EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Coden USA: EJBPBO
Volume: 13
Issue: 3
Year: 2026

ANOREXIA NERVOSA IN AN OSTEOSARCOMA SURVIVOR: SEVERE CACHEXIA COMPLICATED BY PANCYTOPENIA, DYSELECTROLYTEMIA, AND METABOLIC COLLAPSE

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DOI: https://doi.org/10.5281/zenodo.18796830

How to cite this Article: *[1]Dr. E. Narendiran, [2]Dr. A. Rajesh, [3]Dr. G. Rathnakumar, [4]Dr. J. Auspas. (2026). Anorexia Nervosa In An Osteosarcoma Survivor: Severe Cachexia Complicated By Pancytopenia, Dyselectrolytemia, And Metabolic Collapse. European Journal of Biomedical and Pharmaceutical Sciences, 13(3), 274–279.

Article Received on 30/01/2026 **Article Revised on 19/02/2026** **Article Published on 01/03/2026**

ABSTRACT

Anorexia Nervosa (AN) presents a unique diagnostic challenge in cancer survivors, where severe malnutrition can physiologically mimic metastatic recurrence. The sequelae of starvation—including "starvation marrow" and metabolic collapse—create a complex clinical picture often indistinguishable from terminal malignancy. We report the case of a 27-year-old osteosarcoma survivor who presented with profound cachexia and drowsiness following a history of chronic constipation and laxative abuse. Her clinical course was complicated by life-threatening instability, including profound hypoglycemia (25 mg/dL), sinus bradycardia (46 bpm), and marked QTc prolongation (654 ms). Additionally, she exhibited pancytopenia (Hb 7.1 g/dL, Platelets 24,000/mm³), initially raising concern for bone marrow metastasis. This case highlights the critical need to distinguish starvation physiology from oncologic progression to ensure appropriate management.

KEYWORDS: •Anorexia Nervosa • Severe Cachexia • Laxative Abuse • Pancytopenia • Sinus Bradycardia • QTc Prolongation • Hypoglycemia • Osteosarcoma Survivor.

INTRODUCTION

Anorexia Nervosa (AN) is a serious psychiatric disorder characterized by a relentless pursuit of thinness, a distorted body image, and an intense fear of gaining weight, leading to severe restriction of energy intake relative to requirements. It has the highest mortality rate of any psychiatric disorder, driven by both suicide and the profound systemic sequelae of starvation. The pathophysiology involves a state of hypometabolic adaptation to conserve energy, resulting in multi-organ dysfunction. Key clinical features include significant weight loss (often leading to a BMI < 18.5 kg/m²), bradycardia, hypotension, and hypothermia. Systemic complications are widespread, affecting the cardiovascular system (arrhythmias, QTc prolongation), hematopoietic system (pancytopenia due to gelatinous marrow transformation), and endocrine system (hypothalamic amenorrhea, hypoglycemia). In patients with a history of malignancy, the severe cachexia and metabolic derangements of AN can mimic cancer recurrence or paraneoplastic syndromes. Distinguishing between the hypercatabolic state of malignancy and the hypometabolic state of starvation is critical for appropriate management and to avoid unnecessary invasive testing.

CASE PRESENTATION

A 27-year-old female presented to the emergency department with chief complaints of diffuse abdominal pain and progressive weight loss over the preceding two weeks. She also reported generalized weakness and drowsiness. Her past medical history was significant for osteosarcoma of the left lower limb, for which she had undergone wide excision surgery and completed chemotherapy in 2018. She had no other known comorbidities.

Upon further evaluation, the patient disclosed a history of chronic constipation persisting for two years. She admitted to the chronic use of laxatives (Bisacodyl) to

manage her bowel habits. Her history also revealed reduced oral intake, regurgitation of feeds, and dyspepsia. She denied any history of hematochezia, vomiting, loose stools, fever, headache, or blurring of vision.

On general examination, the patient was conscious but drowsy and appeared severely emaciated and cachectic. There was significant wasting of subcutaneous fat, specifically noted as a loss of buccal fat pads and wasting of temporal and periorbital fat. Generalized muscle wasting was evident, with prominent wasting of the small muscles of the hand (thenar and hypothenar eminences).

Dermatological and mucosal examination revealed

- Pallor.
- Bilateral ichthyosis of the feet with intertrigo in the web spaces.
- Onychomycosis of the great toes.
- Oral cavity findings included pale glossitis, mucosal erosions over the tongue and palate, and a whitish membrane suggestive of oral candidiasis.

The patient was hemodynamically stable but exhibited signs of hypometabolism:

- **Blood Pressure:** Hypotensive at 90/60 mmHg.
- **Pulse Rate:** Significant bradycardia at 46–50 beats per minute.
- **Respiratory Rate:** 16–18 breaths per minute.
- **Saturation:** 99% on room air.
- **Weight:** 23 kg
- **Height:** 157cm
- **BMI:** 9.3 kg/m^2

Systemic Examination

- **Abdomen:** Soft and non-tender with audible bowel sounds; no organomegaly or free fluid was palpable.
- **Cardiovascular:** S1 and S2 were heard; no murmurs or added sounds were appreciated.
- **Respiratory:** Bilateral air entry was equal with normal vesicular breath sounds.
- **Central Nervous System:** The patient was oriented, with normal gait and movement in all four limbs.

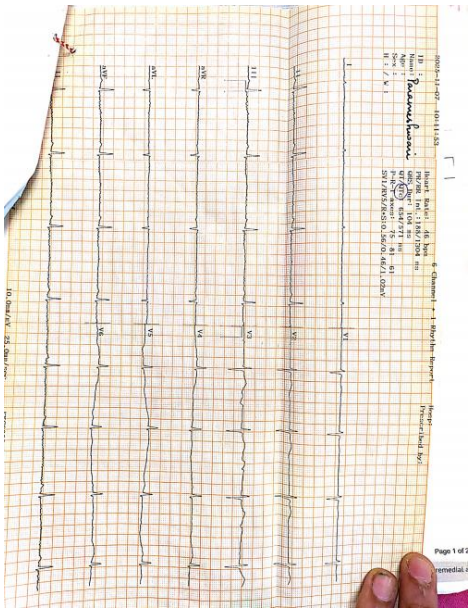
INVESTIGATIONS

Category	Parameter	Value(s) / Trend	Date / Context
Hematology	**Hemoglobin (Hb)**	**7.7 g/dL**	12/11/2025 (Admission)
		7.1 g/dL	Nadir recorded
	Total WBC Count	**1,900 cells/mm³** (Leukopenia)	12/11/2025
	Platelets	**56,000 /mm³**	12/11/2025
		24,000 /mm³	Manual count (Nadir)
	Peripheral Smear	Normocytic normochromic anemia, relative lymphocytosis, thrombocytopenia	09/11/2025
	Bone Marrow	Normocellular, Erythroid Hyperplasia, No atypical cells	Aspiration Report
Metabolic & Glucose	**Random Blood Sugar**	**32.3 mg/dL**	15/11/2025 (Severe Hypoglycemia)
		25 mg/dL	Previous record (04/10)
		60 mg/dL	07/11/2025
Electrolytes	**Potassium (K+)**	**2.4 mmol/L**	12/11/2025 (Severe Hypokalemia)
		2.9 mmol/L	15/11/2025
	Sodium (Na+)	**124 mEq/L**	15/11/2025 (Hyponatremia)
	Calcium (Ca)	**7.5 mg/dL**	09/11/2025 (Hypocalcemia)
Renal Function	**Urea**	32.3 mg/dL	15/11/2025
		35 mg/dL	07/11/2025
	Creatinine	1.0 mg/dL	15/11/2025
		0.7 mg/dL	07/11/2025
Liver Function	**Albumin**	**2.7 g/dL**	15/11/2025 (Hypoalbuminemia)
	Total Bilirubin	1.32 mg/dL	15/11/2025
	SGOT (AST)	28 U/L	08/11/2025
	SGPT (ALT)	23 U/L	08/11/2025
Arterial Blood Gas	**pH**	7.44	16/11/2025
(ABG)	**pCO2**	34 mmHg	16/11/2025
	HCO3	23 mmol/L	16/11/2025
	Lactate	1.0 mmol/L	16/11/2025
Endocrinology	**Serum Cortisol**	**32.41 µg/dL**	Morning sample (Normal/High stress)
	Thyroid Profile	FT3: 80, FT4: 8.2, TSH: 2.8	Previous admission (04/10)

Urinalysis	**Protein**	Trace	15/11/2025
	WBC / Pus Cells	4–6 /hpf	15/11/2025
	Specific Gravity	1.005	15/11/2025
	Ketones	Negative	15/11/2025

Diagnostic imaging was unremarkable; chest radiography showed normal lung fields, and abdominal ultrasonography revealed bilateral kidneys with Grade 2 echoes but no free fluid or organomegaly. Upper GI Endoscopy (OGD): The study revealed **corporal and fundal gastritis**. No ulceration, stricture, or growth was noted. Colonoscopy: The study was normal. However, the electrocardiogram demonstrated critical abnormalities, specifically **sinus bradycardia at 46 bpm and a prolonged QTc interval of 571 ms.** Infectious etiology was ruled out with negative urine cultures and negative sputum CBNAAT for tuberculosis. Additionally, the lipid profile reflected a hypometabolic state with **low total cholesterol (112 mg/dL) and triglycerides (38 mg/dL).**

ECG



CHEST XRAY

DIAGNOSTIC CRITERIA

Criterion	Patient Clinical Findings
A. Restriction of energy intake leading to significantly low body weight relative to age, sex, and physical health.	• **Severe Emaciation:** Described as "severely emaciated" with "generalized muscle wasting" • **Fat Loss:** Specific "loss of buccal pad of fat" and "temporal/periorbital fat wasting" • **Weight Loss:** Documented "progressive weight loss" over 2

	weeks prior to admission.
B. Intense fear of gaining weight or persistent behavior that interferes with weight gain.	• **Purging Behavior:** Admitted "chronic laxative abuse" (Bisacodyl) for 2 years • **Dietary Restriction:** Documented "restrictive intake" and "regurgitation of feeds" • **Preoccupation:** Notes indicate "marked preoccupation with weight".
C. Disturbance in self-perception or persistent lack of recognition of the seriousness of current low body weight.	• **Psychiatric Context:** "Psychiatry opinion was obtained to evaluate for anorexia" after organic causes were ruled out. • **Lack of Organic Cause:** Extensive workup (OGD, Colonoscopy, CT) found "no organic cause" for the profound cachexia.
D. Physiological Severity Specifiers (Harrison's / Medical Complications)	• **Hemodynamic Instability:** Severe **sinus bradycardia** (46 bpm) and **hypotension** (90/60 mmHg). • **Metabolic Collapse:** Critical **hypoglycemia** (25 mg/dL) and **hypokalemia** (2.4 mmol/L). • **Hematologic Suppression: Pancytopenia** (Hb 7.1 g/dL, Platelets 24,000/mm³) consistent with "starvation marrow".

Differential Diagnoses

1. Malignancy Recurrence (Metastatic Osteosarcoma)

- **Suspicion:** Given the patient's history of osteosarcoma (treated in 2018), the presentation of severe cachexia, weight loss, and pancytopenia (anemia and thrombocytopenia) strongly raised the suspicion of bone marrow infiltration or metastatic disease.
- **Discussion:** This was ruled out by a bone marrow aspiration study, which showed normocellular marrow with erythroid hyperplasia and **no atypical or malignant cells**. Additionally, chest X-ray and abdominal ultrasound did not show evidence of metastasis or organomegaly.

2. Adrenal Insufficiency (Addison's Disease)

- **Suspicion:** The patient presented with classic metabolic signs of adrenal failure, including hyponatremia (124 mEq/L), hypoglycemia (25 mg/dL), and hypotension (90/60 mmHg).
- **Discussion:** The medical team listed "Secondary Adrenal Insufficiency" as a differential. However, morning serum cortisol levels were found to be **32.41 µg/dL** (normal range), effectively ruling out adrenal failure.

3. Chronic Infection (Tuberculosis / HIV)

- **Suspicion:** Severe weight loss, cachexia, and leukopenia in a tropical setting often point towards chronic infections like disseminated tuberculosis or HIV.
- **Discussion:** Infectious etiology was investigated and excluded. Sputum AFB and CBNAAT were **negative** for tuberculosis. Serology for HIV, HBsAg, and HCV was also **negative**.

4. Gastrointestinal Malabsorption / Organic Bowel Disease

- **Suspicion:** The patient had a 2-year history of constipation, abdominal pain, and severe malnutrition, raising concern for structural GI pathology causing malabsorption.
- **Discussion:** Endoscopic evaluation was performed to rule this out. OGD (Upper GI endoscopy) showed only gastritis, and a **Colonoscopy was normal**. No organic cause for the weight loss was identified in the GI tract.

5. Anorexia Nervosa (Purging Type)

- **Suspicion:** The combination of "restrictive intake," "chronic laxative abuse," and "marked preoccupation with weight" in a young female pointed toward an eating disorder.
- **Discussion:** This was the **final diagnosis of exclusion**. The clinical picture of "starvation marrow" (pancytopenia), metabolic collapse (hypoglycemia, hypokalemia), and sinus bradycardia was attributed to severe physiological adaptation to starvation and purging behaviours.

TREATMENT

The patient was managed with a multidisciplinary approach focusing on acute stabilization and rigorous nutritional rehabilitation. Initial treatment involved intravenous fluid resuscitation and aggressive correction of electrolyte imbalances, specifically targeting severe hypokalemia and hypocalcemia, alongside replacement of magnesium, phosphate, and zinc to mitigate the risk of refeeding syndrome and address severe malnutrition. A supervised protein-rich diet with macronutrient replacement was initiated, supplemented by prophylactic thiamine (100 mg) to prevent Wernicke's encephalopathy. Pharmacotherapy included broad-spectrum antibiotics (Ceftriaxone, Metronidazole), acid suppression (Omeprazole, Ranitidine), and antispasmodics (Dicyclomine) for symptomatic relief, while anxiety was managed with low-dose Clonazepam. Following specialist consultations with Surgery, Orthopaedics, and Psychiatry to exclude malignancy recurrence and organic pathology, she was discharged on a maintenance regimen of oral hematinics (Ferrous

Sulfate, Folic Acid), calcium supplementation, and strict dietary instructions.

PROGNOSIS

The prognosis for this patient is favorable regarding her oncologic history, as extensive evaluation including bone marrow aspiration and orthopedic review confirmed no recurrence of osteosarcoma. Her immediate physiological prognosis is also good; the life-threatening metabolic and hematological complications ("starvation marrow," hypoglycemia, and bradycardia) showed signs of reversibility with acute nutritional stabilization and electrolyte correction. However, the long-term prognosis remains guarded and is contingent upon strict adherence to psychiatric and nutritional rehabilitation. Given the chronicity of her symptoms (2 years of constipation/laxative abuse) and the severity of her initial presentation (BMI < 15 equivalent, QTc prolongation), she remains at high risk for relapse of Anorexia Nervosa. Sustained recovery will require multidisciplinary outpatient management to prevent future metabolic collapse and to address the underlying behavioural pathology.

DISCUSSION

The presentation of severe cachexia and weight loss in a cancer survivor invariably raises the specter of malignancy recurrence. In this case, the patient's history of osteosarcoma (treated in 2018) combined with profound emaciation and pancytopenia created a clinical picture strongly suggestive of metastatic marrow infiltration. However, this case illustrates the critical importance of recognizing Anorexia Nervosa (AN) as a "great mimicker" in oncologic patients. The absence of malignant cells on bone marrow aspiration and the lack of structural lesions on imaging shifted the diagnosis from oncologic progression to severe physiological starvation. A pivotal feature of this case was severe pancytopenia (Hb 7.1 g/dL, Platelets 24,000/mm³, WBC 1,900/mm³). In the context of Anorexia Nervosa, this phenomenon is often due to Gelatinous Marrow Transformation (GMT) (also known as serous atrophy of fat). GMT is a specific histological pattern where hematopoietic cells are replaced by an extracellular matrix of hyaluronic acid due to the depletion of body fat stores. While our patient's marrow showed erythroid hyperplasia rather than frank aplasia, the peripheral pancytopenia reflects the marrow's inability to sustain hematopoiesis under severe caloric deprivation. Crucially, unlike metastatic bone disease, these hematological abnormalities are reversible with nutritional rehabilitation. The patient exhibited classic cardiovascular adaptations to starvation, specifically profound sinus bradycardia (46 bpm) and hypotension (90/60 mmHg). In AN, this is a physiological downregulation mediated by increased vagal tone to conserve energy. Of greater concern was the marked QTc prolongation (654/571 ms). This repolarization abnormality is a known complication of AN, exacerbated by electrolyte derangements—specifically the hypokalemia (2.4 mmol/L) driven by her admitted laxative abuse. This triad of bradycardia, QTc prolongation, and electrolyte depletion places such patients at imminent risk of sudden cardiac death from Torsades de Pointes. The patient's use of bisacodyl for two years contributed significantly to her metabolic collapse. Purging behaviors in AN often lead to a hypokalemic, hypochloremic metabolic alkalosis. In this patient, the "metabolic mayhem" was further compounded by profound hypoglycemia (25 mg/dL), a direct consequence of glycogen store depletion and impaired gluconeogenesis typical of the late stages of starvation. The coexistence of hypoglycemia and cardiac instability necessitated aggressive but careful correction to avoid Refeeding Syndrome, a potentially fatal shift in fluids and electrolytes (particularly phosphate and magnesium) that can occur when nutrition is reintroduced to a starved patient.

CONCLUSION

This case serves as a poignant reminder of the diagnostic challenges involved in managing cancer survivors presenting with severe weight loss and systemic collapse. While the immediate clinical instinct is to investigate for malignancy recurrence, particularly given the hematological suppression and profound emaciation observed here, clinicians must remain vigilant for psychiatric and behavioral etiologies such as Anorexia Nervosa and surreptitious laxative abuse. The "metabolic mayhem" observed—characterized by critical hypoglycemia, severe electrolyte derangements, and life-threatening QTc prolongation—represents a state of physiological failure that closely mimics terminal organic disease. The presence of pancytopenia in this context highlights the importance of the "starvation marrow" concept, where hematopoietic failure is a reversible consequence of extreme caloric deprivation rather than irreversible malignant infiltration. This patient's recovery was predicated on a multidisciplinary stabilization strategy involving aggressive electrolyte correction and supervised nutritional rehabilitation. Ultimately, the long-term prognosis for such patients depends on the transition from acute medical stabilization to sustained psychiatric intervention. By recognizing that the devastating systemic signs of starvation are potentially reversible, medical teams can avoid the pitfalls of misattributing symptoms to a terminal prognosis, thereby offering patients a path toward both physiological and psychological recovery.

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