Urology Case Reports 3 (2015) 59–62

Contents lists available at ScienceDirect

Urology Case Reports

journal homepage: www.elsevier.com/locate/eucr

Oncology

Oligometastatic Prostate Cancer to the Navicular Bone: Case Report

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ARTICLE INFO

Article history:
Received 3 March 2015
Accepted 9 March 2015
Available online 28 March 2015

Keywords:
Prostate cancer
Oligometastases
Navicular bone metastasis

ABSTRACT

This case of oligometastatic prostate cancer to the foot highlights the importance of: 1) metastasis remaining high in the differential for unexplained malady, in the setting of a primary cancer, despite an atypical presentation, and 2) comparing sequential imaging studies to baseline images, especially when remote, because subtle findings can declare themselves over time.

Introduction

Although prostate cancer metastasis to the axial skeleton is common, oligometastasis to the foot, especially as a solitary site, is rare. We present the case of a gentleman with known non-metastatic prostate cancer, who suffered an ankle/foot injury with progressively worsening disability who was then diagnosed with metastasis after 18 months.

Case presentation

The patient is a 64 y/o gentleman with a history of high-risk prostate cancer, cT4N0M0, treated with definitive radiotherapy and 2 years of hormone therapy (HT). Six years ago, in 2008, while in his usual state of good health, he presented with acute urinary obstruction. Diagnostic work-up revealed a PSA of 47.5 ng/mL and a digital rectal examination notable for a hardened and enlarged prostate gland. Biopsy of the prostate demonstrated Gleason 4 + 5 = 9 disease. Bone scan revealed degenerative disease bilaterally, but no evidence of skeletal metastatic disease (Fig. 1A), and CT scans demonstrated no metastatic disease. He was staged T4 owing to likely bladder neck invasion. HT was initiated with leuprolide and bicalutamide.

His PSA after 1 month of HT was 4.9. The patient was then treated with Image Modulated Radiation Therapy (IMRT) to the pelvis and prostate (4556 cGy, 1/8/09–2/10/09), followed by IMRT to the prostate only (3000 cGy, 2/11/09–3/3/09). Bicalutamide was then discontinued and he continued with 2 years of HT with leuprolide. After 6 months his PSA decreased to 0.2 and thereafter was undetectable until 12/2011 when his PSA became detectable at 0.3.

In 12/2012 his PSA rose to 1.0 and then on 6/17/2013 it demonstrated an increase to 4.4. During the same month, he twisted and sprained his ankle while playing golf. He was able to ambulate and did not seek immediate medical attention. On 7/12/2013, while having continued discomfort in the left ankle and foot, a re-staging bone scan showed increased focal radiotracer uptake in the left ankle and tarsometatarsal joints compatible with his 1-month history of trauma (Fig. 1B). There was no evidence of osseous metastatic disease. The swelling increased in his left leg, ankle and foot. In August 2013 his primary medical doctor (PMD) diagnosed a deep vein thrombosis (DVT), for which he was treated with 6 months of warfarin.

In December 2013, his PSA increased to 16 and therapy was initiated with peripheral androgen blockade (PAB) (finasteride (5 mg daily) and bicalutamide (50 mg daily)). A re-staging bone scan again showed increased radiotracer uptake in the left foot and ankle, consistent with his recent injury. There was no evidence of other skeletal metastases. The CT scan showed no evidence of adenopathy. The PSA decreased to 5.57 in April 2014 – the PAB was continued and in June 2014 when the PSA decreased to 3.2 the PAB was interrupted. Then in June 2014, the PSA increased to 11, and the PAB was restarted.

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http://dx.doi.org/10.1016/j.eucr.2015.03.003

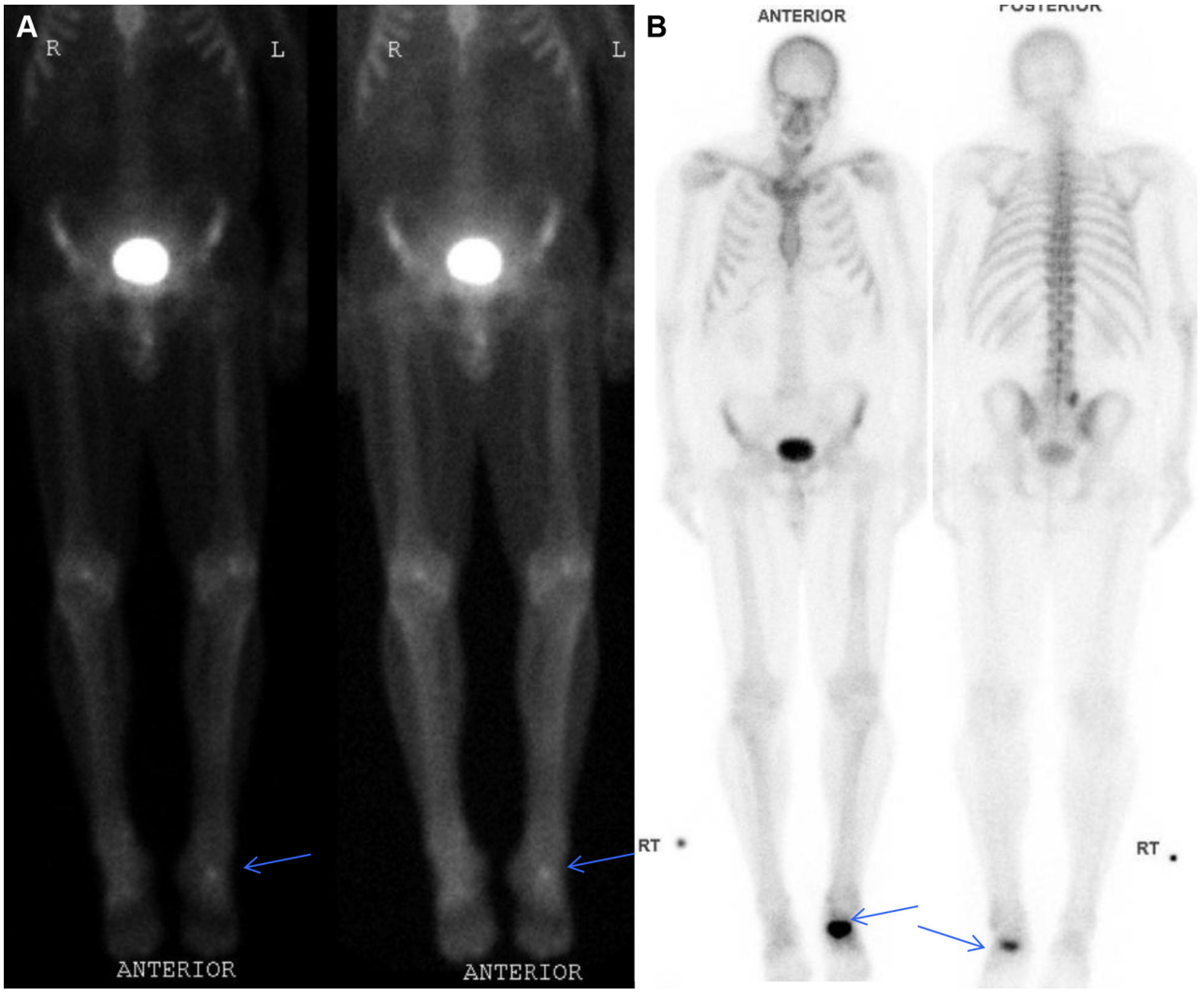

Figure 1. Bone scans demonstrating navicular necrosis. (A) Baseline (October 2008), whole body bone scan, delayed-phase anterior projections following intravenous 26.3 mCi Tc99 m, demonstrated "no evidence of skeletal metastatic disease". Note the subtle radiotracer uptake in left tarsal region (blue arrows). (B) Follow-up (July 2013, 1 month after foot trauma), whole body bone scan, 3-hour delay planar bone images in anterior and posterior projections following intravenous 26.6 mCi Tc-99m, demonstrated "no evidence of osseous metastatic disease; left tarsometatarsal joint focal increased radiotracer uptake correlated with 1-month history of ankle sprain" (blue arrows).

In August 2014, the patient experienced increased left ankle pain, as well as swelling with ambulation. His PMD ruled out a DVT, prescribed a supportive boot, and referred him to a foot and ankle surgeon. The subsequent MRI (Fig. 2) showed navicular fragmentation with multiple fracture lines and a complex talonavicular joint effusion containing blood products and marked synovitis, in addition to extensive adjacent soft tissue and bone marrow edema with mild heterogenous enhancement of the navicular. The features were

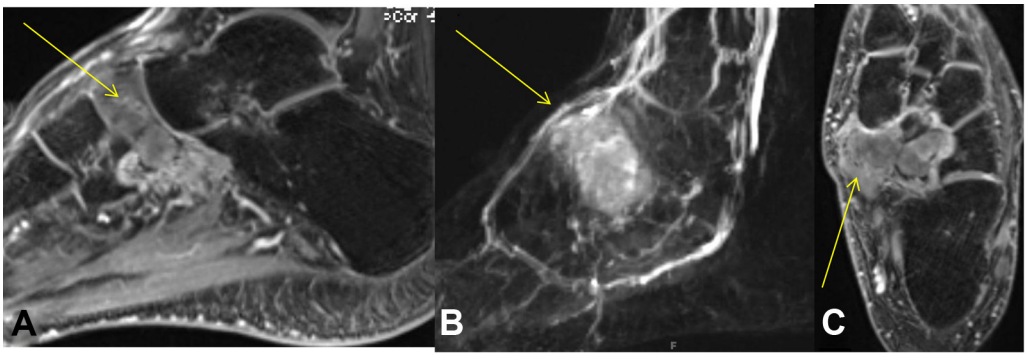

Figure 2. Contrast enhanced magnetic resonance imaging of left foot navicular bone, 14-months post foot/ankle trauma; sagittal view (A), fat saturated images in sagittal view (B), and axial view (C), demonstrated a "fragmented navicular bone, with 0.5 cm focus of nonviable bone; continued abnormal signal within the remaining fragmented navicular, with enhancement on post contrast. Imaging features more suggestive of infection rather than osteonecrosis. Tumor considered unlikely" (yellow arrows).