jones KR. Tigecycline-induced acute pancreatitis in a cystic fibrosis patient: A case report and

literature review. J Cyst Fibros. 2016 Jan;15(1):e9-11. [**PMID**: 26282838]

[29] Bernas Albeniz A, Aveiga Valencia DA, Etxeberria Zabala L, Zaldibar-Gerrikagoitia Bilbao J, Aguilera Celorrio L. Acute pancreatitis in ICU secondary to treatment with tigecycline. Rev Esp Anestesiol Reanim. 2017 Jan;64(1):46-49. English, Spanish. [**PMID**: 27609674]

[30] Akhter S, Krishnan P, Kaul P. Tigecycline-Associated Acute Pancreatitis. Am J Ther. 2018 Nov/Dec;25(6):e749-50. [**PMID**: 29672331]

[31] Lin J, Wang R, Chen J. Tigecycline-induced acute pancreatitis in a renal transplant patient: a case report and literature review. BMC Infect Dis. 2018 May 2;18(1):201. [**PMID**: 29720098]

[32] Wang PF, Zou H, Zhu JH, Shi FE. Acute pancreatitis caused by tigecycline: A case report and literature review. Medicine (Baltimore). 2021 Dec 23;100(51):e28245. [**PMID**: 34941095]

[33] Yazirli B, Kara E, Inkaya AC, Maden S, Ozberk U, Yildirim T, Parlak E, Uzun O, Yilmaz SR, Arici M. A case report of tigecycline induced acute pancreatitis in a renal transplant patient and review of the literature: Should we avoid tigecycline in patients on calcineurin inhibitors? Transpl Infect Dis. 2021 Aug;23(4):e13593. [**PMID**: 33655683]

[34] Chang C, Qiao W, Wang H, Zhang X. Tigecycline-associated acute pancreatitis in a child with pulmonary cystic fibrosis: A case report and literature review. Int J Clin Pharmacol Ther. 2022 Jan;60(1):41-45. [**PMID**: 34605395]

[35] Fredj H, Ben Ali H, Mokline A, Ben Saad M, Jami I, Gasri B, Messadi AA. Pancréatitis Aiguë Liée à La Tigécycline Chez Des Patients Brûlés En Réanimation [Acute Pancreatitis Related to Tigecycline in ICU Burn Patients]. Ann Burns Fire Disasters. 2023 Jun 30;36(2):120-24. French. [**PMID**: 38681942]

[36] Maringanti BS, Ganipisetti VM, Jun SS, Flores MI. Tigecycline Tango: A Case of Antibiotic-Induced Pancreatitis. Cureus. 2023 Sep 1;15(9):e44538. [**PMID**: 37790018]

[37] Li ZK, Zheng P. Acute pancreatitis caused by tigecycline and furosemide combination treatment and hypofibrinogenemia caused by tigecycline: A case report. Int J Clin Pharmacol Ther. 2023 Oct;61(10):466-70. [**PMID**: 37548456]

[38] Ren D, Lv M, Ye D, Jin D, Ouyang Y. Bedside ultrasound in tigecycline-associated acute pancreatitis: a case description. Quant Imaging Med Surg. 2023 Dec 1;13(12):8793-98. [**PMID**: 38106276]

[39] Wee HC, Ng RS, Ong LM. Tigecycline-Induced Acute Pancreatitis with Electrocardiogram Changes in Renal Insufficiency Elderly. Eur J Case Rep Intern Med. 2024 Dec 9;11(12):005033. [**PMID**: 39790845]

[40] McGovern PC, Wible M, Korth-Bradley JM, Quintana A. Pancreatitis in tigecycline Phase 3 and 4 clinical studies. J Antimicrob Chemother. 2014 Mar;69(3):773-78. [**PMID**: 24216769]

[41] Okon E, Engell C, van Manen R, Brown J. Tigecycline-related pancreatitis: a review of spontaneous adverse event reports. Pharmacotherapy. 2013 Jan;33(1):63-68. [**PMID**: 23307547]

[42] Tan Q, Zhang Y, Liu M, Tian D, Wu X, Zhou L, Fan W. Clinical characteristics and risk factors for tigecycline-induced pancreatitis in a tertiary hospital: A retrospective study. Br J Clin Pharmacol. 2023 Sep;89(9):2788-97. [**PMID**: 37161703]

[43] Feng L, Hong Y, Fan J, Yang C, Huang Y, Xu Y, Liao G, Su Y. Clinical characteristics and risk factors of tigecycline-induced acute pancreatitis in kidney transplant recipients: a retrospective study. J Antimicrob Chemother. 2025 Jul 1;80(7):1980-87. [**PMID**: 40405828]

[44] Yang M, Li Y, Zhao H, Xiao D, Zhou Q, Qin W, Xu X. Clinical analysis of acute pancreatitis after kidney transplantation: a single-center retrospective observational study. BMC Gastroenterol. 2025 Apr 29;25(1):305. [**PMID**: 40301828]

[45] Lu Y, Wang C, Maidaiti X, Deng A, Cheng G, Chen L, Lu W, Zhang J, Ding N, Zhang Q, Gong W, Zhang L, Yang F, Long P, Ke X, Li W, Gao S, Yang K, Hu W, Sun F, Cheng H. Factors linked to tigecycline-associated acute pancreatitis in a multicenter retrospective nested case-control study in patients. iScience. 2026 Mar 3;29(4):115201. [**PMID**: 41890964]

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