



Case Reports

Corticosteroids and Bandemia: A Case Report and Review of the Literature

Jessica Moore, M.D.¹, Fred Schiffman, MD, MACP²

¹ Department of Internal Medicine, Boston Medical Center,

² Division of Hematology and Oncology, Department of Medicine, Brown University

Keywords: Bandemia, dexamethasone, immune thrombocytopenic purpura

Journal of Brown Hospital Medicine

Vol. 5, Issue 1, 2026

Article Information

Keywords: Bandemia, dexamethasone, immune thrombocytopenic purpura

<https://doi.org/10.56305/001c.154359>

Submitted: August 10, 2025
EDT

Accepted: December 14, 2025
EDT

Published: January 01, 2026
EDT

Abstract

While it is known that glucocorticoids can affect the immune system and increase white blood cell count, there is limited evidence suggesting that they directly lead to the presence of immature neutrophils (bandemia) in the bloodstream. To our knowledge, studies specifically looking at bandemia after corticosteroid use, in humans, have not been done. Our case highlights that a single high dose of dexamethasone may cause bandemia in otherwise healthy patients without signs of infection, or inflammation.

BACKGROUND

Glucocorticoids are used pharmacologically to treat inflammatory, allergic, and immunological disorders. Inhibition of phospholipases and cytokine transcription has been reported as a major mechanism of this anti-inflammatory action.¹ Research shows that glucocorticoids markedly increase the number of circulating polymorphonuclear leukocytes (PMNs) available to participate in the inflammatory response. Several possible mechanisms for the glucocorticoid-induced granulocytosis have been proposed, including enhanced release of PMNs from bone marrow, delayed apoptosis of PMNs in the circulation, reduced egress of PMNs into inflamed tissues, and decreased expression of adhesion molecules such as Mac-1 (CD11b) and L-selectin (CD62L), which reduce neutrophil adherence to the endothelium and promote their release into the bloodstream.¹⁻³ Mishler and colleagues found that the maximum neutrophil count occurred 4-6 hours after oral or intravenous (IV) administration of dexamethasone and was due almost entirely to an increase in mature neutrophils.⁴

While it is known that glucocorticoids can affect the immune system and increase white blood cell (WBC) count, there is limited evidence suggesting that they directly cause the release of immature neutrophils in the bloodstream. Bandemia refers to an elevated number of immature white blood cells in the peripheral blood, called band cells. Band neutrophils are immature forms of neutrophils characterized morphologically by a nucleus that is curved or horseshoe-shaped but not yet segmented, distinguishing them from mature segmented neutrophils. It is not a specific diagnostic marker but can be seen in

various clinical contexts, such as infection or inflammation, that might stimulate the bone marrow to release immature cells. Bandemia has demonstrated high specificity (often >90%) for infection, though sensitivity is moderate, and its presence should heighten suspicion for occult infection, especially in patients with otherwise normal WBC counts. Older animal studies conducted before the year 2000 have shown that administration of glucocorticoids can increase the number of nonsegmented PMNs (band form) in the circulation, perhaps due to bone marrow release of PMNs and demargination from the endothelial surfaces of blood vessels.

To our knowledge, studies specifically examining bandemia after corticosteroid use in humans have not been conducted. The objective of the current report is to demonstrate that a single high dose of dexamethasone may cause bandemia in patients without signs of infection or inflammation.

CASE PRESENTATION

A 59-year-old woman with no significant past medical history presented to the emergency department after her complete blood count (CBC) showed a platelet count of 6000/uL during a primary care visit. She reported feeling fatigued and bruising easily over the previous weeks. Laboratory studies showed that her hemoglobin and WBC count were within normal range. Peripheral blood smear review revealed isolated thrombocytopenia with large platelets, without schistocytes, suggesting immune thrombocytopenic purpura. The patient was started on IV dexamethasone 40 mg once a day for four days.

Table 1. WBC and platelet counts before and following dexamethasone therapy

	Before dexamethasone	6 hours after one dose of 40mg dexamethasone	15 hours after one dose of 40mg dexamethasone	24 hours after second dose of 40mg dexamethasone	One week after four doses of 40mg dexamethasone was complete
Platelet count/uL	6,000	8,000	19,000	62,000	63,000
WBC count/uL	7,900	8,700	23,100	30,000	13,200
% Bands	Undetectable	Undetectable	8%	5%	Undetectable

Six hours after the patient received her first dose of dexamethasone, her CBC showed a platelet count of 8,000/uL and white blood cell (WBC) count of 8,700/uL with no evidence of bandemia. The following morning, before her second dose of dexamethasone, her platelet count was 19,000/uL, WBC 23,100/uL, and 8% bands. The patient reported feeling well with normal vital signs. She then received her second dose of 40mg IV dexamethasone. The following morning, her platelet count had increased to 62,000/uL, her WBC count increased to 30,000/uL, and there were 5% bands. There were no symptoms or other signs of infection. One week after her four-day course of dexamethasone was completed, her WBC count was 13,200/uL and bands were undetectable, and her platelet count had improved to 63,000/uL.

DISCUSSION

Bandemia is defined as an increased proportion of immature neutrophils, or bands, in the peripheral blood, when greater than 10% of the total WBC differential or a band count greater than or equal to 1,500 cells/mm³.⁵ This threshold is widely used in clinical practice and research, and is rooted in its inclusion as a sign of systemic inflammatory response syndrome (SIRS) and sepsis. Studies show odds ratios for bandemia and infection ranging from 6 to 8 and specificity around 90%, but sensitivity is low (40–43%). The predictive value is inferior to procalcitonin (PCT) and to C-reactive protein (CRP), but it remains clinically useful, especially when combined with other findings.⁵

Measurement of bandemia is typically performed by manual differential count on a peripheral blood smear, in which a laboratory technologist visually identifies and counts band forms among 100 leukocytes. Some automated hematology analyzers attempt to quantify bands, but manual review remains the standard in most settings. There are significant limitations to band count measurements, such as inter-observer variability in reported band counts, as poor agreement among laboratory personnel occurs in distinguishing bands from segmented neutrophils, especially in cases that are not morphologically clear.⁶ Vergara-Lluri et al. demonstrated marked variability

in band counts on the 100-cell differential count for both College of American Pathologists (CAP) proficiency testing (PT) program participants and CAP Hematology and Clinical Microscopy Committee (HCMC) members (coefficients of variation, 55.8% and 32.9%, respectively).⁶ Another limitation is that laboratories report band counts without established reference ranges, and the criteria for identifying bands are not universally standardized.⁶ Finally, automated analyzers may not reliably distinguish bands, and manual counts are labor-intensive and subject to human error.⁶ Given these limitations, some hematology experts and professional organizations recommend grouping bands with segmented neutrophils in reporting, rather than reporting bands separately. Despite these issues, bandemia remains a commonly used, though imperfect, marker of infection and inflammation in clinical practice.

Corticosteroid-induced leukocytosis is typically characterized by an increase in mature neutrophils without a significant left shift or bandemia. However, corticosteroids can induce leukocytosis with a left shift, including increased band forms. This effect is due to corticosteroid-induced demargination of neutrophils and inhibition of neutrophil apoptosis, leading to increased circulating neutrophils and, in some cases, immature forms.³ Corticosteroids also stimulate the release of mature neutrophils from the bone marrow reserve and impede neutrophil migration out of the circulation to sites of inflammation, further increasing blood counts.⁷ The most common patient populations affected by corticosteroid-induced leukocytosis in clinical practice are those receiving corticosteroids for chronic inflammatory, autoimmune, or pulmonary conditions, as well as patients with malignancies or undergoing immunosuppressive therapy.

Prior studies have established that even small doses of glucocorticoids, administered over short or prolonged periods, can induce leukocytosis.^{8–10} This phenomenon makes diagnosing suspected infection in patients prescribed steroids particularly challenging, especially considering that one of the most important adverse effects of corticosteroids is increased susceptibility to infection. It is commonly understood that a left shift in peripheral white blood cells (more than 6 percent band forms) and the ap-

pearance of toxic granulation and vacuoles in neutrophils may assist in the differential diagnosis of infection. Prior studies involving animals found corticosteroids to induce bandemia that is not necessarily related to infectious causes. To our knowledge, research specifically examining bandemia after corticosteroid use in humans has not been conducted. Our case report suggests that bandemia can occur shortly after a dose of 40mg dexamethasone, without any signs of infection.

Using rabbits as test subjects, Nakagawa and colleagues discovered that dexamethasone increased WBC counts from $8,900 \pm 2,000 \times 10^6/\text{uL}$ to $13,400 \pm 1,100 \times 10^6/\text{uL}$ at 6 hours ($P=0.028$ at 4 to 6 hours), and counts returned to the baseline value by 48 hours. Dexamethasone increased PMN counts from $4,600 \pm 800 \times 10^6/\text{uL}$ to $11,600 \pm 1.1 \times 10^6/\text{uL}$ at 6 hours ($P=0.004$ at 4 to 24 hours), and counts returned to baseline by 48 hours.² Similar to these findings and other studies involving humans, our patient mounted an elevated WBC count shortly after a dose of steroids. Nakagawa et al. also established that dexamethasone increased the band cell counts from $280 \pm 50 \times 10^6/\text{uL}$ to $1,280 \pm 250 \times 10^6/\text{uL}$ in rabbits at 6 hours ($P=0.016$ at 4 to 12 hours), and counts returned to the baseline value by 48 hours. The percentage of band cell counts increased from $6.1 \pm 1.0\%$ to $11.0 \pm 1.6\%$ at 6 hours and $11.0 \pm 1.4\%$ at 12 hours ($P=0.044$ at 2 to 12 hours), and values returned to the baseline value by 48 hours after dexamethasone treatment.² The authors postulated that the origin

of some of the band cells may be in the intravascular marginated PMN pools in the lung microvessels. Similar to these findings, our patient also developed bandemia (8% bands) after one dose of dexamethasone 40mg. Her lack of clinical or laboratory findings suggestive of infection, and the findings from Nakagawa and colleagues, support that our patient's bandemia was associated with dexamethasone.²

A limitation of this report is that there was no manual smear review; therefore, the band number may have been overestimated. Future research might investigate the incidence of bandemia in more patients taking corticosteroids who do not have signs of infection. Similar to our understanding that corticosteroids can increase mature WBC, the finding that they may also cause bandemia might help interpret this finding in other patients.

Disclosures/Conflicts of Interest

None

Corresponding author

Jessica Moore, M.D
Department of Internal Medicine
Boston Medical Center, Boston, MA
Email: jessica.moore@bmc.org



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