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Laboratory Interventions for Detecting and Preventing Pseudothrombocytopenia in Complete Blood Count Testing

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ABSTRACT: Pseudothrombocytopenia (PTCP) is an in vitro artifact in which platelets agglutinate in the presence of the anticoagulant ethylenediaminetetraacetic acid (EDTA), producing a spuriously low platelet count on automated hematology analyzers despite normal in vivo platelet numbers. Because the resulting thrombocytopenia is artificial, failure to recognize it can lead to unnecessary platelet transfusions, inappropriate steroid

or immunoglobulin therapy, delayed surgery, and additional costly diagnostics. Laboratory interventions therefore focus on early recognition and systematic verification before any clinical decision is made. The first line of defense is analytical: reviewing the complete blood count histogram and platelet scatterplot for abnormal distribution curves or clumping flags, examining the peripheral blood smear for platelet aggregates at the feathered edge, and comparing the automated count against manual chamber counting. Discrepancies among these methods, particularly a low automated count with visible clumping and a normal manual estimate, strongly suggest PTCP and should trigger a documented verification protocol rather than silent reporting of the erroneous value. Prevention and confirmation strategies build on this initial suspicion through specimen-level interventions designed to demonstrate anticoagulant dependence and to obtain a true count. Recollecting the sample in an alternative anticoagulant—most commonly sodium citrate, and in some cases lithium heparin—often restores the platelet count, while a parallel EDTA tube collected simultaneously allows direct comparison to confirm the EDTA-dependent mechanism. Additional interventions include maintaining consistent pre-analytical conditions such as avoiding underfilled tubes, minimizing venipuncture trauma and prolonged tourniquet application, ensuring prompt mixing and timely analysis, and using warm incubation or aminoglycoside additives in selected refractory cases. Laboratories should also embed interpretative comments and delta-check rules within the reporting workflow to flag unexplained isolated thrombocytopenia, and maintain a structured communication pathway with clinicians so that verified PTCP is clearly annotated in the medical record. Together, these interventions transform PTCP from a hidden source of misdiagnosis into a controlled, transparent analytical event.

INTRODUCTION

Pseudothrombocytopenia (PTCP) is a laboratory artefact in which an automated haematology analyser reports a falsely low platelet count even though the patient's true circulating platelet mass is normal or near normal. The condition is not a true bleeding disorder, but it can mimic serious thrombocytopenia and may trigger unnecessary platelet transfusion, corticosteroid therapy, bone marrow examination, or delay in surgery. Because the reported platelet count is often the single most influential laboratory value guiding these decisions, PTCP is best understood not as a curiosity of the coagulation laboratory but as a patient-safety issue that requires structured laboratory intervention [1].

The most common form of PTCP is EDTA-dependent pseudothrombocytopenia, in which platelet clumping occurs in blood collected into ethylenediaminetetraacetic acid (EDTA) tubes. EDTA chelates calcium and alters the conformation of platelet membrane glycoprotein complexes, exposing cryptic epitopes that are recognised by naturally occurring or acquired antibodies. The resulting platelet agglutinates are too large to be counted as individual platelets by impedance-based analysers, and the analyser therefore reports a spuriously low count. The phenomenon was recognised decades ago and remains a persistent challenge in routine complete blood count (CBC) testing [2].

The clinical importance of PTCP extends beyond the individual result. Patients with unrecognised PTCP may be labelled as thrombocytopenic and subjected to repeated blood tests, unnecessary consultations, and invasive procedures. In some cases, the artefact coexists with genuine thrombocytopenia, making interpretation even more difficult. Laboratories therefore need a systematic approach that includes pre-analytical control, analyser flag recognition, smear review, alternative anticoagulation, and selective disaggregation methods [3].

A further reason for emphasising laboratory intervention is that PTCP is often invisible at the clinical level. The patient may have no bleeding, no bruising, and no history suggestive of a platelet disorder, yet the analyser result may be markedly abnormal. Conversely, a patient with genuine thrombocytopenia may have platelet clumping superimposed on a true low count, which complicates both diagnosis and monitoring. The laboratory is therefore uniquely positioned to distinguish artefact from disease and to prevent the downstream harms of a false result [4]. Effective prevention depends less on a single sophisticated test than on a chain of relatively simple measures applied consistently, including proper phlebotomy, timely analysis, attention to analyser warnings, and a low threshold for microscopic review [5].

Mechanisms of Pseudothrombocytopenia

The central mechanism of EDTA-dependent PTCP involves antibody-mediated platelet agglutination. After EDTA chelates calcium, the GPIIb/IIIa complex on the platelet surface undergoes a conformational change that exposes neoepitopes. Antibodies, most often IgG but sometimes IgM or IgA, bind to these exposed sites and bridge adjacent platelets. The resulting clumps may contain only a few platelets or may be large enough to be counted as leukocytes or ignored altogether by the analyser. This mechanism explains why the platelet count falls in EDTA but may remain normal in citrate or heparin, and why the phenomenon is temperature- and time-dependent [6].

A second mechanism is platelet satellitism, in which platelets adhere to leukocytes, most commonly neutrophils. This adhesion is mediated by bridging antibodies that bind platelet GPIIb/IIIa on one side and FcγRIII (CD16) on the neutrophil surface on the other. Platelet satellitism can produce a low platelet count without the prominent platelet clumps seen in classical EDTA-dependent agglutination, and it may be missed unless the smear is examined carefully at the feathery edge and around leukocytes [7]. A third and less common mechanism is platelet phagocytosis, in which platelets are internalised by neutrophils or other leukocytes, further reducing the number of freely countable platelets [8].

Pre-analytical variables can also produce or amplify platelet clumping. Traumatic venipuncture, prolonged tourniquet application, inadequate mixing of blood with anticoagulant, under-filled tubes, and delayed analysis can all promote platelet activation and aggregation. These factors may act independently of EDTA-dependent antibodies or may worsen an existing tendency to clump. The distinction matters because pre-analytical clumping is potentially preventable through standardised collection and handling, whereas antibody-mediated EDTA-dependent PTCP requires a change in anticoagulant or a disaggregation strategy [9]. Table 1 summarises the principal mechanisms and their morphological correlates.

Table 1. Mechanisms and Morphological Patterns of Pseudothrombocytopenia

Mechanism	Key mediator	Morphological finding on smear	Typical anticoagulant association
EDTA-dependent platelet agglutination	Anti-GPIIb/IIIa autoantibodies	Platelet clumps, often at smear tail	EDTA
Platelet satellitism	Bridging antibodies binding GPIIb/IIIa and FcγRIII	Platelets rosetting around neutrophils	EDTA
Platelet phagocytosis	Antibody- or activation-mediated	Platelets within leukocyte cytoplasm	EDTA, occasionally other
Pre-analytical activation	Inadequate mixing, traumatic venipuncture	Fibrin strands, small aggregates	Any anticoagulant

Prevalence and Clinical Significance

The prevalence of PTCP depends on the population studied and on the definition used. In the general population, EDTA-dependent PTCP is estimated at approximately 0.1%. Among hospitalised patients, estimates range from 0.1% to 2%, and in selected outpatient populations evaluated for isolated thrombocytopenia the proportion can be considerably higher. Higher rates have been reported in critically ill patients, oncology patients, and individuals with autoimmune disease. The variability reflects differences in ascertainment, since many cases are never confirmed by smear review or alternative anticoagulation [10].

The clinical significance of PTCP lies in its capacity to mimic genuine thrombocytopenia. A spuriously low platelet count may lead to inappropriate platelet transfusion, which carries risks of transfusion reaction, infection, and alloimmunisation. It may also lead to unnecessary bone marrow biopsy, misdiagnosis of immune thrombocytopenia, or cancellation of surgery. In patients with known haematological disease, PTCP can obscure the true platelet trend and complicate the assessment of response to treatment. Laboratory recognition of the artefact therefore has direct implications for patient management and resource use [11].

The problem is particularly relevant in emergency and perioperative settings, where a low platelet count may determine whether a procedure proceeds. A rapid laboratory response is needed, but speed must not compromise accuracy. The most effective

approach is to combine a high index of suspicion with a predefined workflow: when a low platelet count is unexplained, the laboratory should review the smear, consider alternative anticoagulants, and communicate the possibility of PTCP to the clinical team. This approach reduces the risk of treating an artefact as if it were disease [12].

Pre-analytical Interventions and Sample Collection

The first line of defence against PTCP is pre-analytical control. Standardised phlebotomy technique, appropriate tube filling, gentle inversion, and prompt transport to the laboratory reduce the likelihood of activation-related clumping. Traumatic venipuncture and prolonged tourniquet application should be avoided because they promote platelet activation and may produce aggregates even in patients without EDTA-dependent antibodies. Under-filled EDTA tubes should be rejected or recollected because the anticoagulant-to-blood ratio is critical for cellular preservation [13].

Time is also an important variable. EDTA-dependent platelet clumping can be time-dependent, with smaller aggregates progressing to larger clumps as the specimen stands. Laboratories should establish maximum turnaround times for EDTA-anticoagulated samples and should prioritise the analysis of samples from patients with known or suspected PTCP. When a sample cannot be analysed promptly, the laboratory should consider whether an alternative anticoagulant should be used for the initial collection, particularly in patients with a previous history of clumping [14].

Training of phlebotomists and laboratory staff is an often overlooked but essential intervention. Staff should understand why tube order, mixing, and filling matter, and they should be able to recognise when a sample is likely to be unsuitable. Feedback from the laboratory to collection sites can reduce repeat collections and improve the overall quality of CBC testing. Pre-analytical quality indicators, such as sample rejection rates and recollect rates for platelet clumping, can be monitored as part of continuous quality improvement [15].

Automated Analyser Flagging and Histogram Analysis

Modern haematology analysers generate flags and histogram patterns that may suggest platelet clumping. Flags such as "PLT clumps?" are intended to alert the operator to an abnormal distribution of platelet events, and platelet histograms may show an abnormal shape or an excessive number of large events. However, the diagnostic accuracy of these flags is imperfect. Small clumps may be counted as leukocytes, producing spurious leukocytosis without a specific platelet flag, while large clumps may cause a low platelet count without a clear warning. The absence of a flag therefore does not exclude PTCP [16].

Analyser flags are best used as triggers for further review rather than as diagnostic endpoints. A clumping flag should prompt smear review, and a low platelet count with an unexplained delta change should prompt review even in the absence of a flag. Platelet indices such as mean platelet volume, platelet distribution width, and platelet-large cell ratio may provide supplementary clues, but their performance varies across analyser platforms and clinical contexts. No index can replace microscopic confirmation when the result is clinically unexpected [17].

The integration of analyser data with laboratory information system rules can improve detection. Delta checks comparing the current platelet count with previous results can flag suspicious falls, particularly when the decrease exceeds a predefined threshold without a clinical explanation. Autoverification rules should be designed to hold low platelet counts for manual review when flags, histogram abnormalities, or delta-check failures are present. This approach allows the laboratory to apply scarce morphological expertise where it is most likely to change the reported result [18].

Peripheral Blood Smear Examination

Microscopic examination of the peripheral blood smear remains the cornerstone of PTCP confirmation. The diagnosis should be suspected in any patient with a low platelet count who lacks signs of bleeding, and it should be actively excluded by smear review when the analyser result is unexpectedly low or when a platelet clumping flag is present. A clump is conventionally defined as a cluster of at least five attached platelets, and such clusters are most easily detected at the margins and tail of the smear. Smear review also identifies platelet satellitism and platelet phagocytosis, which may not produce the same clumping pattern as EDTA-dependent agglutination [19].

The "smear-first" approach is important because mechanical disaggregation can create artefactual aggregates if true clumps are not present. Vortexing a sample that does not contain clumps may introduce small aggregates and produce a false-positive result, while vortexing a sample with true clumps may partially correct the count. The laboratory should therefore confirm the presence of clumps before applying any disaggregation procedure. Smear review should be performed by trained staff and

should include an assessment of platelet morphology, distribution, and association with leukocytes [20]. Table 2 outlines common triggers and actions for smear review.

Table 2. Triggers for Peripheral Blood Smear Review in Suspected Pseudothrombocytopenia

Trigger	Suggested action
Platelet count < 100×10^9/L without bleeding	Review smear for clumps, satellitism, or phagocytosis
Analyser flag "PLT clumps?"	Confirm clumps on smear before reporting
Unexplained fall in platelet count from previous result	Perform delta check and smear review
Platelet count discordant with clinical picture	Review smear and consider alternative anticoagulant
Known history of EDTA-dependent PTCP	Review smear on every subsequent CBC

Alternative Anticoagulants: Citrate, Heparin, and Magnesium Sulphate

When EDTA-dependent PTCP is confirmed or strongly suspected, recollection into an alternative anticoagulant is the most widely used confirmatory and corrective strategy. Sodium citrate is the most common alternative because it is routinely available and preserves platelet counts in many affected patients. However, citrate does not prevent platelet clumping in all cases, and a subset of patients exhibits multi-anticoagulant PTCP with clumping in citrate and heparin as well as in EDTA. Heparin is another option, but it too may fail to resolve the artefact in some individuals [21].

Magnesium sulphate has emerged as a particularly useful alternative because it preserves the integrity of the GPIIb/IIIa complex and thereby keeps the cryptic epitope inaccessible to agglutinins. Studies have shown that magnesium sulphate-anticoagulated samples yield markedly higher platelet counts than EDTA samples in patients with PTCP, without interfering with other blood cell parameters. The choice of alternative anticoagulant should be guided by local availability, the urgency of the result, and the patient's history. In some cases, more than one alternative anticoagulant may need to be tested before a reliable count is obtained [22].

A practical limitation of alternative anticoagulation is that it requires a second venipuncture, which may be inconvenient, time-consuming, and occasionally difficult in patients with poor venous access. Laboratories should therefore establish a clear recollection protocol that specifies which tube should be drawn, how the sample should be labelled, and how the corrected result should be reported. Communication with the clinical team is essential so that treatment decisions are not made on the basis of the initial EDTA result while the confirmatory sample is pending [23]. Table 3 compares the main anticoagulant options.

Table 3. Comparison of Anticoagulants for Platelet Counting in Suspected Pseudothrombocytopenia

Anticoagulant	Mechanism of action	Advantages	Limitations
K_2/K_3-EDTA	Calcium chelation	Standard for CBC; good cellular preservation	Induces GPIIb/IIIa conformational change; common cause of PTCP
Sodium citrate	Calcium chelation	Widely available; often corrects PTCP	Does not resolve all cases; specimen stability limited
Heparin	Antithrombin-mediated	May avoid EDTA-dependent agglutination	Can also induce clumping in some patients
Magnesium sulphate	Preserves GPIIb/IIIa integrity	Effective in many EDTA-PTCP cases; maintains other CBC parameters	Requires additional tube; not universally available

Chemical and Mechanical Disaggregation Strategies

When recollection is impractical or when the clinician requires a rapid result from the original EDTA tube, laboratory interventions aimed at disaggregating platelet clumps can be considered. Vortex mixing is a simple mechanical approach that has been shown to increase platelet counts in EDTA specimens with confirmed clumping. In one study, mean platelet counts

rose substantially after vortexing, while red cell, white cell, and haemoglobin values remained stable. However, vortexing is not uniformly effective, and only a proportion of vortex-mixed specimens achieve platelet counts above the clinically relevant threshold [24].

Aminoglycoside supplementation, particularly with kanamycin or amikacin, has emerged as a chemical disaggregation strategy. Kanamycin added to EDTA blood collection tubes has been reported to dissociate platelet clumps and improve platelet count accuracy, likely by interfering with the antibody-mediated bridging that sustains agglutination. Amikacin has similarly been used to prevent or reverse platelet aggregation in EDTA-dependent PTCP, and it offers the practical advantage of being added directly to the EDTA tube without a second venipuncture. These chemical methods are not universally successful, and they should be reserved for situations in which smear review has already confirmed the presence of clumps [25].

Temperature Modulation

Temperature is a modifiable pre-analytical variable that can influence the stability of platelet clumps. EDTA-dependent antiplatelet antibodies often react optimally at low temperatures, and warming the sample to 37°C can reduce agglutination in some patients. Maintaining the specimen at 37°C until the platelet count is completed has been recommended as part of a practical prevention strategy, particularly when citrate or magnesium sulphate tubes are used [21]. However, temperature modulation is not a stand-alone solution: it does not correct all cases, and it must be combined with appropriate anticoagulant selection and smear confirmation.

Laboratory Protocols, Delta Checks, and Quality Assurance

A robust laboratory response to PTCP requires more than a single intervention; it requires an integrated protocol. Delta checks comparing the current platelet count with previous results can flag suspicious falls, particularly when the decrease exceeds 30–40% without a corresponding clinical explanation. Laboratory information systems can be programmed to recognise EDTA-PTCP patterns and to prompt smear review or alternative anticoagulant testing when predefined criteria are met [16]. Autoverification rules should be designed to hold low platelet counts for manual review, especially when analyser flags, histogram abnormalities, or delta-check failures are present.

Quality assurance should include periodic audit of smear-review concordance, turnaround times for PTCP work-up, and the proportion of recollections that yield corrected platelet counts. Staff training in the recognition of platelet clumps and satellitism is equally important, because the morphological diagnosis remains operator-dependent. Laboratories should also document PTCP status in the laboratory information system so that subsequent CBCs are interpreted with appropriate caution [24]. A written protocol, rather than individual discretion, is the best safeguard against both under-recognition and over-treatment.

Impact on Patient Care and Clinical Communication

The ultimate purpose of laboratory intervention is to protect patients from the consequences of a false result. When PTCP is identified, the laboratory should report the corrected platelet count along with a comment indicating the presence of EDTA-dependent pseudothrombocytopenia, so that clinicians do not misinterpret the initial low value. Communication with the clinical team is essential when a corrected count is not immediately available, particularly in patients for whom treatment decisions—such as platelet transfusion or surgery—depend on the platelet count. Documenting the patient's PTCP status in the laboratory information system enables faster recognition on subsequent encounters and reduces repeat recollections [3].

Emerging Technologies and Future Directions

Newer analytical approaches are beginning to change the laboratory landscape for PTCP. Fluorescence-based and optical platelet counting methods can sometimes distinguish platelets from clumps more effectively than impedance methods, and digital morphology systems are being explored for automated recognition of platelet aggregates. Artificial intelligence-assisted smear analysis holds promise for improving the sensitivity and reproducibility of clump detection, although these technologies are not yet a substitute for trained morphological review [17]. As analysers become more sophisticated, the laboratory protocol for PTCP will likely shift from reactive smear review toward earlier, algorithm-driven suspicion and targeted confirmation.

CONCLUSION

Pseudothrombocytopenia is a benign in vitro phenomenon with potentially serious clinical consequences if unrecognised. Laboratory interventions span the entire testing pathway: meticulous pre-analytical technique, awareness of analyser flags and

histogram clues, prompt smear review, selective use of alternative anticoagulants such as citrate or magnesium sulphate, and, where appropriate, mechanical or chemical disaggregation with vortexing or aminoglycosides. No single intervention is universally effective, and the most reliable approach is a layered protocol that combines delta checks, flag-triggered smear review, and confirmatory recollection when indicated. By integrating these measures into routine practice, laboratories can prevent misdiagnosis, reduce unnecessary procedures, and ensure that the platelet count reported to the clinician reflects the patient's true haematological status.

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