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Beyond Heart Failure: Unmasking Metastatic Prostate Adenocarcinoma Through Marked Alkaline Phosphatase Elevation

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Abstract

An 85-year-old man presented with progressive dyspnea and chest tightness after several months of worsening lower back pain. Initial evaluation showed hypoxemia, peripheral edema, an elevated pro-B-type natriuretic peptide level, cardiomegaly, and small bilateral pleural effusions; CT pulmonary angiography showed no pulmonary embolism. He was treated for presumed acute heart failure, but his dyspnea improved only mildly over the first hospital days. Further evaluation demonstrated a markedly elevated alkaline phosphatase (ALP) level of 2,152 U/L and a prostate-specific antigen level reported as 725 ng/mL. The CT showed diffuse skeletal sclerosis, and PET imaging demonstrated extensive osseous lesions with pelvic and retroperitoneal lymphadenopathy. The CT-guided biopsy of a left iliac bone lesion showed adenocarcinoma consistent with a prostatic primary. This case highlights the importance of reassessing an initial working diagnosis when the clinical course is incompletely explained and considering an underlying malignancy when progressive skeletal pain, marked ALP elevation, and sclerotic bone lesions occur together.

Categories: Oncology, Internal Medicine
Keywords: alkaline phosphatase, bone metastases, case report, diagnostic reassessment, prostate adenocarcinoma, prostate-specific antigen

Introduction

Prostate cancer is one of the most commonly diagnosed malignancies in men and predominantly affects older adults, with its incidence increasing with age [1]. Although many patients initially present with localized disease, advanced prostate cancer may spread to regional lymph nodes and distant organs, particularly the bones. Bone is the most common site of distant metastasis, and prostate cancer bone metastases frequently demonstrate an osteoblastic or osteosclerotic pattern on imaging [1,2]. These lesions may cause persistent skeletal pain, impaired mobility, and substantial morbidity.

Laboratory findings can provide important clues to advanced disease. Prostate-specific antigen (PSA) is an established marker used in the evaluation of prostate cancer, and markedly elevated levels may raise suspicion for advanced disease [3]. Alkaline phosphatase (ALP), an enzyme associated with osteoblastic activity, may also be substantially elevated in patients with extensive skeletal involvement. However, ALP is not specific to prostate cancer, and its elevation should be interpreted alongside clinical findings, imaging, and histopathological results [1].

We report the case of an older male admitted with progressive dyspnea and initially treated for presumed acute heart failure in whom several months of worsening back pain, a markedly elevated ALP level, and subsequent imaging and biopsy findings led to the diagnosis of metastatic prostate adenocarcinoma. This case highlights the importance of reassessing the diagnosis when the clinical course is not fully explained by the initial working diagnosis and emphasizes the diagnostic significance of unexplained biochemical abnormalities in patients with progressive skeletal symptoms. The available information does not establish a direct relationship between the malignancy and the initial cardiopulmonary presentation.

Case Presentation

An 85-year-old male presented to the emergency department with progressive shortness of breath and chest tightness. Approximately one week before presentation, he developed chest tightness and dyspnea while performing exercises during physical therapy for sciatica. Symptoms progressed and were present at rest, prompting his wife to encourage medical evaluation. He also described several months of worsening lower back pain and chronic lower-extremity pain, greater on the right than the left.

His medical history included atrial fibrillation, hypertension, gastroesophageal reflux disease, and sciatica. Medications listed in the source presentation were apixaban, gabapentin, and pantoprazole. Surgical history included bilateral cataract removal and hernia repair. The presentation documented no tobacco, alcohol, or illicit drug use. Family history, urinary symptoms, prior prostate evaluation, and prior PSA results were not

available in the source materials.

At presentation, temperature was 97.4°F, heart rate 82 beats/min, blood pressure 117/74 mmHg, and oxygen saturation 90% on room air. He appeared uncomfortable and had moderate difficulty speaking because of dyspnea. Lung examination showed decreased bilateral air movement without wheezes, rhonchi, or rales. Cardiac examination revealed an irregular rhythm with normal S1 and S2. There was 1+ bilateral lower-extremity edema. The abdomen was soft, non-tender, and non-distended. Range of motion, strength, and sensation were documented as symmetric in the upper and lower extremities; tenderness to palpation was present in both lower extremities and was greater on the right.

Initial laboratory testing showed a white blood cell count of 6.4 × 10^3/µL, red blood cell count of 3.01 × 10^6/µL, hemoglobin 9.6 g/dL, hematocrit 29.4%, mean corpuscular volume 98 fL, and platelet count 128 × 10^3/µL. The D-dimer was 12.41 mg/L, pro-B-type natriuretic peptide (BNP) was 6,064 pg/mL, and two troponin measurements were reported as less than 3 ng/L. The basic metabolic panel was described as within normal limits. Respiratory viral testing was negative.

Chest radiography showed hypoventilation with crowding of vascular markings; superimposed congestion could not be excluded, and small bilateral pleural effusions were noted (Figure *1*). The CT pulmonary angiography showed no pulmonary embolism but demonstrated cardiomegaly, small pleural effusions, and basilar atelectasis (Figure *2*). Bilateral lower-extremity venous ultrasonography showed no evidence of deep venous thrombosis.

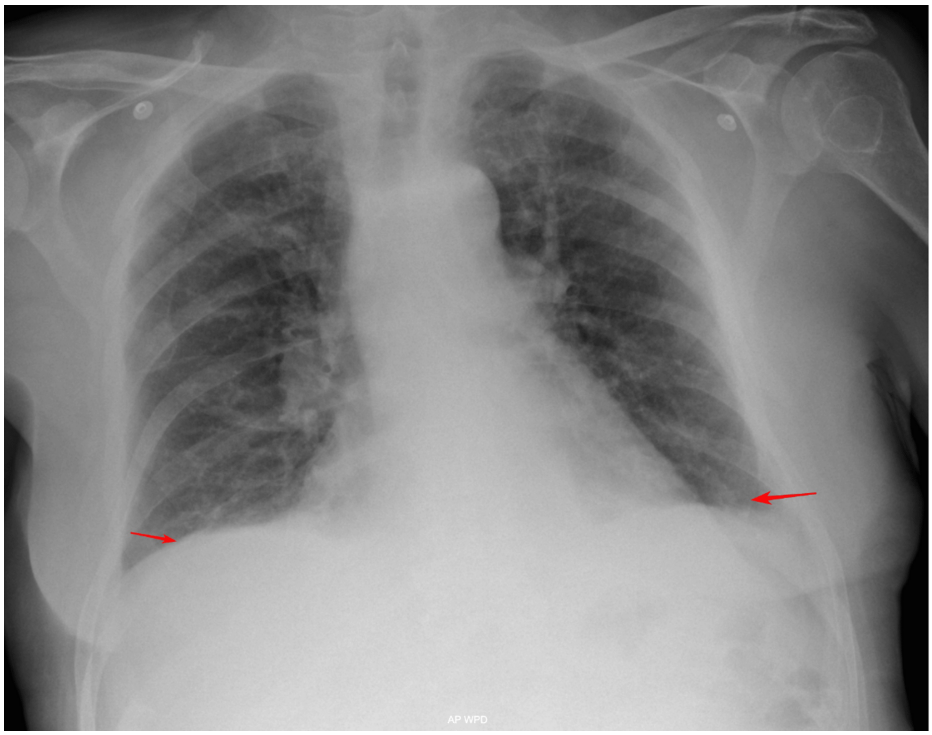


FIGURE 1: Chest radiography demonstrated hypoventilation with crowding of the vascular markings and small bilateral pleural effusions (red arrows)

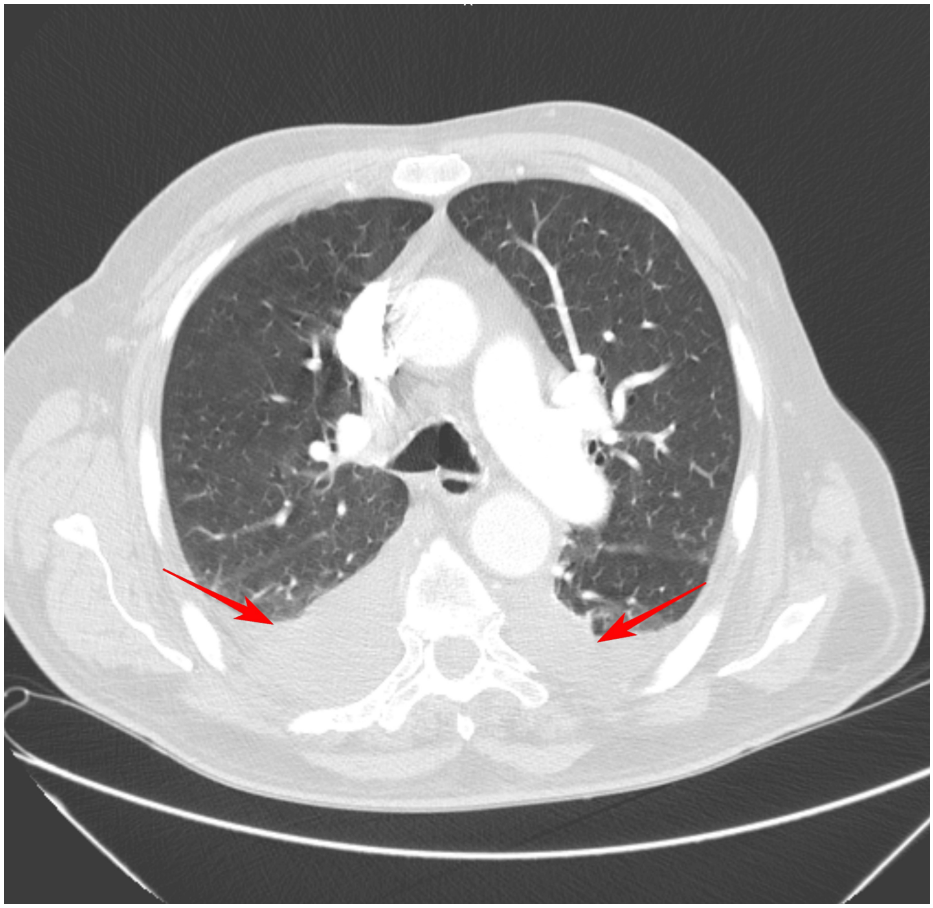


FIGURE 2: CT pulmonary angiography showed no pulmonary embolism, but revealed cardiomegaly, small bilateral pleural effusions, and bibasilar atelectasis (red arrows)

The patient was admitted for treatment of presumed acute congestive heart failure. Management included intravenous furosemide 20 mg twice daily, fluid restriction, strict intake and output monitoring, and daily weights. After two days, improvement was described as mild, and dyspnea persisted. Additional testing revealed ALP 2,152 U/L (reference range 46-116), aspartate aminotransferase (AST) 50 U/L (13-40), alanine aminotransferase (ALT) 22 U/L (7-40), total bilirubin 1.6 mg/dL (0.2-1.1), albumin 3.4 g/dL (3.4-5.0), and total protein 6.8 g/dL (5.7-8.2). Gamma-glutamyl transferase (GGT) was 54 U/L (0-73). Prostate-specific antigen (PSA) was recorded as 725 ng/mL in the source presentation. The relevant laboratory findings, including the complete blood count, cardiac biomarkers, liver-associated tests, and PSA, are summarized in Table *1*.

Parameter	Result (with units)	Reference range
White blood cell count	6.4 × 10^3 /μL	4.0-11.5 K/μL
Red blood cell count	3.01 × 10^6/μL	4.50-5.30 M/μL
Hemoglobin	9.6 g/dL	13.0-16.0 g/dL
Hematocrit	29.4%	40.0-57.0%
Mean corpuscular volume	98 fL	80-99 fL
Platelet count	128 × 10^3/μL	150-450 K/μL
D-dimer	12.41 mg/L	0.00-0.50 mg/L
Pro-BNP	6,064 pg/mL	0-125 pg/mL
Troponin (two measurements)	<3 ng/L	0-45 ng/L
ALP	2,152 U/L	46-116 U/L
AST	50 U/L	13-40 U/L
ALT	22 U/L	7-40 U/L
Total bilirubin	1.6 mg/dL	0.2-1.1 mg/dL
Albumin	3.4 g/dL	3.4-5.0 g/dL
Total protein	6.8 g/dL	5.7-8.2 g/dL
GGT	54 U/L	0-73 U/L
PSA	725 ng/mL	0.00-4.00 ng/mL

TABLE 1: Laboratory findings relevant to the diagnostic evaluation

ALP: Alkaline phosphatase; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; GGT: Gamma-glutamyl transferase; PSA: Prostate-specific antigen; BNP: B-type natriuretic peptide

The CT of the abdomen and pelvis demonstrated diffuse sclerosis of the visualized bones, raising concern for an underlying malignant or myeloproliferative process (Figure *3*). Hematology/oncology was consulted. The prostate-specific membrane antigen positron emission tomography (PSMA PET) scan demonstrated avid osseous metastatic disease with extensive pelvic and retroperitoneal lymphadenopathy (Figure *4*). The CT-guided biopsy of a left iliac bone lesion showed adenocarcinoma consistent with metastatic disease from a prostatic primary.

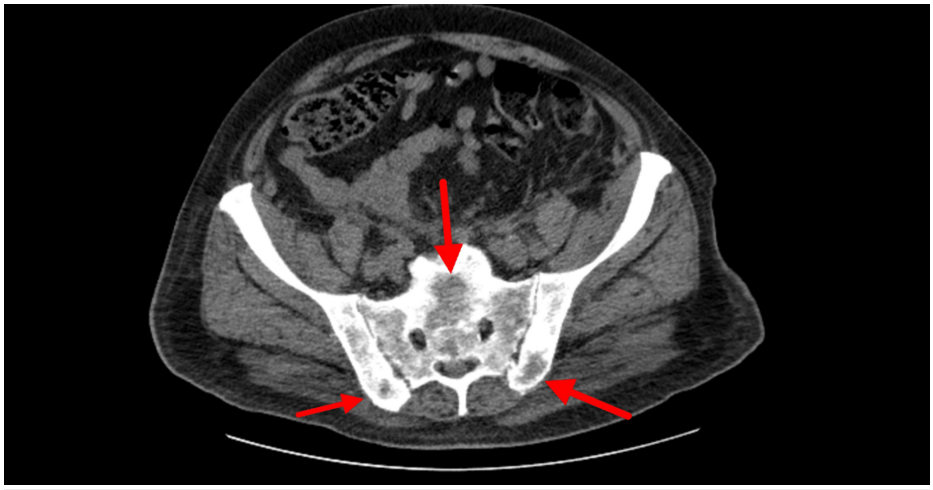


FIGURE 3: Axial CT image through the pelvis demonstrated diffuse osseous sclerosis (red arrows)

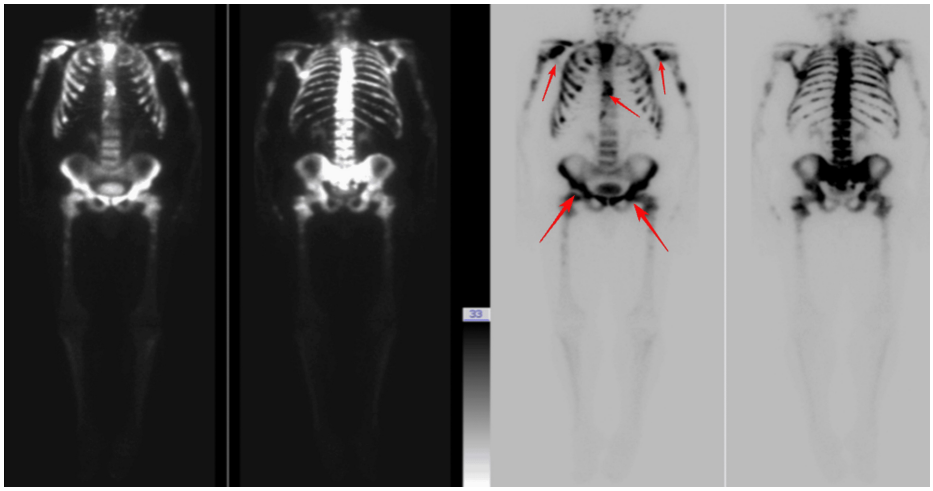


FIGURE 4: PSMA PET scan demonstrated avid osseous metastatic disease (small red arrows) and extensive pelvic and retroperitoneal adenopathy (big red arrows).

PSMA PET: Prostate-specific membrane antigen positron emission tomography

Following the diagnosis of stage IV prostate adenocarcinoma with extensive osseous metastases and pelvic and retroperitoneal lymphadenopathy, the patient declined the proposed oncologic treatment. Further details regarding the subsequent clinical course were unavailable. The chronology of the patient's presentation, diagnostic evaluation, and establishment of the final diagnosis is summarized in Table *2*.

Time point	Clinical events
Several months before admission	Progressively worsening lower back pain and chronic lower-extremity pain
Approximately one week before admission	Dyspnea and chest tightness began during physical therapy for sciatica and progressively worsened
Emergency department presentation	Oxygen saturation 90% on room air; edema, cardiomegaly, small bilateral pleural effusions, elevated pro-BNP; CT pulmonary angiography negative for pulmonary embolism
First two days of hospitalization	Treated for presumed acute heart failure with IV furosemide and supportive monitoring; only mild symptomatic improvement reported
Further inpatient evaluation	ALP 2,152 U/L and PSA reported as 725 ng/mL; CT showed diffuse skeletal sclerosis; PSMA PET showed extensive osseous disease and pelvic/retroperitoneal lymphadenopathy
Tissue diagnosis	CT-guided biopsy of a left iliac bone lesion showed adenocarcinoma consistent with a prostatic primary

TABLE 2: Clinical timeline

ALP: Alkaline phosphatase; PSA: Prostate-specific antigen; BNP: B-type natriuretic peptide; PSMA PET: Prostate-specific membrane antigen positron emission tomography

The final diagnosis was stage IV prostate adenocarcinoma with extensive osseous metastases and pelvic and retroperitoneal lymphadenopathy. The original CT-guided biopsy images and histopathology photomicrographs were unavailable for inclusion in this report. According to the final pathology report, a core biopsy of the left iliac bone lesion revealed carcinoma consistent with metastasis from a prostatic primary. Immunohistochemical staining was positive for pancytokeratin, PSA, and NKX3.1 in more than 50% of tumor cells and negative for TTF-1, CDX2, GATA3, and PAX8.

Discussion

This case demonstrates how an unexpected laboratory abnormality can lead to the diagnosis of advanced malignancy during an admission for another suspected condition. The patient's dyspnea, peripheral edema, cardiomegaly, bilateral pleural effusions, and elevated pro-BNP supported the initial evaluation for acute heart failure. However, the limited improvement after two days of diuretic treatment and the markedly elevated ALP level indicated that further investigation was needed. Although the available findings do not establish a direct relationship between the prostate cancer and the cardiopulmonary presentation, they highlight the importance of reassessing the differential diagnosis when the clinical course is not fully explained.

Bone is the most common site of distant metastasis in prostate cancer, and these metastases frequently have an osteoblastic or osteosclerotic appearance [1,2]. Prostate cancer cells interact with the bone microenvironment and stimulate abnormal bone remodeling, resulting in increased bone formation around metastatic lesions. These changes can appear as areas of increased bone density on CT. Bone metastases are also associated with pain and reduced mobility, which may substantially affect patients' quality of life [1]. In this case, the history of progressive back and lower-extremity pain preceded the diagnosis by several months. The diffuse skeletal sclerosis identified on CT was consistent with the known imaging pattern of prostate cancer metastases. When considered together with the markedly elevated PSA and subsequent biopsy results, these findings supported the diagnosis of advanced prostate adenocarcinoma.

The ALP level of 2,152 U/L was approximately 18.6 times the upper limit of the laboratory reference range and was an important clue in the diagnostic evaluation. Alkaline phosphatase can originate from bone or hepatobiliary tissue, so an elevated result is not specific for metastatic disease. In prostate cancer, increased ALP may reflect osteoblastic activity and can be associated with the extent of skeletal involvement [1]. However, ALP alone cannot establish the presence or burden of bone metastases, and its interpretation depends on other clinical and laboratory findings. In this patient, GGT and ALT were within their reference ranges, while AST and total bilirubin were mildly elevated. This pattern made a predominantly hepatobiliary source less apparent but did not confirm a bone origin. An ALP isoenzyme analysis or bone-specific ALP testing could have provided additional information if available. The combination of marked ALP elevation, progressive skeletal pain, diffuse osteosclerosis, and an elevated PSA was more informative than any single finding.

The PSA level, reported as 725 ng/mL, further increased suspicion for advanced prostate cancer. Although PSA is an important marker in the evaluation of prostate malignancy, it should be interpreted alongside

clinical findings, imaging, and histopathology. The American Urological Association and Society of Urologic Oncology (AUA/SUO) recommend obtaining a tissue diagnosis from the primary tumor or a metastatic site when advanced prostate cancer is suspected and histologic confirmation has not previously been established, provided biopsy is clinically feasible [3]. In this case, CT-guided biopsy of a left iliac bone lesion showed adenocarcinoma consistent with a prostatic primary. This finding provided histologic confirmation and supported the diagnosis suggested by the biochemical and radiographic results.

The imaging studies also had complementary roles. The CT demonstrated diffuse skeletal sclerosis and raised concern for an underlying malignant process. The PSMA PET subsequently showed extensive osseous disease and pelvic and retroperitoneal lymphadenopathy. The PSMA PET is increasingly used in prostate cancer because it can detect metastatic disease and help define its distribution; current guidelines recognize its role in the staging evaluation of advanced disease [3]. In this patient, the PET findings helped characterize the extent of the abnormalities, while the biopsy established their histologic nature. This distinction is important because imaging can strongly suggest metastatic prostate cancer, but the interpretation of an individual case should incorporate tissue findings when available.

The patient's anemia (hemoglobin 9.6 g/dL) and mild thrombocytopenia (platelet count 128 × 10^3/μL) were additional findings that deserved consideration. Extensive skeletal metastases can raise concern for bone marrow involvement, but the available information does not establish that marrow infiltration caused these abnormalities. Other causes of anemia and thrombocytopenia cannot be excluded because additional hematologic evaluation and bone marrow findings were unavailable. These abnormalities should therefore be reported as concurrent findings rather than attributed directly to the malignancy.

An important limitation of this case is that the cause of the initial cardiopulmonary presentation remains uncertain. The patient's symptoms and investigations supported evaluation and treatment for possible acute heart failure, but the available information does not establish whether this condition was independently present, whether the malignancy contributed to the presentation, or whether another process was involved. The mild improvement after diuresis does not, by itself, confirm or exclude heart failure. Similarly, the anemia and thrombocytopenia were not fully investigated in the available records. The patient declined the proposed oncologic treatment, and subsequent clinical information was unavailable; therefore, treatment response and longer-term outcomes cannot be assessed.

The main strength of this case is the convergence of several independent findings: progressive skeletal pain, markedly elevated ALP and PSA, diffuse osteosclerosis on CT, extensive disease on PSMA PET, and biopsy confirmation. These findings illustrate why unexplained bone pain and substantial biochemical abnormalities should prompt further evaluation, even when the initial presentation suggests a different acute condition. The case also emphasizes the importance of distinguishing confirmed findings from possible explanations, avoiding unsupported causal conclusions, and following established case-reporting guidelines [4,5].

Conclusions

This case highlights the importance of diagnostic reassessment when a patient's clinical course is not fully explained by the initial working diagnosis. In an older adult presenting with progressive dyspnea and treated for presumed acute heart failure, the combination of chronic skeletal pain, markedly elevated alkaline phosphatase, diffuse osteosclerosis, and a substantially elevated PSA prompted further investigation and ultimately led to the diagnosis of metastatic prostate adenocarcinoma. This case underscores the value of integrating clinical findings, laboratory abnormalities, imaging, and tissue diagnosis when evaluating unexplained abnormalities. Although the malignancy was established, the available evidence does not demonstrate a direct causal relationship between the prostate cancer and the initial cardiopulmonary presentation.

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: Kirill Efremov, Sofia Efremoff, Brody Walker, Paula Shinder

Acquisition, analysis, or interpretation of data: Kirill Efremov, Matthew Bambino

Drafting of the manuscript: Kirill Efremov, Sofia Efremoff, Brody Walker

Critical review of the manuscript for important intellectual content: Kirill Efremov, Paula Shinder, Matthew Bambino

Supervision: Kirill Efremov

Disclosures

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