

When the Donor Is the Root Cause: Platelet Concentrate Quality Control Failure Due to Constitutional Macroplatelets Detected During a Vertical Audit

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Trupti Lokhande ¹, Dnyaneshwar Patale ¹, Vinita Paswan ², Arunpreet Kaur ¹

¹. Transfusion Medicine, All India Institute of Medical Sciences, Raebareli, Raebareli, IND ². Pathology, All India Institute of Medical Sciences, Raebareli, Raebareli, IND

Corresponding author: Trupti Lokhande, drtrupti.lokhande02@gmail.com

Abstract

Quality control (QC) failure of a whole blood-derived platelet concentrate (PC) is usually attributed to process-related factors, such as centrifugation settings, separation technique, holding time, or storage conditions. We report an isolated PC QC failure in which the root cause was traced to donor platelet morphology. During the first vertical audit of component preparation at our newly established blood centre, four whole blood units processed by the platelet-rich plasma (PRP) method were prospectively traced. One PC showed absent swirling during processing and contained only 5×10^5 platelets/ μL in 55 mL, corresponding to a yield of 0.03×10^{10} (2.75×10^8) platelets, far below the regulatory requirement of $\geq 4.5 \times 10^{10}$. By contrast, the three PCs processed concurrently had platelet yields of 5.40 – 6.69×10^{10} and met QC requirements. The index donor was a 42-year-old male with a normal pre-donation platelet count ($356 \times 10^3/\mu\text{L}$), but the mean platelet volume (MPV) was elevated (12.4 fL), with a platelet-large cell ratio (P-LCR) of 43.7% and an analyser flag for giant platelets. Peripheral smear examination demonstrated numerous large and giant platelets. Component tracing revealed that only 0.17% of the donor's circulating platelets were recovered in the PC, compared with 47.4–71.8% in the other three units. Conversely, 26.0% of the donor's platelets were retained in the packed red cell component, compared with 4.4–13.0% in the other units. Furthermore, the few platelets recovered in the PC were smaller, with an MPV of 9.5 fL and P-LCR of 18.7%, compared with 12.4 fL and 43.7% in the donor's whole blood sample. These findings suggest size-dependent co-sedimentation of macroplatelets with red cells during the soft-spin step of PRP preparation, resulting in markedly reduced platelet recovery in the PC. The donor had no bleeding history, and macroplatelets persisted on repeat testing. This case highlights that donor-related platelet morphology can be an important and overlooked cause of isolated PC QC failure. Evaluation of platelet indices and peripheral smear morphology should therefore be considered during root cause analysis of an unexpected, isolated PC failure, particularly in populations where constitutional macroplatelet disorders may occur.

Categories: Hematology, Pathology, Quality Improvement

Keywords: blood component quality control, giant platelets, inherited macrothrombocytopenia, macroplatelets, platelet concentrate, platelet-rich plasma method, platelet swirling, platelet yield, root cause analysis, vertical audit

Introduction

Platelet concentrates (PCs) prepared from whole blood remain an important source of platelet support in Indian blood centres and are produced using either the platelet-rich plasma (PRP) or buffy-coat method. In the PRP method, whole blood is first centrifuged at low force (light spin) so that red cells sediment while platelets remain suspended in the plasma; the PRP is then centrifuged at high force (heavy spin) to pellet the platelets, which are resuspended in a small volume of plasma. Because sedimentation velocity increases with particle size, the efficiency of this separation depends on the size difference between platelets and red cells. Under the Drugs and Cosmetics Rules, a random donor PC prepared from a 450 mL whole blood donation should contain at least 4.5×10^{10} platelets in at least 75% of the units tested [1]. Indian audits have reported that approximately four out of five whole blood-derived PCs meet the prescribed quality-control (QC) requirements [2].

When a PC fails QC, the investigation generally focuses on process-related factors, including centrifugation speed, time and temperature, pre-separation holding time, component expression technique, and storage conditions [2,3]. However, donor-related factors may also influence platelet recovery and are less commonly considered during root cause analysis. In particular, platelet size and morphology may affect the distribution of platelets during differential centrifugation.

Asymptomatic constitutional macrothrombocytopenia, later termed Harris platelet syndrome, was first described in healthy blood donors from West Bengal [4]. It is characterised by giant platelets, mild-to-moderate thrombocytopenia, normal platelet aggregation, and the absence of bleeding and appears to be autosomal dominant [5,6]. A recent multicentric study of 2,574 Indian blood donors found

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macrothrombocytopenia in 4%, concentrated in the eastern zone but also present in the north, west, and south [7], and a similar prevalence has been reported among migrants from northern and eastern states [8]. Importantly, a proportion of affected donors have a normal platelet count with a mean platelet volume (MPV) above 12 fL [5], so the trait may go unnoticed when only the platelet count is screened.

The relevance of donor macroplatelets to apheresis donor selection has been discussed [9], but their effect on whole blood-derived components has received little attention. We describe a PC that failed QC during a vertical audit, in which root cause analysis led back to constitutional macroplatelets in an otherwise healthy donor.

Case Presentation

Setting and audit design

Our blood centre was newly established, and in August 2023, the first vertical audit of component preparation was conducted, with individual blood units traced through the entire process from donor selection to preparation of the final components. On the day of the audit, four whole blood donations from donors weighing more than 55 kg were selected at random. All donors met the eligibility criteria specified in the Drugs and Cosmetics Rules and the departmental standard operating procedure (SOP).

Each donor underwent a standard 450 mL whole blood donation into a quadruple blood bag system containing citrate-phosphate-dextrose (CPD)/saline-adenine-glucose-mannitol (SAGM) (Polymed). An ethylenediaminetetraacetic acid (EDTA) sample was collected from the diversion pouch for pre-donation complete blood count (CBC) analysis.

All four units were processed within eight hours of collection by the PRP method in the same batch, by the same technician, using the same centrifuge. A light spin at 1,450 RPM (684 g), 12 minutes, 22°C, separated the PRP, which was expressed into a satellite bag. A heavy spin at 3,950 RPM (5076 g), five minutes, 22°C, then pelleted the platelets, and platelet-poor plasma was removed, leaving the PC in 50–60 mL of plasma. The PC was rested for one hour and stored at 20–24°C with continuous agitation. Component volumes were derived from net weight and specific gravity. Complete blood counts were performed on the pre-donation sample and on samples from the packed red blood cells (PRBCs) and the PC using a Sysmex XN-1000 analyser (Sysmex Corporation, Kobe, Japan). Swirling was assessed visually by gently agitating each bag against a light source and recorded as present or absent.

Non-conforming unit

Swirling was absent in the PC from one donor at the time of processing. Its platelet count was $5 \times 10^3/\mu\text{L}$ in a volume of 55 mL, giving a yield of 0.03×10^{10} (2.75×10^8 platelets), less than 1% of the regulatory minimum. The PCs from the other three donors had yields of 5.78, 5.40, and 6.69×10^{10} all showed swirling, and all met the QC criteria (Table 1). Overall, 71.8%, 47.4%, and 48.6% of the whole blood platelets were recovered in these three PCs, compared with 0.17% for the index unit. The few platelets in the failed PC were smaller than those in the donor's whole blood (MPV 9.5 vs. 12.4 fL; P-LCR 18.7% vs. 43.7%) (Table 2). The PRBC unit from the same donation had a platelet count of $122 \times 10^3/\mu\text{L}$, against $19\text{--}30 \times 10^3/\mu\text{L}$ in the other three units, and retained 26.0% of the platelets in the whole blood unit compared with 4.4–13.0% in the others (Table 1). About 74% of the index donor's platelets were therefore not accounted for in either the PRBC or the PC, compared with 15–47% in the other units; this fraction would include platelets in the plasma component and any platelets not detected by the analyser.

Parameter	Donor 1	Donor 2	Donor 3	Index donor	Reference / specification
Whole blood volume (mL)	452	450	450	454	450 ± 10% [1]
Whole blood platelet count (×10 ³ /μL)	178	253	306	356	150–410*
PRBC volume (mL)	350	345	316	345	—
PRBC platelet count (×10 ³ /μL)	30	21	19	122	—
Platelets retained in PRBC (% of whole blood)	13.0	6.4	4.4	26.0	—
PC volume (mL)	60	50	55	55	50–70 [1]
PC platelet count (×10 ³ /μL)	963	1079	1217	5	—
PC platelet yield (×10 ¹⁰ /unit)	5.78	5.40	6.69	0.03	≥4.5 in ≥75% of units tested [1]
Platelets recovered in PC (% of whole blood)	71.8	47.4	48.6	0.17	—
Swirling	Present	Present	Present	Absent	Present [1]
QC outcome	Pass	Pass	Pass	Fail	

TABLE 1: Component volumes and platelet distribution in the four units traced during the vertical audit
Platelets retained or recovered = (count × volume in PRBC or PC) ÷ (count × volume in whole blood) × 100. Yield = count (/μL) × volume (mL) × 1000.
*Laboratory reference range (Sysmex XN-1000). Dashes indicate parameters without a regulatory specification.
PRBC: packed red blood cells; PC: platelet concentrate; QC: quality control

Donor investigations

The donor was a 42-year-old male who was originally from North India. The pre-donation complete blood count showed haemoglobin of 13.5 g/dL, white cell count of 9.94 × 10³/μL, platelet count 356 × 10³/μL, MPV 12.4 fL, platelet distribution width (PDW) 16.5 fL, P-LCR 43.7%, and plateletcrit 0.44% (Table 2). The analyser flagged the platelet parameters as unreliable and raised a "Giant Platelet?" message. It also flagged mild eosinophilia (8.2%; 0.82 × 10³/μL), which we considered incidental. A Leishman-stained peripheral smear showed numerous large and giant platelets, many approaching or exceeding the diameter of adjacent red cells, along with occasional small platelet clumps (Figure 1). Neutrophils showed no Döhle-like cytoplasmic inclusions, and red cell morphology was unremarkable.

Parameter	Donor sample	Platelet concentrate	Unit	Laboratory reference range (adult male)
White blood cells	9.94	0.03	×10 ³ /μL	4.0–10.0
Red blood cells	4.60	0.01	×10 ⁶ /μL	4.5–5.5
Haemoglobin	13.5	0.1	g/dL	13.0–17.0
Haematocrit	43.6	0.0	%	40–50
MCV	94.8	—	fL	83–101
Platelet count	356	5	×10 ³ /μL	150–410
Mean platelet volume	12.4	9.5	fL	9.4–12.3
Platelet distribution width	16.5	8.0	fL	9.8–16.2
Platelet-large cell ratio	43.7	18.7	%	19.7–42.4
Plateletcrit	0.44	0.01	%	0.17–0.35
Eosinophils	0.82 (8.2%)	—	×10 ³ /μL	0.02–0.50
Analyser flags	Giant platelet?; eosinophilia	None		

TABLE 2: Haematological parameters of the index donor's pre-donation sample and of the failed platelet concentrate (Sysmex XN-1000)

Reference ranges are the laboratory's reference ranges for the Sysmex XN-1000 analyser and apply to the donor sample. Platelet parameters in the donor sample were flagged by the analyser as of reduced reliability.

MCV: mean corpuscular volume

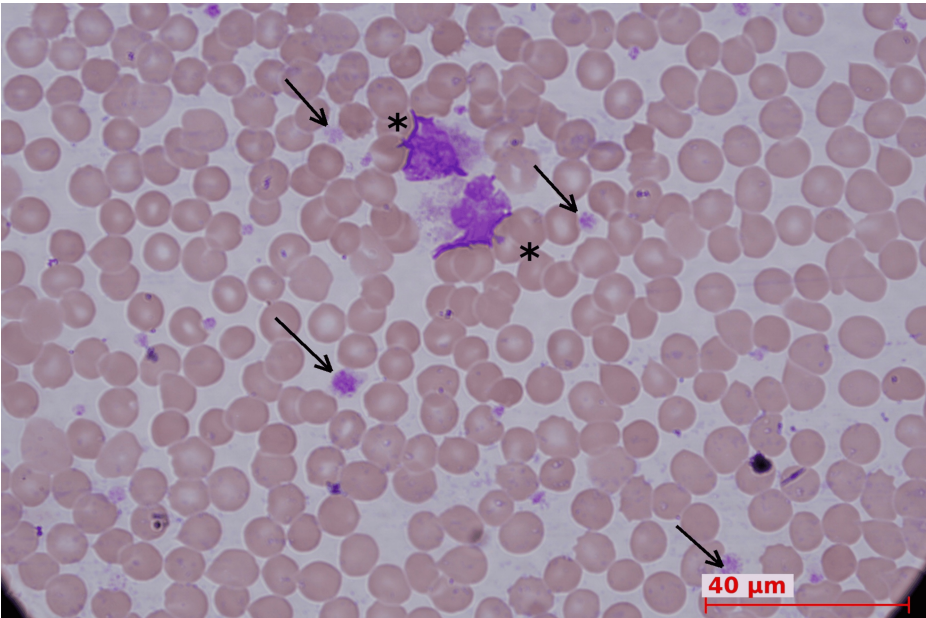


FIGURE 1: Peripheral blood smear of the index donor showing giant platelets

Peripheral blood smear of the index donor showing numerous large and giant platelets scattered among red cells, with two platelet clumps in the centre of the field (Leishman stain ×1000 oil immersion; scale bar 40 μm).

The donor had been found fit on pre-donation medical examination. When contacted by telephone, the donor reported no history of epistaxis, gum bleeding, easy bruising, or prolonged bleeding after injury or

dental extraction, no recent drug intake (including antiplatelet agents), and no known family history of a bleeding disorder or low platelet count. A repeat EDTA sample taken one week after donation and analysed on the same analyser showed a platelet count of $342 \times 10^3/\mu\text{L}$ and an MPV of 12.8 fL, with persistent large and giant platelets on the smear. Family screening, platelet function testing and genetic testing were not performed. The donor was counselled about the finding and advised to undergo haematology evaluation, and has not donated since.

Root cause analysis

Process-related causes were examined systematically (Table 3). The three units processed in the same run, with the same equipment, consumables and staff, recovered 47.4–71.8% of their whole blood platelets in the PC, whereas the index unit recovered 0.17%. A failure of this magnitude confined to one unit made a process cause unlikely. Process-related causes could not be completely excluded, but the donor's macroplatelets were considered the most likely contributing factor. As corrective and preventive action, the departmental SOP was amended so that the donor's MPV, P-LCR, and analyser flags, and a peripheral smear, are reviewed whenever an individual PC fails QC, before process changes are made.

Category	Factor examined	Finding
Personnel	Training, adherence to SOP	Same trained technician processed all four units
Equipment	Centrifuge programme, calibration, temperature; plasma expressor	Same run; calibration valid; other units passed, recovering 47–72% of platelets
Method	Holding time, spin settings, expression, resting	Within SOP for all units
Materials	Bag type, lot, anticoagulant	Same lot
Measurement	Analyser IQC, sampling and mixing of the PC	IQC within limits ,count verified on repeat
Donor	Platelet count, indices and morphology	Normal count; MPV 12.4 fL; P-LCR 43.7%; giant platelets on smear — root cause

TABLE 3: Root cause analysis of the platelet concentrate QC failure

IQC: internal quality control; SOP: standard operating procedure; MPV: mean platelet volume; P-LCR: platelet-large cell ratio

Discussion

This case demonstrates that a PC may fail QC because of a donor-related factor rather than a problem in component preparation. The three units processed in the same run showed platelet recovery in the PC ranging from 47.4% to 71.8%, whereas only 0.17% of the donor's platelets were recovered in the index PC. The marked disparity, with the abnormality confined to a single unit processed under the same conditions, made a generalized process-related failure unlikely and prompted investigation of donor-specific factors.

The stepwise platelet counts obtained during the vertical audit were particularly valuable in localizing the abnormality. Rather than simply documenting a low platelet yield in the final PC, the audit demonstrated redistribution of the donor's platelets, with a substantial proportion retained in the packed red cell component. Without this component-by-component tracing, the isolated QC failure could have been attributed to random process variation and might have led to unnecessary reassessment or modification of centrifugation parameters.

Proposed mechanism

The most likely mechanism is physical and relates to differential sedimentation during the light-spin step of the PRP method. During this step, red cells sediment while platelets, because of their smaller size and lower effective sedimentation behaviour, are preferentially retained in the plasma phase [10]. According to Stokes' law, under idealized conditions, sedimentation velocity increases with the square of particle radius. Thus, an increase in platelet size can substantially increase its sedimentation tendency. Markedly enlarged platelets may consequently sediment more rapidly than normal-sized platelets and become preferentially associated with the red cell layer or the red cell-plasma interface, reducing their recovery in the PRP.

Our findings support such size-selective platelet loss. The PRBC component from the index unit retained 26.0% of the donor's platelets, compared with 4.4–13.0% in the other three units. Moreover, the platelets recovered in the PC were substantially smaller than those present in the donor's circulation, with an MPV of 9.5 fL and P-LCR of 18.7% compared with 12.4 fL and 43.7%, respectively, in the donor sample. Because the PRP and plasma fractions were not counted, we cannot determine whether these platelets remained in the

plasma, were lost during the heavy spin, or were simply not detected by the analyser. The data therefore establish a near-complete failure of platelet recovery associated with donor macroplatelets, but not the step at which the platelets were lost.

The absence of swirling was the first visible sign. Swirling depends on an adequate concentration of discoid platelets [11]; it is usually lost through platelet activation or a fall in pH during storage, but in this unit it reflected the near-absence of platelets. Absent swirling in a freshly prepared PC should therefore prompt a platelet count rather than being attributed automatically to poor processing.

The proportion of platelets retained in the PRBC component is also likely to be underestimated. Automated impedance-based cell counters classify particles according to their measured size and may misclassify very large platelets as red cells, particularly in samples containing a high concentration of erythrocytes [12]. In addition, platelet clumping observed on the donor's peripheral smear may have further reduced the number of individually detected platelets in the component samples. These effects could underestimate the whole blood and PRBC platelet counts and make the recovery percentages imprecise. They are unlikely to explain the PC result, however, because the PC contained almost no red cells ($0.01 \times 10^6/\mu\text{L}$) and no visible aggregates. Processing variability was minimised because all four units were processed in the same run under the same SOP, but a unit-specific handling factor cannot be completely excluded. An absent bleeding history does not exclude an inherited platelet disorder, and without family, functional, or genetic studies, the donor's phenotype remains a suspected constitutional trait rather than an established diagnosis.

Taken together, these findings provide a plausible explanation for the exceptionally low platelet recovery in the index PC: the donor's large and giant platelets were preferentially excluded from the PRP fraction and retained with the red cell component, leaving predominantly smaller platelets available for recovery in the PC. Whether a similar size-selective effect occurs with the buffy-coat method remains uncertain and warrants further investigation.

Donor phenotype and differential diagnosis

The donor had marked macroplatelets without thrombocytopenia. This phenotype is consistent with the spectrum described in Harris platelet syndrome, in which affected individuals may have a normal platelet count with an MPV above 12 fL [5]. However, as genetic testing was not performed in this case, we have used the descriptive term constitutional macroplatelets rather than assigning a specific inherited diagnosis.

The differential diagnosis included MYH9-related disorders, which may be associated with large platelets and Döhle-like neutrophil inclusions and can occur with extra-haematological manifestations such as hearing loss, nephropathy and cataract [12]. Bernard-Soulier syndrome was also considered, although the absence of a bleeding history made this less likely. In vitro platelet clumping, including EDTA-dependent pseudothrombocytopenia, was another consideration because it can produce spuriously low platelet counts and abnormal platelet indices.

The absence of bleeding symptoms, absence of leukocyte inclusions, normal platelet count, and persistence of large and giant platelets on repeat testing argued against an analytical artefact and supported a constitutional macroplatelet phenotype. Although such phenotypes are generally considered clinically innocuous, mild bleeding has occasionally been reported in individuals with Bengal macrothrombocytopenia [13]. A detailed bleeding history was therefore obtained before attributing the finding to an apparently benign constitutional phenotype.

Practical implications

This case has practical implications for both QC and donor evaluation. Automated impedance analysers classify platelets largely according to particle size and may misclassify very large platelets as red cells, resulting in an underestimated platelet count [12]. In donors with suspected macroplatelets, optical or fluorescence-based platelet counting, or manual verification, may therefore provide a more accurate assessment [12].

Macroplatelets have been reported in approximately 4% of Indian blood donors [7]. Although the present case represents an isolated observation, this relatively common phenotype raises the possibility that donor-related platelet morphology may contribute to a proportion of otherwise unexplained PC QC failures in Indian blood centres. Based on this single observation, review of the analyser's giant-platelet flag, the MPV and P-LCR and, where indicated, a peripheral smear may be considered during investigation of an isolated PC failure. This approach has not yet been evaluated prospectively and requires validation in a larger series before wider adoption.

The findings also raise an important question regarding future donor management. Whether whole blood from donors with marked macroplatelets should be preferentially diverted from whole blood-derived PC preparation, or whether such donors may be suitable candidates for platelet apheresis, remains uncertain. Previous observations of good post-transfusion platelet recovery from products collected by apheresis from donors with giant platelets [9] provide a basis for further investigation, but prospective studies are required

before any change in donor selection or component-processing policy can be recommended.

This report has several limitations. It describes a single donor. Platelet counts were not performed on the intermediate PRP or platelet-poor plasma fractions, so a complete platelet mass balance could not be made and about 74% of the donor's platelets were unaccounted for. All counts were impedance counts, without fluorescence, manual or citrate-sample verification, and may be affected by giant platelets and clumping. Family screening, platelet function testing, and genetic testing were not performed. Finally, the amended SOP has not yet been evaluated in subsequent QC investigations

Conclusions

In this vertical audit, a PC prepared from a healthy donor with constitutional macroplatelets recovered almost none of the donor's platelets, whereas three units processed alongside it performed normally. Donor macroplatelets were the most likely contributing factor; size-dependent co-sedimentation with red cells is a plausible mechanism, but intermediate fractions were not measured and most of the platelet mass was not accounted for, so the mechanism requires confirmation. Including donor platelet indices, analyser flags and morphology in the investigation of isolated PC failures may help avoid unnecessary process changes.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Trupti Lokhande

Acquisition, analysis, or interpretation of data: Trupti Lokhande, Dnyaneshwar Patale, Vinita Paswan, Arunpreet Kaur

Drafting of the manuscript: Trupti Lokhande

Critical review of the manuscript for important intellectual content: Trupti Lokhande, Dnyaneshwar Patale, Vinita Paswan, Arunpreet Kaur

Disclosures

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