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Occult Metastatic Prostate Carcinoma Presenting With Decompensated Heart Failure and Bicytopenia

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Abstract

Decompensated heart failure (DHF) may occasionally conceal an underlying systemic malignancy. Persistent hematological abnormalities, particularly thrombocytopenia and markedly elevated alkaline phosphatase (ALP), extending beyond the acute phase of illness warrant systematic evaluation for bone marrow pathology or infiltrative disease, irrespective of the presenting diagnosis.

A 53-year-old man with no prior comorbidities presented with DHF, bicytopenia (anemia and thrombocytopenia), and markedly elevated ALP. He developed disseminated intravascular coagulation (DIC) during hospitalization, managed with fresh frozen plasma (FFP), platelet concentrates, and cryoprecipitate, with subsequent resolution; however, thrombocytopenia and ALP elevation persisted. Bone marrow examination revealed infiltration by metastatic adenocarcinoma. Despite comprehensive evaluation including gastrointestinal endoscopies, contrast-enhanced computed tomography (CT), and 18-fluorodeoxyglucose positron emission tomography-CT (18-FDG PET-CT), the primary site remained elusive in the setting of multiple elevated tumor markers: prostate-specific antigen (PSA), carcinoembryonic antigen (CEA), and carbohydrate antigen (CA) 19-9. Immunohistochemistry of the bone marrow biopsy demonstrated positivity for cytokeratin (CK) and NKX3.1, establishing prostatic origin. Subsequent prostate biopsy confirmed left-lobe prostatic adenocarcinoma with a Gleason score of 4+3=7 (Grade Group 3), tumor comprising 70% of the biopsy core, and perineural invasion, consistent with high-risk locally advanced disease. The patient was commenced on androgen deprivation therapy (ADT).

Persistent thrombocytopenia and markedly elevated ALP may represent the sole initial manifestation of occult metastatic prostate cancer, even in young patients with no urological symptoms. Immunohistochemistry, particularly NKX3.1, is indispensable in establishing prostatic origin when the primary site remains radiologically occult after comprehensive investigation.

Categories: Hematology, Internal Medicine, Oncology
Keywords: acute decompensated heart failure, alkaline phosphatase, bone marrow metastasis, disseminated intravascular coagulation, myelophthisis, nkx3.1, prostate adenocarcinoma, thrombocytopenia

Introduction

Heart failure represents one of the most prevalent and burdensome cardiovascular conditions worldwide, accounting for millions of hospitalizations annually and carrying significant morbidity and mortality across all age groups. Decompensated heart failure is characterized by a rapid onset or worsening of symptoms and signs of heart failure, frequently necessitating intensive care-level management with hemodynamic support, intravenous diuretics, and close monitoring of end-organ function [1]. In the acute setting, systemic manifestations of decompensated heart failure, including renal impairment, hepatic congestion, anemia, and thrombocytopenia, are routinely attributed to hemodynamic compromise and are expected to normalize following the restoration of adequate cardiac output and decongestion. As a result, hematological abnormalities in the context of decompensated heart failure are rarely investigated beyond the acute episode.

Thrombocytopenia is encountered frequently in critically ill patients, with an estimated incidence of 30-50% across intensive care unit populations, arising from a complex interplay of consumptive, hemodilution, drug-induced, and inflammatory mechanisms [2]. In the vast majority of cases, platelet counts recover in tandem with clinical improvement [2]. However, persistence or worsening of thrombocytopenia beyond the resolution of the acute illness constitutes a clinically significant finding that warrants systematic evaluation. Bone marrow infiltration by metastatic malignancy, myelophthisis, is among the most important and underrecognized causes of persistent cytopenias in hospitalized patients and may present without any symptoms referable to the primary tumor site.

Prostate cancer is the second most common malignancy diagnosed in men globally and the leading cause of cancer-related mortality in men in many regions. While the classical clinical presentation involves elderly

men with lower urinary tract symptoms (LUTS), elevated prostate-specific antigen (PSA), and bone pain from skeletal metastases, a subset of patients presents with distinctly atypical features. Prostate cancer has a well-established predilection for osteoblastic bone metastases, present in up to 90% of patients with advanced disease at autopsy. Skeletal involvement drives markedly elevated alkaline phosphatase (ALP) through osteoblastic activity and, in advanced cases, may infiltrate the bone marrow, causing myelophthisic cytopenias and, rarely, disseminated intravascular coagulation (DIC) [3,4]. Young-onset prostate cancer, defined as diagnosis at or before 55 years of age, accounts for fewer than 5% of all cases and is enriched for higher-grade pathology and more aggressive biological behavior at presentation, as detailed further in the Discussion. In these patients, the absence of urological symptoms and the atypical age of onset make prostate cancer an unlikely diagnostic consideration, increasing the risk of diagnostic delay.

The coexistence of decompensated heart failure, bicytopenia, markedly elevated ALP, and DIC as the initial clinical phenotype of occult metastatic prostate cancer in a young patient without LUTS represents a diagnostic scenario that is exceptionally uncommon and carries substantial potential for misattribution of findings to the presenting cardiac illness. When the primary tumor site remains elusive despite comprehensive radiological investigation, immunohistochemistry, particularly NKX3.1, a prostate-specific homeobox transcription factor with sensitivity approaching 100% in metastatic prostatic adenocarcinoma, plays an indispensable role in establishing the diagnosis and directing targeted biopsy [5].

We present the case of a 53-year-old man with no prior medical history who presented with decompensated heart failure, bicytopenia, and markedly elevated ALP, in whom a comprehensive and stepwise diagnostic evaluation ultimately revealed occult high-risk metastatic prostate cancer. This case underscores the imperative of pursuing persistent hematological abnormalities beyond the index diagnosis, demonstrates the critical diagnostic utility of NKX3.1 immunohistochemistry in carcinoma of unknown primary, and highlights the clinical profile of young-onset metastatic prostate cancer presenting without classical urological features. This case is reported in accordance with the CARE (CAse REport) guidelines [6].

Case Presentation

A 53-year-old man with no prior medical history, no known comorbidities, or prescribed medications presented with bilateral lower limb swelling of two months duration, progressively worsening to include abdominal distension and periorbital puffiness. He reported breathlessness for 15 days with orthopnea and paroxysmal nocturnal dyspnea. He had a significant history of chronic alcohol consumption spanning more than 20 years. There was no family history of malignancy. Critically, the patient explicitly denied any LUTS, including difficulty initiating micturition, urinary frequency, nocturia, or hematuria, and reported no bone pain, weight loss, or constitutional symptoms.

On examination, the patient was hemodynamically compromised (blood pressure 86/56 mmHg, heart rate 110/min, respiratory rate 24/min) and markedly pale with icteric sclerae, bilateral pitting pedal edema extending to the thighs, periorbital puffiness, and elevated jugular venous pressure. Bilateral basal crepitations were noted on auscultation. Abdominal examination revealed mild hepatosplenomegaly and shifting dullness.

A diagnosis of decompensated heart failure was established. The patient was admitted to the intensive care unit and managed with inotropic support, intravenous diuretics (furosemide), and fluid restriction. Echocardiography revealed dilation of the right atrium, right ventricle, and left ventricle with globally hypokinetic ventricular walls and an ejection fraction of 41%; no significant valvular pathology was noted, findings provisionally attributed to alcoholic cardiomyopathy.

Initial investigations revealed severe normocytic anemia (hemoglobin 4.15 g/dL), severe thrombocytopenia (platelet count $12\times10^3/\mu L$), and a total leukocyte count (TLC) of $10,860/mm^3$. Serum creatinine was 1.38 mg/dL, and ALP was 453 IU/L. Serial hematological and biochemical parameters are summarized in Table *1*.

Timeline	Hb (g/dL)	TLC (/mm³)	Platelets (×10³/μL)	Urea (mg/dL)	Creatinine (mg/dL)	ALP (IU/L)
Day 1	4.15	10,860	12	56	1.38	453
Day 4	7.4	5,470	20	132	1.51	987
Day 25	7.9	7,530	24	26.3	0.80	1,980
Day 28	8.9	8,660	108	-	-	3,357

TABLE 1: Serial hematological and biochemical parameters during hospitalization

Hb: hemoglobin; TLC: total leukocyte count; ALP: alkaline phosphatase

During hospitalization, thrombocytopenia and anemia worsened progressively. The patient developed new purpuric lesions, ecchymoses, ischemic changes of the bilateral toes, and jaundice. He fulfilled the International Society on Thrombosis and Haemostasis (ISTH) criteria for overt DIC, with D-dimer 7.36 μg/mL and fibrinogen 49 mg/dL [7,8]. DIC was managed with fresh frozen plasma (FFP), platelet concentrate transfusions, and cryoprecipitate to replenish fibrinogen, with the subsequent resolution of coagulopathy and hemodynamic stabilization.

Despite DIC resolution, thrombocytopenia and anemia failed to recover to normal levels. Although a partial improvement in platelet count was observed by day 28, this remained below the normal range, and ALP continued to rise precipitously over four weeks, reaching levels exceeding 3000 IU/L (Table *1*). Contrast-enhanced computed tomography (CT) of the abdomen and pelvis revealed splenomegaly.
This disproportionate ALP elevation occurring without biliary pathology on imaging was notable. Combined with the incomplete recovery of bicytopenia, it raised a strong clinical suspicion for osteoblastic skeletal metastases with bone marrow infiltration.

Bone marrow examination was subsequently performed and revealed diffuse replacement of normal hematopoietic elements by sheets of metastatic adenocarcinoma.

A comprehensive search for the primary malignancy was undertaken. Serum tumor markers returned markedly elevated across multiple lineages (Table *2*), PSA at 135 ng/mL, CEA at 14.02 ng/mL, and CA 19-9 at 67.5 U/mL, creating a diagnostic dilemma. Upper gastrointestinal endoscopy and colonoscopy were unremarkable. 18-Fluorodeoxyglucose positron emission tomography-CT (18-FDG PET-CT) demonstrated diffuse sclerotic skeletal uptake involving the axial and appendicular skeleton consistent with widespread osteoblastic metastases and bilateral axillary lymph node uptake, but failed to identify a definitive primary lesion.

Tumor marker	Value	Reference range
PSA	135 ng/mL	<4 ng/mL
CEA	14.02 ng/mL	<5 ng/mL
CA 19-9	67.5 U/mL	<37 U/mL
AFP	6.32 ng/mL	<7 ng/mL
CA 72-4	1.34 U/mL	<6.9 U/mL

TABLE 2: Serum tumor markers at the time of diagnosis

PSA: prostate-specific antigen; CEA: carcinoembryonic antigen; CA: carbohydrate antigen; AFP: alpha-fetoprotein

Immunohistochemistry of the bone marrow biopsy specimen demonstrated strong positivity for CK and nuclear positivity for NKX3.1 (Figure *1*), a prostate-specific homeobox transcription factor with high sensitivity and specificity for prostatic origin [9-11]. This directed targeted transrectal ultrasound-guided prostate biopsy, which confirmed prostatic adenocarcinoma involving the left lobe (Figure *2*) with a Gleason score of 4+3=7 (Grade Group 3), tumor comprising 70% of the biopsy core, and perineural invasion (Figure *3*), consistent with high-risk locally advanced disease.

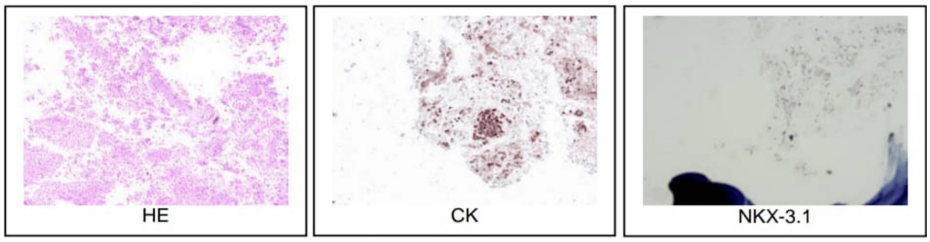


FIGURE 1: Immunohistochemistry panel of the bone marrow biopsy (×200). (A) H&E staining demonstrating the replacement of normal hematopoietic marrow by sheets of metastatic adenocarcinoma. (B) CK immunostaining showing diffuse positivity in tumor cells, confirming epithelial origin. (C) NKX3.1 immunostaining demonstrating strong nuclear positivity, establishing prostatic differentiation

H&E: hematoxylin and eosin; CK: cytokeratin

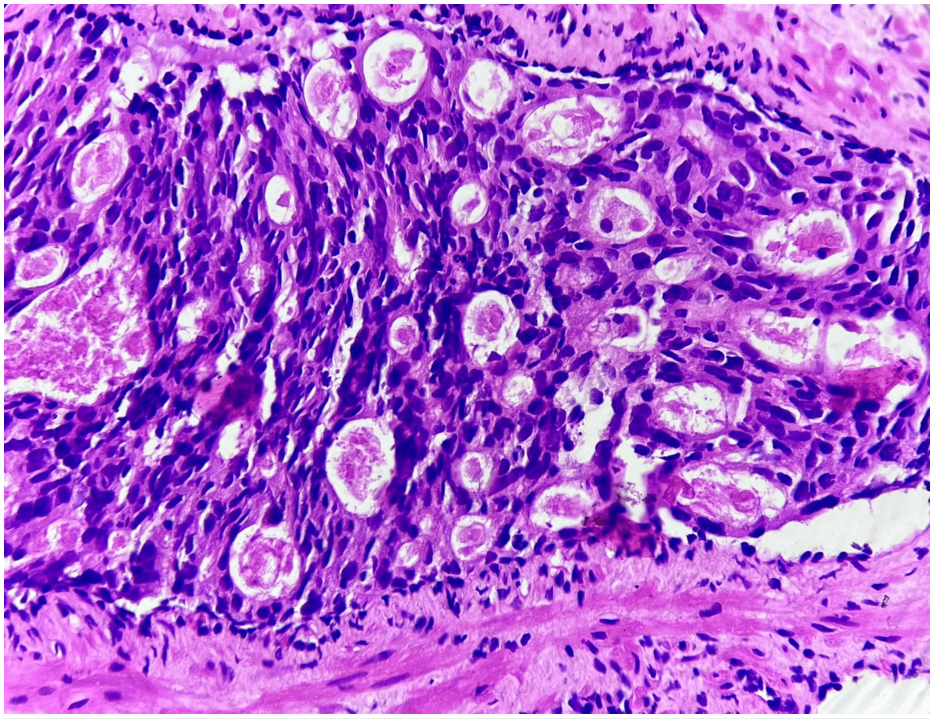


FIGURE 2: H&E staining of the prostate core biopsy, medium-power view (×100), demonstrating crowded irregular malignant glands with intraluminal crystalloids and prominent nucleoli, consistent with prostatic adenocarcinoma

H&E: hematoxylin and eosin

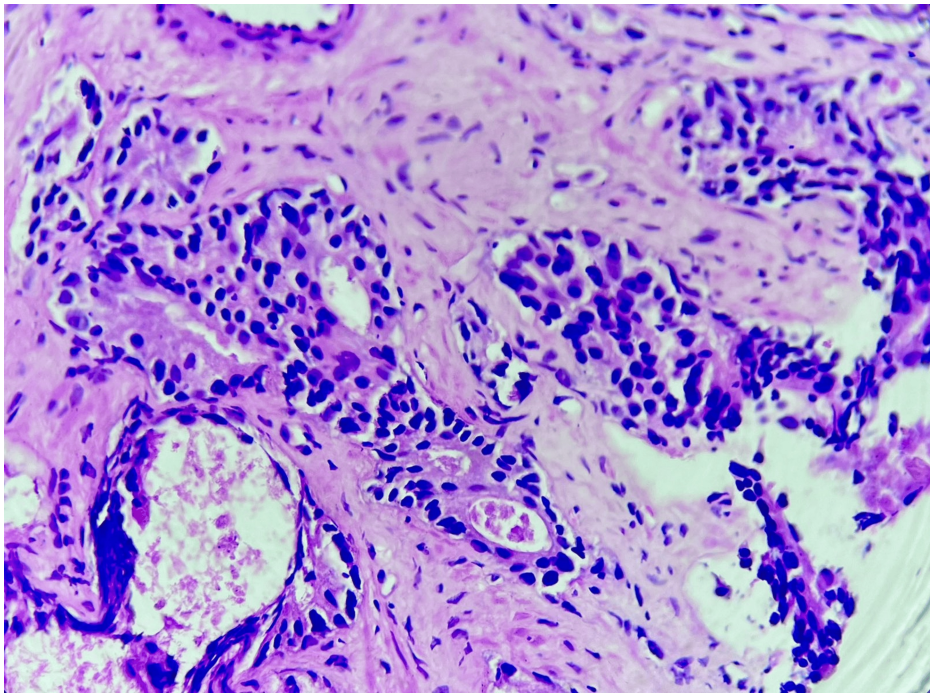


FIGURE 3: H&E staining, high-power view (×400), showing infiltrating malignant cells with nuclear enlargement, prominent nucleoli, and perineural invasion within fibromuscular stroma

Gleason score 4+3=7; Grade Group 3; tumor in 70% of biopsy core

H&E: hematoxylin and eosin

The patient was commenced on androgen deprivation therapy (ADT) with a GnRH antagonist in combination with an anti-androgen. Given the extensive skeletal metastatic burden satisfying CHAARTED criteria for high-volume metastatic hormone-sensitive prostate cancer (mHSPC), treatment intensification was discussed in a multidisciplinary tumor board meeting, in accordance with current evidence-based guidelines.

Unfortunately, the patient was subsequently lost to follow-up, did not follow the advised chemotherapy regimen, and also did not attend scheduled review appointments. Approximately four weeks following discharge, the patient was declared deceased. Formal post-discharge biochemical data, including PSA, platelet count, and ALP trends, were therefore unavailable. The rapid clinical deterioration observed in this case is consistent with the aggressive biological behavior associated with high-volume metastatic prostate cancer and extensive bone marrow infiltration, further underscoring the prognostic severity of this presentation.

Figure *4* depicts the diffuse skeletal uptake in a PET-CT scan.



FIGURE 4: Diffuse skeletal uptake in a PET-CT scan

PET-CT: positron emission tomography-computed tomography

Discussion

This case presents an instructive diagnostic scenario in which a 53-year-old man presenting with decompensated heart failure was ultimately diagnosed with high-risk metastatic prostate cancer through the systematic pursuit of two persistent laboratory abnormalities: thrombocytopenia that failed to recover following DIC resolution and ALP that rose precipitously despite hemodynamic stabilization. The case merits discussion across five dimensions: the initial diagnostic reasoning, the role of DIC in occult malignancy, the significance of elevated ALP and myelophthisis, the diagnostic challenge of multiple tumor markers, and the therapeutic implications of high-volume mHSPC.

The initial presentation of decompensated heart failure in the setting of chronic alcohol use appropriately

directed clinical attention toward alcoholic cardiomyopathy with hepatic dysfunction. Notably, this cardiac framework also provided a plausible physiological explanation for the accompanying cytopenias, since hepatic congestion, splenic sequestration from portal hypertension, and reduced hepatic synthetic function are all recognized causes of thrombocytopenia and anemia in decompensated heart failure. This overlap is precisely what rendered the underlying marrow pathology occult: the cardiac diagnosis was not merely a coincidental first impression but an actively plausible unifying explanation for the hematological findings, delaying recognition of an infiltrative process until the cytopenias failed to track with cardiac recompensation. Thrombocytopenia in this context is readily attributable to alcohol-related myelosuppression, hypersplenism, or nutritional deficiency [1]. The fundamental clinical principle illustrated here is that thrombocytopenia persisting and worsening after the resolution of all plausible attributable causes demands a paradigm shift in diagnostic reasoning. The 2021 scoping review by Jonsson et al. demonstrated that incident thrombocytopenia during critical illness is associated with significantly elevated mortality, an observation that must trigger active investigation rather than passive reassurance [2].

The development of DIC during hospitalization added both morbidity and diagnostic complexity. While DIC in critically ill patients is classically attributed to sepsis, organ dysfunction, and consumptive processes, malignancy-associated DIC, particularly from mucin-secreting adenocarcinomas, is well established in the literature. In retrospect, the malignancy-associated DIC in this case was likely present from the outset, sustained by tumor-derived tissue factor expression and microangiopathic changes secondary to extensive marrow infiltration. Critically, the resolution of DIC parameters following supportive therapy while thrombocytopenia persisted constituted a characteristic and diagnostically important dissociation, pointing away from a single consumptive etiology and toward structural marrow pathology. This pattern should consistently prompt bone marrow evaluation per current DIC guidelines [7,8].

ALP exceeding 3000 IU/L in the absence of hepatobiliary obstruction on imaging is virtually diagnostic of osteoblastic skeletal metastases and should be regarded as an oncological emergency necessitating urgent evaluation. Osteoblastic metastasis in prostate cancer is driven by the tumor-induced stimulation of osteoblastic activity through endothelin-1, bone morphogenetic proteins, and Wnt signaling pathway activators, resulting in marked ALP elevation [3,4]. In the landmark autopsy series by Bubendorf et al. of 1589 patients with advanced prostate cancer, skeletal metastases were present in 90% of cases, confirming bone as the overwhelmingly dominant site of distant spread [5]. Bone marrow infiltration by metastatic adenocarcinoma, myelophthisis, results in cytopenias through the mechanical displacement of hematopoietic precursors and the disruption of the marrow microenvironment. Shahait et al. demonstrated that only 6% of patients with newly diagnosed metastatic castration-sensitive prostate cancer have biopsy-proven bone marrow involvement at diagnosis and that elevated ALP and anemia are the key clinical features associated with this finding, rendering this initial presentation distinctly uncommon [6].

The co-elevation of PSA, CEA, and CA 19-9 presented a formidable diagnostic challenge. While PSA at 135 ng/mL with diffuse osteoblastic skeletal lesions on PET-CT is, in retrospect, highly characteristic of metastatic prostate cancer, the concurrent elevation of gastrointestinal tumor markers appropriately mandated the exclusion of pancreatic, biliary, colorectal, and gastric primaries. Non-specific tumor marker elevation in advanced malignancy arises through tumor dedifferentiation, aberrant antigen expression, and cross-reactivity. This case reinforces that immunohistochemistry is the gold standard in carcinoma of unknown primary and should be initiated early, particularly when NKX3.1 is clinically suspected, rather than after exhausting often unrevealing radiological investigation [10,11].

NKX3.1 is a prostate-specific homeobox transcription factor encoded on chromosome 8p21.2, whose expression is driven by androgen receptor signaling and maintained in metastatic lesions, including post-castration specimens, with sensitivity approaching 100% and near-absolute specificity for prostatic origin as demonstrated in head-to-head comparisons with PSA, PSAP, and HOXB13 in metastatic tumor cohorts [9-11]. Its nuclear positivity in the bone marrow biopsy was the single most consequential investigation, redirecting the diagnostic pathway toward the prostate and enabling targeted biopsy. This underscores that in any bone marrow biopsy showing metastatic adenocarcinoma with elevated PSA, NKX3.1 should be reflexively incorporated in the immunohistochemistry panel regardless of the concurrent elevation of other tumor markers.

The histopathological findings of Gleason score 4+3=7 (Grade Group 3), 70% tumor core involvement, and perineural invasion confirm a high-risk, biologically aggressive phenotype. The extensive skeletal metastatic burden satisfies the CHAARTED definition of high-volume mHSPC, four or more bone metastases with at least one beyond the axial skeleton. Evidence from two landmark randomized controlled trials mandates treatment intensification beyond ADT alone in this setting: the CHAARTED trial demonstrated a significant overall survival benefit with the addition of docetaxel to ADT (median OS: 57.6 vs. 44 months; p=0.0006), while the LATITUDE trial demonstrated a comparable survival advantage with abiraterone acetate plus prednisone in high-risk mHSPC. Multidisciplinary evaluation and patient counseling regarding treatment intensification, as performed in this case, are therefore essential per current European Association of Urology (EAU) 2024 and National Comprehensive Cancer Network (NCCN) guidelines [12-15].

Several features of this case are educationally important in characterizing the atypical spectrum of prostate cancer presentation [16,17]. The patient was 53 years of age, considerably younger than the global median

age at diagnosis of 66 years. Young-onset prostate cancer (diagnosed at or before age 55) accounts for fewer than 5% of all cases and, as demonstrated in the systematic review and meta-analysis by Zheng et al. [18], is enriched for higher Gleason grade at diagnosis and more aggressive pathological features, including higher rates of positive surgical margins and seminal vesicle invasion. Germline genetic testing for BRCA1/2 mutations and DNA damage repair pathway defects is therefore recommended in this patient, given the young age of diagnosis and extent of disease, as these carry prognostic significance and may influence eligibility for targeted therapy with PARP inhibitors [12,13]. The complete absence of LUTS, bone pain, or constitutional symptoms further underscores that prostate cancer should not be excluded in young men presenting with hematological failure when biochemical clues, particularly a disproportionately elevated ALP, are present.

The fatal outcome in this case, occurring within four weeks of treatment initiation, reflects the advanced disease burden at presentation and highlights that even prompt initiation of guideline-concordant therapy may be insufficient to alter the trajectory of high-volume metastatic prostate cancer complicated by extensive myelophthisis and DIC.

Conclusions

Persistence of thrombocytopenia and markedly elevated ALP beyond the resolution of the acute presenting illness must not be attributed to the index diagnosis without systematic evaluation for bone marrow pathology. This case demonstrates that occult high-risk metastatic prostate cancer may present exclusively as hematological failure, bicytopenia, DIC, and markedly elevated ALP, in young patients with no urological symptoms, rendering the diagnosis non-intuitive and susceptible to delay. When standard radiological investigation fails to identify the primary site in the setting of metastatic adenocarcinoma, NKX3.1 immunohistochemistry is an indispensable tool for establishing prostatic origin. Early recognition of hematological red flags, timely bone marrow biopsy, judicious use of immunohistochemistry, and multidisciplinary management with treatment intensification per current guidelines are the pillars of appropriate care in such presentations.

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: Srinidhi Rao V R, Ramangouda Malipatil

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Drafting of the manuscript: Srinidhi Rao V R, Ramangouda Malipatil

Critical review of the manuscript for important intellectual content: Ravi Krishnegowda

Supervision: Ravi Krishnegowda

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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