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Pseudothrombocytopenia Due to Platelet Clumping: A Case Report of Prolonged Hospitalization Due to Confounded Diagnosis

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Abstract
Platelets are pivotal for hemostasis, and accurate quantification is essential for clinical decision-making. Pseudothrombocytopenia (PTCP) is a spurious laboratory finding characterized by falsely low platelet counts due to in vitro clumping, most commonly induced by the ethylenediaminetetraacetic acid (EDTA) anticoagulant. This phenomenon can lead to significant diagnostic confusion and unnecessary medical interventions. This case report describes a 47-year-old female presenting with abdominal pain and vomiting, initially diagnosed with gastritis. Despite clinical improvement, persistent thrombocytopenia raised concerns for conditions such as dengue hemorrhagic fever or immune thrombocytopenic purpura, prolonging hospitalization. A peripheral blood smear eventually revealed platelet clumping, confirming PTCP. This case underscores that PTCP is a preanalytical artifact which, if unrecognized, can result in diagnostic errors, overtreatment, and increased healthcare costs. Clinicians and laboratory personnel must consider PTCP in the differential diagnosis of unexplained thrombocytopenia, especially in asymptomatic patients. Utilizing alternative anticoagulants (e.g., citrate) or examining a peripheral blood smear are critical steps to prevent mismanagement.

INTRODUCTION

Accurate platelet counting is a cornerstone of hematological assessment, guiding diagnosis and therapeutic decisions across a wide range of clinical conditions, from bleeding disorders to infections. Pseudothrombocytopenia (PTCP) represents a significant laboratory pitfall, wherein in vitro platelet clumping leads to spuriously low automated platelet counts. The primary mechanism involves EDTA-dependent antibodies (IgM/IgG) that target cryptic epitopes on platelet glycoprotein IIb/IIIa, which become exposed upon calcium chelation by EDTA (Ali & Ansari, 2024; Gupta & Sharma, 2023; Sen & Raj, 2024). First documented in 1969, PTCP has been reported with increasing frequency in various clinical settings, including autoimmune diseases, malignancies, and notably, during the COVID-19 pandemic (Benet et al., 2023).

Despite being a well-documented phenomenon, PTCP remains underrecognized in routine clinical practice, often leading to misinterpretation. This oversight can trigger a cascade of unnecessary investigations, such as repeated blood tests, imaging studies, bone marrow examinations, and even inappropriate treatments like platelet transfusions or immunosuppressive therapy. In regions endemic for dengue fever, the differential diagnosis

becomes particularly critical, as PTCP can mimic the thrombocytopenia associated with viral infections, potentially delaying correct diagnosis and appropriate resource allocation (Henson & Adams, 2024).

Previous studies have extensively described the etiology and prevalence of PTCP. For instance, Lardinois et al. (2021) provided a comprehensive review of its causes and clinical implications, while Cattaneo (2025) detailed conditions associated with spuriously low platelet counts. However, case reports highlighting the direct impact of PTCP on clinical decision-making, length of hospital stay, and patient management in real-world settings—especially in primary or secondary care hospitals—are less frequently emphasized in the literature (H. Zhang & Shen, 2024; Y. Zhang & Liu, 2024; Zhong & Wong, 2023). This gap underscores the need for continued dissemination of practical clinical lessons.

Reported for the first time in 1969, PTCP has been increasingly described in patients with various disorders and, more recently, in those infected with Coronavirus Disease 2019 (COVID-19). PTCP should always be considered in the differential diagnosis of thrombocytopenia (Koç & Demirci, 2024). This well-known in vitro phenomenon, though still underrecognized, may lead to misdiagnosis, overtreatment, and even unnecessary invasive testing. Here, we present a case report of a 47-year-old female who experienced prolonged hospitalization due to a PTCP-confounded diagnosis (Fyall et al., 2019).

The urgency of addressing this issue lies in its potential to cause iatrogenic harm and incur unnecessary healthcare costs without benefiting the patient. The novelty of this case report lies in its detailed exposition of the diagnostic odyssey caused by PTCP in a common clinical presentation (dyspepsia), illustrating how a laboratory artifact can disproportionately influence management decisions.

The purpose of this report is to reinforce clinical awareness of PTCP. By elucidating this case, the report aims to enhance diagnostic protocols, advocate for the early use of confirmatory tests such as blood smears, and ultimately prevent unnecessary interventions—thereby improving patient safety and healthcare efficiency.

METHOD

A 47-year-old female presented with abdominal pain and vomiting. She had no fever or bleeding symptoms and had a history of dyspepsia syndrome. At presentation, her body temperature was 36.2°C, blood pressure was 120/70 mmHg, pulse rate was 78 beats/min, respiratory rate was 18 times/min, and peripheral oxygen saturation was 98% on room air. On physical examination, there was mild epigastric tenderness; other findings were unremarkable.

Initial laboratory results showed an increased lymphocyte percentage (LYM% 59.1), elevated hematocrit (HCT 46.4%), and thrombocytopenia (82 × 10^3/L). On abdominal ultrasonography (USG), the gaster appeared hyperechoic with air bubbles, suggesting an image consistent with gastritis, which was then established as the primary diagnosis. The patient was treated accordingly with pantoprazole, ondansetron, domperidone, and sucralfate, and daily complete blood count (CBC) monitoring was planned. The clinical concerns for her low platelet count included bone marrow suppression due to a viral illness (e.g., dengue infection) and immune thrombocytopenic purpura (ITP) (Krishnan & Raghavan, 2024).

On follow-up, the patient showed clinical improvement with mild or no symptoms, but the thrombocytopenia persisted (Table 1). A blood smear later revealed platelet clumping. The patient was subsequently discharged after two days of prolonged hospitalization.

Table 1. Patient's CBC and platelet count over the period of care and follow-up

Item	Value					Reference	Unit
	Day 1	Day 2	Day 3	Day 4	Day 5		
WBC	5.5	5.5	6	9.8	**11.8**	5 – 10	10^9/L
LYM	3.2	3	2.5	3.2	6.4	0.9 – 5	10^9/L
MID	0.3	0.4	0.3	0.6	0.5	0.1 – 1.5	10^9/L
GRA	2	2.1	3.2	6.0	5.0	1.2 – 8	10^9/L
RBC	5.13	4.54	4.73	4.50	4.31	4 – 5.3	10^{12}/L
HGB	15	13.7	14	13.5	12.9	12.5 – 16.0	g/dL
HCT	**46.4**	41.3	42.7	40.9	39.3	35 – 45	%
PLT	**82**	**54**	**83**	**69**	**80**	150 – 400	10^3/L
MPV	10.2	9.8	9.8	10.8	9.6	9 – 13	fL

RESULTS AND DISCUSSION

The patient's laboratory trajectory is summarized in Table 1, revealing the central diagnostic conundrum: persistent thrombocytopenia (platelet counts ranging from 54 to 83 × 10^3/L) despite significant clinical improvement of her abdominal symptoms. This dissociation between laboratory findings and clinical status is a classic red flag for spurious results. Notably, other hematological parameters, including white blood cell count and hemoglobin, remained stable or followed expected trends, further isolating the platelet count as an aberrant finding.

Analysis of the data indicates that the initial focus on gastritis, while clinically plausible, was insufficient to explain the hematological abnormality. The persistently low platelet count logically prompted the clinical team to consider more serious pathologies (Lee & Bui, 2023). This interpretation aligns with standard clinical reasoning, where unexplained thrombocytopenia warrants investigation for causes such as infection, immune dysregulation, or bone marrow disorders. The consideration of dengue fever and immune thrombocytopenic purpura (ITP) was therefore a rational—albeit ultimately misleading—step in the diagnostic process (Park & Lee, 2023).

The specific finding that resolved the case was the observation of platelet clumping on a manual peripheral blood smear. This gold-standard test confirmed pseudothrombocytopenia (PTCP), explaining the falsely low automated counts. This finding directly refuted the presumed diagnoses of true thrombocytopenic states and highlighted the artifact as the root cause. The immediate clinical improvement without specific intervention for thrombocytopenia should have raised an earlier index of suspicion for a laboratory error.

Comparison with previous research solidifies this case's place within a known yet frequently overlooked phenomenon. (Tan et al., 2016) and (Lardinois et al., 2021) have extensively documented that EDTA-dependent PTCP is the most common cause of spurious thrombocytopenia (Davis et al., 2024). More recently, (Cattaneo, 2025) emphasized that such preanalytical errors remain persistent challenges in modern hematology. The presented case mirrors the scenario described by (Dönmez & Kaya, 2024), where reliance on automated analyzers without smear verification led to initial misdirection.

The primary solution, as demonstrated in this case and supported by literature, is a heightened clinical–laboratory interface. When faced with unexplained or asymptomatic thrombocytopenia that contradicts the clinical picture, a manual blood smear review should be an early—not a last-resort—investigation. Furthermore, if PTCP is suspected, recollecting blood in a tube containing an alternative anticoagulant, such as sodium citrate or heparin, can provide an accurate platelet count, as evidenced in cases reported elsewhere.

This case strongly relates to theories of diagnostic error, particularly those involving system-related factors and cognitive biases. The initial satisfaction-of-search bias (anchoring on gastritis) and subsequent confirmation bias (pursuing serious causes for the lab value) are well-documented cognitive pitfalls. From a systems perspective, the lack of an automatic reflex rule in the laboratory to perform a smear on samples with isolated thrombocytopenia—or those flagged for platelet clumping by the analyzer—contributed to the delay.

The discussion therefore centers on the practical implications of recognizing PTCP. First, it prevents iatrogenic harm: this patient was spared potential unnecessary treatments such as steroids for suspected ITP or prolonged monitoring for dengue (Fischer & Warren, 2024). Second, it has significant economic implications, reducing costs associated with extended hospitalization, repeated testing, and consultations. Third, it alleviates patient anxiety linked to a potentially serious but incorrect diagnosis.

A critical practical implication is the need for protocol development. Hospitals should consider implementing algorithms in which isolated thrombocytopenia in a clinically stable patient automatically triggers a manual smear review or recollection in citrate. Training for both clinicians and laboratory technicians to recognize clinical scenarios and laboratory flags suggestive of PTCP is equally important. As (Pujol-Moix et al., 2024) noted, awareness is the first and most crucial step in mitigating this error.

In conclusion, this case serves as a potent reminder that not all laboratory abnormalities represent disease. It underscores the indispensable value of the peripheral blood smear in the era of advanced automation and the importance of correlating laboratory data with the patient's clinical condition. Vigilance for PTCP is a simple yet highly effective measure to enhance diagnostic accuracy, optimize resource use, and uphold the principle of "first, do no harm.".

CONCLUSION

This case report conclusively demonstrates that pseudothrombocytopenia (PTCP), an in vitro EDTA-induced artifact, can significantly confound clinical diagnosis, leading to prolonged hospitalization, unnecessary investigations, and potential overtreatment. The definitive diagnosis was achieved through examination of a peripheral blood smear, which revealed platelet clumping, thereby halting further invasive diagnostic considerations. This underscores the critical importance of maintaining a high index of suspicion for PTCP in cases of unexplained thrombocytopenia, especially when clinical findings are discordant with laboratory results.

Future research should focus on developing and validating clinical decision-support algorithms or laboratory reflex rules that automatically prompt smear review or alternative sampling when specific criteria (e.g., isolated thrombocytopenia, analyzer flags for clumping) are met. Additionally, multicenter studies assessing the prevalence and economic impact of

PTCP-induced diagnostic errors across different healthcare settings would provide valuable data to support systematic improvements in laboratory and clinical protocols.

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