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A Perfect MDS Mimic: Zinc-Driven Copper Deficiency After Gastric Bypass in a Patient Using Denture Adhesive

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, 58-year-old
Final Diagnosis: Copper deficiency • neuropathy • zinc toxicity
Symptoms: Leukopenia • MDS • severe anemia
Clinical Procedure: —
Specialty: Hematology • General and Internal Medicine

Objective: Unusual clinical course

Background: Copper deficiency—an underrecognized cause of anemia, neutropenia, and neuropathy—can mimic myelodysplastic syndrome (MDS), particularly in patients with prior bariatric surgery or excess zinc exposure. Recognition is critical because hematologic and neurologic manifestations may resemble clonal bone marrow disorders but are potentially reversible with appropriate treatment.

Case Report: A 58-year-old woman with prior Roux-en-Y gastric bypass displayed progressive fatigue, exertional dyspnea, and distal paresthesias. Laboratory evaluation revealed severe hypoproliferative anemia (hemoglobin 5.8 g/dL) and neutropenia (absolute neutrophil count 980/μL; nadir <500/μL). Peripheral smear showed anisopoikilocytosis and pseudo-Pelger-Huët-like changes, suggesting MDS. Subsequently, the patient developed febrile neutropenia and coronavirus disease 2019. Vitamin B12 (910 pg/mL) and folate (6.8 ng/mL) levels were normal. Hemolysis markers were not significantly elevated (lactate dehydrogenase 149 U/L, haptoglobin 205 mg/dL). Serum copper was below 10 μg/dL (reference, 77-206 μg/dL), ceruloplasmin was below 60 mg/L (reference, 200-600 mg/L), and serum zinc was elevated (130 μg/dL [reference, 60-106 μg/dL]). Bone marrow biopsy showed preserved trilineage hematopoiesis without increased blasts, supporting a nutritional etiology. Further assessment revealed nonadherence to recommended multivitamin and mineral supplementation, along with chronic excessive use of zinc-containing denture adhesive. The patient received intravenous followed by oral copper supplementation and underwent discontinuation of zinc exposure, resulting in rapid hematologic improvement and partial resolution of neurologic symptoms.

Conclusions: Copper deficiency should be considered in patients with unexplained cytopenias and neurologic symptoms, especially after bariatric surgery or under chronic zinc exposure. With early recognition, this condition is reversible; patients can avoid unnecessary evaluation, misdiagnosis, and complications.

Keywords: Anemia • Copper Deficiency • Gastric Bypass • Hematology • Zinc

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952171

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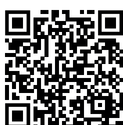

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Introduction

Copper deficiency commonly manifests as fatigue, pallor, anemia, leukopenia or neutropenia, recurrent infections, and peripheral neuropathy. Copper-dependent enzymes, including ceruloplasmin and hephaestin, are essential for iron mobilization and effective erythropoiesis; impaired activity contributes to hypoproliferative anemia, which may be normocytic or microcytic [1]. Copper is also a critical cofactor for enzymes involved in cell division and protein synthesis; thus, deficiency can result in cytopenias.

Neutropenia secondary to copper deficiency has been attributed to impaired maturation and vacuolization of myeloid precursors, as well as ineffective granulopoiesis [2]. Peripheral blood and bone marrow findings can substantially overlap with myelodysplastic syndrome (MDS), a clonal bone marrow disorder characterized by dysplastic hematopoiesis and cytopenias. In cases of copper deficiency, bone marrow may exhibit ring sideroblasts and dysplastic-appearing changes; unlike findings in MDS, copper-deficiency-impacted bone marrow most often demonstrates preserved trilineage hematopoiesis with characteristic vacuolization of erythroid and myeloid precursors, rather than clonal dysplasia [3]. This overlap is clinically relevant because nutritional marrow changes are potentially reversible, whereas MDS carries distinct prognostic and therapeutic implications.

Copper deficiency can also cause neurologic manifestations, including sensory ataxia and distal paresthesias, potentially due to dysfunction of copper-dependent enzymes such as cytochrome c oxidase and impaired dorsal column function. These findings may be clinically indistinguishable from vitamin-B12-deficiency-related myelopathy [4]. In many patients, hematologic abnormalities improve more rapidly than neurologic symptoms, and delayed recognition can result in incomplete neurologic recovery.

Copper deficiency may arise from reduced intake, malabsorption, or excess zinc exposure. Copper is predominantly absorbed in the stomach and proximal small intestine. Accordingly, patients who undergo Roux-en-Y gastric bypass have a particularly high risk because these absorptive segments are bypassed [5]. Postoperative nutritional surveillance often focuses on iron, folate, and vitamin B12 deficiency; however, copper deficiency may remain undetected until clinically significant cytopenias or neurologic symptoms develop.

Zinc toxicity often results from supplements, cold remedies, or inadvertent sources such as zinc-containing denture adhesives. Zinc—primarily absorbed in the duodenum and jejunum—induces metallothionein, which preferentially binds copper and reduces systemic copper availability [5,6]. Thus, unrecognized chronic zinc exposure can precipitate or exacerbate copper deficiency.

Recent studies in bariatric populations indicate that abnormalities in copper and zinc metabolism are not uncommon. Zarshenas and colleagues reported measurable copper and zinc abnormalities both before and after bariatric surgery, emphasizing the need for long-term trace element monitoring beyond the early postoperative period [7]. These findings underscore the importance of considering micronutrient deficiencies when evaluating unexplained hematologic abnormalities in patients with a history of bariatric surgery.

Multiple reports have described denture-adhesive-associated zinc excess resulting in clinically significant hypocupremia, cytopenias, and neurologic complications [8,9]. Given that denture adhesives are often overlooked during medication reconciliation, chronic exposure might remain unrecognized unless clinicians specifically inquire about dental products and other nonprescription sources of zinc.

Here, we describe a patient who presented with cytopenias due to zinc-induced copper deficiency after Roux-en-Y gastric bypass and chronic exposure to zinc-containing denture adhesive.

Case Report

A 58-year-old woman with a history of Roux-en-Y gastric bypass presented with progressive fatigue, exertional dyspnea, and paresthesias in her fingers and toes. Outpatient testing revealed a hemoglobin level of 5.8 g/dL, prompting hospital admission.

On presentation, she was hemodynamically stable but considerably pale, without hepatosplenomegaly or lymphadenopathy. Initial laboratory evaluation demonstrated bicytopenia, with a white blood cell count of 2.0×10^3/µL (absolute neutrophil count [ANC] 980/µL), hemoglobin 5.8 g/dL, and platelets 232×10^3/µL; platelet count later declined to a nadir of 104×10^3/µL. The reticulocyte production index was 0.4, consistent with hypoproliferative anemia.

Additional laboratory testing showed vitamin B12 910 pg/mL and folate 6.8 ng/mL, both within their normal ranges. Hemolysis markers were not significantly elevated (lactate dehydrogenase 149 U/L, haptoglobin 205 mg/dL). Iron studies demonstrated ferritin 198.5 ng/mL, serum iron 38 µg/dL, total iron-binding capacity 279 µg/dL, transferrin 199 mg/dL, and transferrin saturation 14%. Key hematologic and biochemical findings are summarized in **Table 1**.

Peripheral smear showed anisopoikilocytosis and pseudo-Pelger-Huët anomaly, raising concern for MDS. While awaiting bone marrow biopsy, the patient's ANC fell below 500/µL, and she developed neutropenic fever. Testing subsequently

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Table 1. Hematologic and biochemical laboratory findings during hospitalization.

Laboratory parameter	Result	Reference range
White blood cell count	2.0×10^3/μL	4.0-10.0×10^3/μL
Absolute neutrophil count	980/μL (nadir <500/μL)	1500-8000/μL
Hemoglobin	5.8 g/dL	12-16 g/dL
Platelets	232×10^3/μL (nadir 104×10^3/μL)	150-400×10^3/μL
Reticulocyte production index	0.4	>2 suggests appropriate response
Vitamin B12	910 pg/mL	200-914 pg/mL
Folate	6.8 ng/mL	>4 ng/mL
Lactate dehydrogenase	149 U/L	120-250 U/L
Haptoglobin	205 mg/dL	30-200 mg/dL
Ferritin	198.5 ng/mL	13-150 ng/mL
Serum iron	38 μg/dL	50-170 μg/dL
Total iron-binding capacity	279 μg/dL	250-370 μg/dL
Transferrin saturation	14%	20-50%
Serum copper	<10 μg/dL	77-206 μg/dL
Ceruloplasmin	<60 mg/L	200-600 mg/L
Serum zinc	130 μg/dL	60-106 μg/dL

Values demonstrate severe hypoproliferative anemia and neutropenia in the context of profound copper deficiency and elevated serum zinc levels.

showed coronavirus disease 2019 (COVID-19) positivity. Broad-spectrum antimicrobials and supportive care were initiated.

Given her bariatric history and clinical presentation, micronutrient testing was performed, revealing profound copper deficiency (serum copper <10 μg/dL [reference, 77-206 μg/dL]; ceruloplasmin <60 mg/L [reference, 200-600 mg/L]). Subsequent testing showed concomitant zinc excess (130 μg/dL [reference, 60-106 μg/dL]). Further history revealed chronic daily use of a zinc-containing denture adhesive (Fixodent®) for several years since cleft palate surgery. The patient reported applying amounts that exceeded the manufacturer's recommended dosage. This exposure had not initially been regarded as clinically relevant.

Bone marrow biopsy demonstrated trilineage hematopoiesis without significant dysplasia; findings were compatible with nutritional deficiency, rather than a clonal myeloid neoplasm. Representative bone marrow aspirate and core biopsy findings are shown in **Figures 1 and 2**. Flow cytometry demonstrated polyclonal B cells and normal T-cell populations, without immunophenotypic evidence of leukemia or lymphoma. Cytogenetic studies were unavailable.

She received intravenous copper (2 mg daily for 5 days), followed by oral copper supplementation, and was advised to discontinue the zinc-containing denture adhesive. Her counts promptly improved, such that the white blood cell count increased from 0.7 to 2.7×10^3/μL and hemoglobin rose from 5.8 to 8.8 g/dL prior to discharge. Systemic symptoms improved; distal paresthesias partially regressed but did not fully resolve during early follow-up.

Discussion

Copper deficiency is a well-documented but often overlooked cause of anemia, neutropenia, and bone marrow abnormalities that can closely mimic MDS [1,2,10,11]. The overlap between these conditions is clinically significant because both may present with cytopenias, morphologic abnormalities on peripheral smear or marrow examination, and nonspecific systemic symptoms. Consequently, patients with copper deficiency may initially undergo extensive hematologic evaluation for suspected clonal marrow disease.

In the present case, severe hypoproliferative anemia, neutropenia, and pseudo-Pelger-Huët forms on peripheral smear appropriately raised concern for MDS. However, the clinical context provided important clues suggesting an alternative diagnosis. The patient had a history of Roux-en-Y gastric bypass, which predisposes to trace element deficiencies, and she

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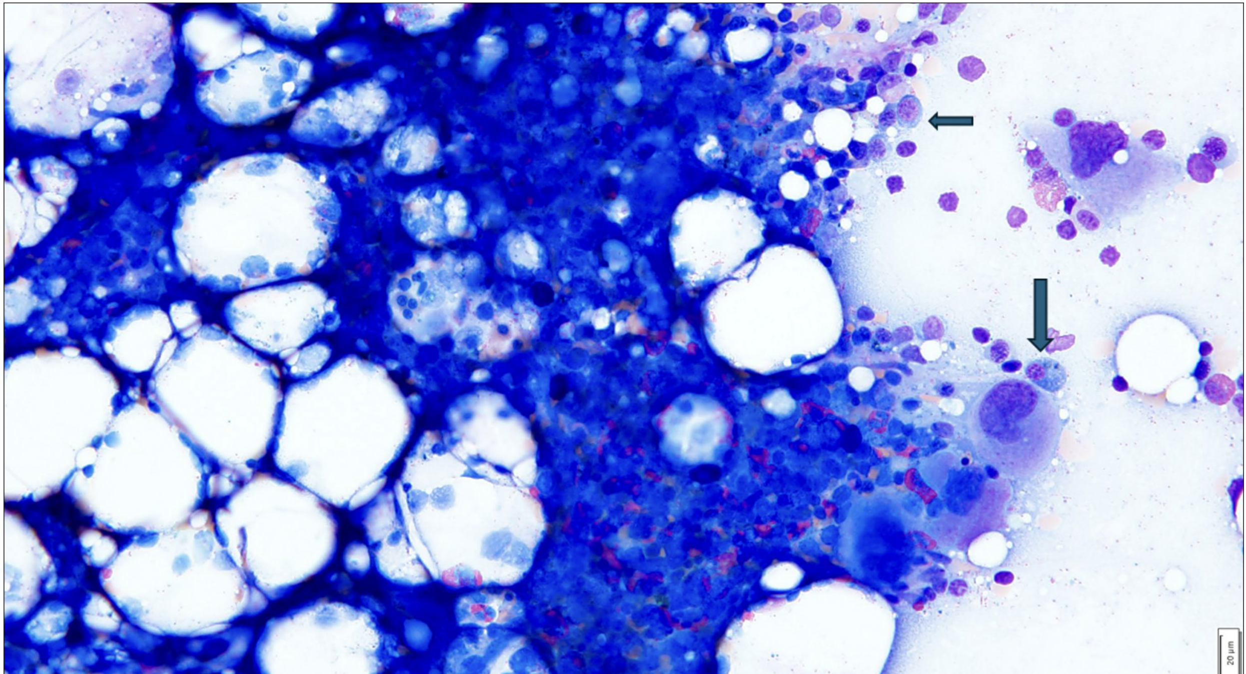

Figure 1. Bone marrow aspirate smear showing cytoplasmic vacuolization characteristic of copper deficiency. Wright-Giemsa-stained bone marrow aspirate demonstrating cytoplasmic vacuolization in erythroid and myeloid precursor cells (arrows). These vacuolated precursors reflect impaired maturation associated with copper deficiency and can mimic morphologic features observed in myelodysplastic syndrome. Arrows indicate representative erythroid precursors with prominent cytoplasmic vacuoles. Original magnification ×1000 (oil immersion; scale bar, 20 μm).

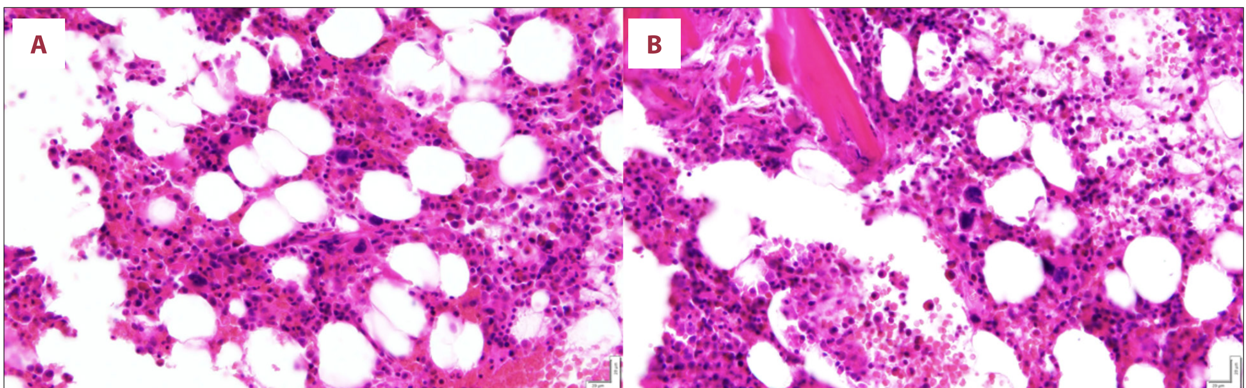

Figure 2. Bone marrow core biopsy demonstrating preserved hematopoiesis without increased blasts. (**A, B**) Hematoxylin- and eosin-stained bone marrow core biopsy sections from the left iliac crest showing variably cellular marrow with preserved trilineage hematopoiesis. Erythroid precursors exhibit mild nuclear irregularities, and occasional megakaryocytes show subtle hypolobation without clustering or overt micromegakaryocytic proliferation. No clinically significant increase in blasts is identified. Cytoplasmic vacuolization is not readily appreciable in core biopsy sections. Original magnification ×40 (scale bar, 20 μm).

reported chronic excessive exposure to zinc-containing denture adhesive. Collectively, these factors increased the likelihood of copper depletion.

The pathophysiology of zinc-induced copper deficiency is well established. Excess zinc stimulates intestinal metallothionein synthesis. Metallothionein binds copper more strongly than zinc, trapping copper within enterocytes and increasing fecal copper loss as enterocytes are shed [6]. In patients with bariatric-surgery-induced impairment of gastrointestinal absorption, this mechanism may convert marginal copper balance into severe systemic deficiency. Such a combined effect likely explains the profound cytopenias observed in our patient.

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The bone marrow findings also supported a nutritional etiology. Copper deficiency can produce morphologic abnormalities, including cytoplasmic vacuolization of erythroid and myeloid precursors, ring sideroblasts, and mild dyspoietic changes. These findings can resemble manifestations in MDS and therefore present a diagnostic challenge [3,10]. However, the absence of increased blasts and preservation of trilineage hematopoiesis favor a nonclonal process. The rapid hematologic response to copper replacement further supported the diagnosis of a reversible nutritional deficiency.

Neurologic manifestations of copper deficiency vary in severity. Some patients present with mild distal paresthesias, whereas others develop sensory ataxia, spastic gait, or myeloneuropathy that may only partially improve despite treatment [2,4]. Taylor and Jeyarajan recently reported severe copper deficiency myelopathy after bariatric surgery, demonstrating how delayed recognition can lead to substantial neurologic disability [12]. In the present case, neurologic symptoms were limited to distal paresthesias without gait disturbance; partial improvement occurred after copper replacement, suggesting relatively early recognition.

Trace element deficiencies after bariatric surgery are increasingly recognized. Zarshenas and colleagues investigated the prevalences of copper and zinc abnormalities in bariatric patients, revealing that such abnormalities can occur both before and after surgery [7]. These findings emphasize the importance of ongoing nutritional surveillance in the bariatric surgery population and support early evaluation of trace elements in patients who present with unexplained cytopenias.

The denture adhesive exposure in the present case is particularly instructive. Zinc-containing dental fixatives represent a modifiable and frequently overlooked source of excessive zinc intake. Prior case reports have linked chronic use of zinc-containing denture adhesives to hypocupremia, cytopenias, and neurologic injury [8,9]. Patients often apply larger-than-recommended amounts in an attempt to improve denture stability; they may be unaware of the systemic effects of chronic zinc exposure.

From a clinical perspective, our case illustrates an important principle: reversible nutritional deficiencies should be excluded before considering clonal hematologic disorders. Formal diagnosis of MDS carries substantial emotional and clinical implications, including long-term surveillance and potential therapeutic interventions. In contrast, copper deficiency is often simple to treat once recognized. Accordingly, this case underscores the importance of careful history-taking, including specific questions about nutritional supplements, denture adhesives, and other potential sources of zinc exposure. In patients with prior bariatric surgery, evaluation of trace element levels should be considered early during the diagnostic workup of unexplained cytopenias.

Conclusions

Copper deficiency should be considered in the differential diagnosis of unexplained cytopenias and neurologic symptoms, especially among patients with a history of bariatric surgery or chronic exposure to zinc-containing products. Recognition of this reversible cause of an MDS-like presentation is critical: timely copper replacement leads to rapid hematologic recovery, whereas delayed diagnosis can result in preventable infectious morbidity and irreversible neurologic injury.

Institution Where Work Was Done

Charleston Area Medical Center, Charleston, WV, USA.

Patient Consent

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

Disclosure

An artificial-intelligence-based language model was used solely to assist with language editing and clarity during manuscript preparation. All clinical data, interpretation, and conclusions were generated and verified by the authors.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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