



The "Fisherman`s Waders" Sign on Bone Scan: A Case Report

Abdulkareem Alkahtani¹, Ahmad Subahi^{2*}, Nouf Alaqla³
and Hussein Algahtani²

¹Department of Nuclear Medicine, King Abdulaziz Medical City, Jeddah, Saudi Arabia.

²King Saud Bin Abdulaziz University for Health Sciences, Jeddah, Saudi Arabia.

³Department of Neurology, King Abdulaziz Medical City, Jeddah, Saudi Arabia.

Authors' contributions

Authors contributed evenly in this work and approved the final manuscript all over together.

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Case Study

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ABSTRACT

Fisherman`s Waders sign is caused by several medical conditions including venous thrombosis, portal hypertension and lymphoma. It is characterized by increased uptake of radiotracer in soft tissues below the mid-thorax level with normal tracer clearance above that level in bone scan. This case reports the Fisherman`s Waders sign in a patient with lymphoma complicated by Inferior Vena Cava (IVC) obstruction by the aid of CT scan and bone scan. Also, literature review of this rare sign is performed in this report. This sign can be found incidentally on bone scan and high level of awareness by the nuclear medicine physicians may alert the treating team for the underlying diagnosis.

Keywords: Fisherman`s Waders sign; IVC obstruction; lymphoma with IVC; bone scan; CT abdomen.

Key Points: Bone scan abnormality can be an indicator to the Fisherman`s Waders sign; CT scan show IVC obstruction; Fisherman`s Waders Sign is a rare sign seen in nuclear medicine.

*Corresponding author: E-mail: SubahiAH@ngha.med.sa;

ABBREVIATIONS AND ACRONYMS

IVC: Inferior Vena Cava; KAMC: King Abdulaziz Medical City; SVC: Superior Vena Cava; DVT: Deep Venous Thrombosis.

1. INTRODUCTION

Fisherman's Waders sign is an interesting bone scan finding which is caused by several medical conditions including venous thrombosis, portal hypertension and lymphoma [1]. It is an edematous manifestation characterized by increased uptake of radiotracer in soft tissues below the mid-thorax level with normal tracer clearance above that level. It is named after its resemblance a pair of fisherman's waders [2]. We are reporting a case of Fisherman's Waders sign in a patient with lymphoma complicated by Inferior Vena Cava (IVC) obstruction and we are also reviewing the literature.

2. CASE REPORT

A 59-year old woman, presented to King Abdulaziz Medical City (KAMC) - Jeddah with four months history of progressive abdominal pain and distention, associated with fever and significant weight loss (15 kg over 3 months). She is a known case of hypertension, dyslipidemia and hypothyroidism. Physical examination revealed a blood pressure of 116/55 mm Hg, temperature of 36.6°C, heart rate of 106 beats per minute and respiratory rate of 19 breaths per minute. She was pale with several enlarged hard, painless and movable lymph nodes in the right axilla (1.5 cm x 1.0 cm). Abdominal examination showed a distended abdomen with hepatomegaly (20 cm below the costal margin) and large non-mobile solid epigastric mass (20 cm x 16 cm).

Laboratory investigation showed normal serum creatinine, electrolytes and thyroid function tests. Blood urea nitrogen (BUN) was 11.5 mmol/L (normal range 3.0 - 9.2 mmol/L) and the LDH was 1417 U/L (normal range 125 - 243 U/L). White blood cells (WBC) was elevated to 12.8 mm³ (normal range 4 - 11 mm³). Her hemoglobin was low at 7.2 gm/dL (normal range 11.5 - 16.5 gm/dL), hematocrit of 7.2% (normal range 11.5 - 17.5%) and red blood cells (RBC) was 2.8 mm³ (normal range 4.5 - 6.5 mm³). Differential count showed elevated neutrophils at 10.59 (normal range 2.00 - 7.50) with normal other cells. Platelet count was 821/mm³ (normal range 150 - 450/mm³) with normal prothrombin time and INR.

Liver enzymes showed AST 261 U/L (normal range 5 - 34 U/L), ALT 62 U/L (normal range 5 - 55 U/L), total protein 67 g/L (normal range 60 - 83 g/L), GGT 418 IU/L (normal range 36 - 90 IU/L) and ALK 424 U/L (normal range 40 - 150 U/L). Adjusted calcium was 2.72 mmol/L (normal 2.10 - 2.55 mmol/L) and uric acid 609 µmol/L (normal range 150 - 350 µmol/L). Serology for both Hepatitis B and C viruses were negative.

Neck, chest, abdomen and pelvis CT showed enlarged liver with multiple lesions, ascites, and small bilateral pleural effusions. It also showed multiple bilateral cervical, axillary, subpectoral, mesenteric, and retroperitoneal lymph nodes. In addition, a large dense mass measuring 20 x 20 cm, encasing the hepatic area and causing compression on the portal and mesenteric vessels. Biopsy from the liver lesion showed diffuse large B cell lymphoma (see Fig. 1).

Bone scan was performed by intravenous injection of Tc-99^m MDP (20 mCi). Anterior and posterior whole body planar imaging was acquired at 3 hours with static images of the chest and the pelvis. It showed abnormal diffuse uniform soft tissue activity inferior to the nipple line likely due to the compression of the inferior vena cava by the intra-abdominal lymphomatous mass (Fisherman's Waders Sign) (see Fig. 2). There was a linear, moderate increased activity of horizontal orientation at the level of L5, where the site of compression fracture was seen on CT scan. It also shows nonspecific mild increased activity of the shoulders, hips, femurs and proximal tibiae (may be due to physiological bone marrow expansion).

3. DISCUSSION

The inferior vena cava (IVC) is the largest vein in the body, which is formed by the union of the common iliac veins (see Fig. 3). It begins anterior to the body of L5 vertebrae. The union occurs approximately 2.5 cm to the right of the median plane, inferior to the aortic bifurcation and posterior to the proximal part of the right common iliac artery. The IVC ascends on the right side of the lumbar vertebrae body (L3 - L5), psoas major and aorta. It leaves the abdomen by passing through the caval opening in the diaphragm and enters the thorax at the T8 vertebral level. The overall length of the IVC is 7 cm, which is greater than that of the abdominal aorta. The IVC has no valves except for a variable, non-functional one segment at its orifice in the right atrium of the heart [3].

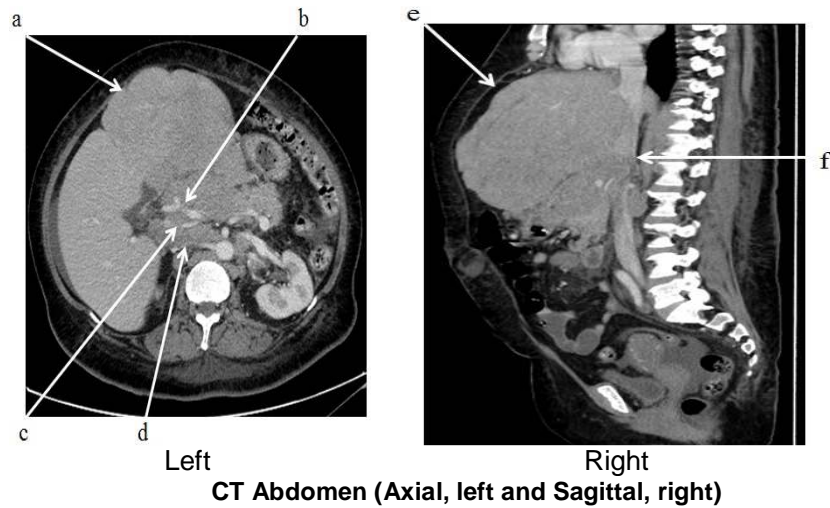


Fig. 1. Contrast enhanced CT abdomen (axial, left and sagittal, right), showing enlarged left lobe of the liver with an iso-dense mass, representing the known lymphomatous mass (arrows a & e). It is encasing the hepatic artery (arrow b) with causing compression, and stretching of portal and superior mesenteric vein (arrow c) as well as compressing the inferior vena cava (arrow f). However, no obvious venous thrombosis spotted. There are multiple lymphadenopathy porta-hepatis, mesenteric and retroperitoneal lymphadenopathy with encasement of the aorta at a level of renal artery (arrow d) with retrocaval extension, which is extend in the left side till the level of aortic bifurcation, representing multiple matted enlarged lymph nodes

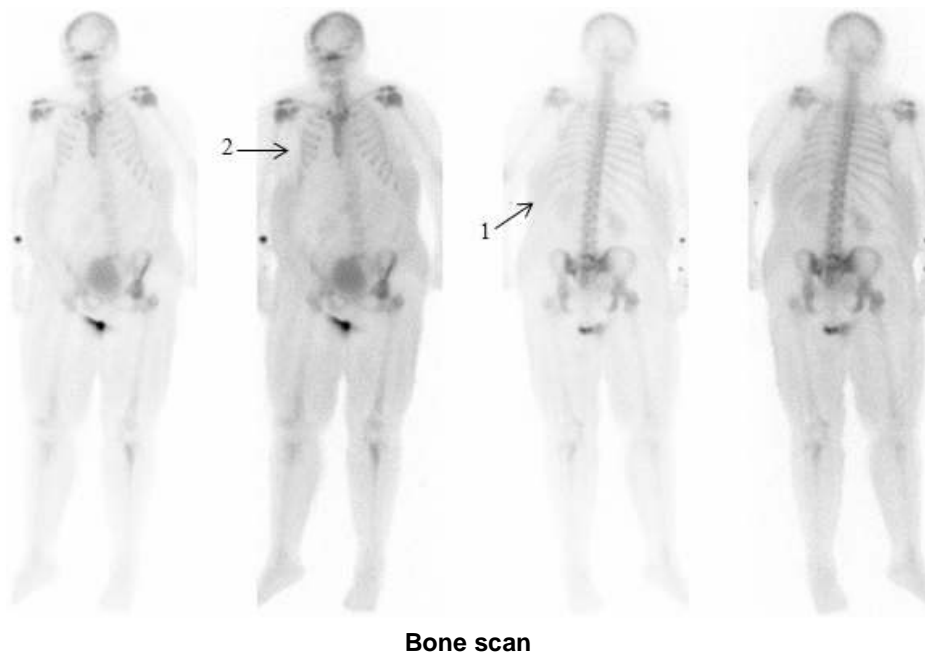


Fig. 2. Bone scan with Tc-99m MDP (20 mCi) injected I.V (whole body planar, anterior and posterior imaging was acquired for 3 hours), showing abnormal diffuse uniform soft tissue activity (arrow 1), which is inferior to the nipple line (arrow 2). This is observed likely due to the known compression of the inferior vena cava by the intra-abdominal lymphomatous mass (Fisherman's Waders sign)

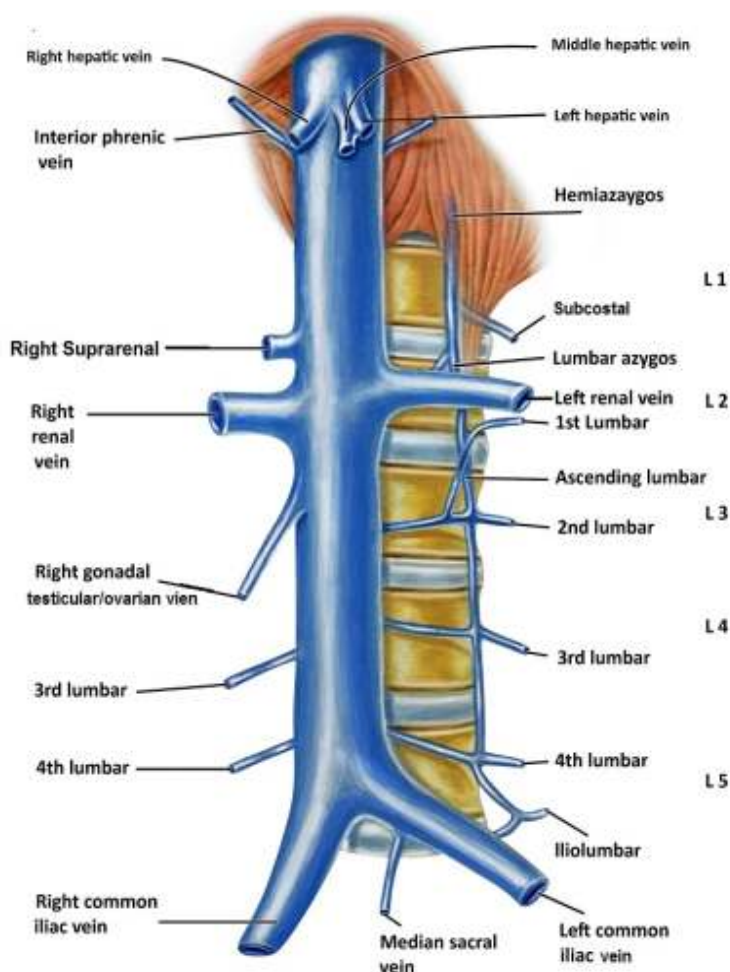


Fig. 3. Tributaries of the inferior vena cava and lumbar veins [4]

Poorly oxygenated blood from the lower extremities, most of the back, the abdominal walls and the abdomenopelvic viscera is carried by the IVC. Almost all the blood from the digestive tract is collected by the hepatic portal system and passes through the hepatic veins to the IVC. The tributaries of the IVC correspond to the paired visceral and parietal branches of the abdominal aorta. The veins that correspond to the unpaired visceral branches of the aorta are instead tributaries of the hepatic portal vein. The branches corresponding to the paired visceral branches of the abdominal aorta include the right suprarenal vein, the right and left renal veins, and the right gonadal (testicular or ovarian) vein. The left suprarenal and gonadal veins drain indirectly into the IVC because they are tributaries of the left renal vein. Paired parietal branches of the IVC include the inferior phrenic veins, the 3rd (L3) and 4th (L4) lumbar veins and

the common iliac veins. The ascending lumbar and azygos veins connect the IVC and superior vena cava (SVC), either directly or indirectly providing collateral pathways [3].

In the presence of the IVC obstruction, the collateral venous channels can be divided into 4 major pathways [1]:

- A. Superficial Pathways: The inferior epigastric vein communicates with the superior epigastric veins and the internal mammary veins. In addition, the circumflex iliac and superficial epigastric vein communicates with the axillary veins.
- B. Intermediate Pathways: The periureteric veins drain into the ipsilateral renal vein. In addition, the gonadal vein drains into the infrarenal IVC on the right and into the renal vein on the left.

- C. Deep Pathway: The ascending lumbar venous system communicates with the azygous-hemiazygous system.
- D. Portal Pathway: The internal iliac veins communicate with the inferior mesenteric vein via the haemorrhoidal venous plexuses. In addition, the inferior epigastric veins may anastomose with the umbilical and para-umbilical veins and communicate with the left branch of the portal vein.

The first report of Fisherman's Waders sign was addressed by Rodmann et al in 1990. He described a patient with hypernephroma and tumorous IVC obstruction. Recognition of the Fisherman's Waders sign in a bone scan, a frequently practiced study, may allow identification of IVC obstruction [1].

The exact mechanisms explaining increased soft tissue uptake of Tc-99^m MDP on bone-scanning are not clear, however several mechanisms may contribute including expanded interstitial volume, malignant new bone formation, dystrophic and/or metastatic calcifications, transchelation with metals, radiopharmaceutical factors, and abnormal retention of activity in the intravascular space. Normally, there is a dynamic equilibrium between osseous uptake of bone-seeking agents and their presence in the intravascular and interstitial volume. The case presented in this report had severe edema rising up to the mid chest due to IVC obstruction, thus resulting in soft tissue accumulation of bone imaging radiotracer (see Fig. 2). Theoretically, the bone scan may show a Fisherman's Waders sign in any patient with severe generalized edema. However, this sign may be more obvious in patients with IVC obstruction because IVC obstruction does not affect the venous return of the upper limbs [1].

IVC obstruction has similar primary mechanisms in the pathophysiology to lower limb deep venous thrombosis (DVT). These mechanisms include hypercoagulability related to hematological or neoplastic abnormalities, venous stasis secondary to external compression from tumors or inflammatory processes, and vessel injury due to trauma [5]. Other causes of IVC obstruction include tumor invasion of the IVC, aortic dissection causing displacement and compression of IVC, and extrinsic compression of the IVC due to adenopathy, metastatic disease, lymphoma, or granulomatous disease [6]. Extrinsic compression of the IVC by tumors such as testicular cancer, lymphoma, pancreatic

cancer, Wilms' tumor and sarcoma, are relatively rare events associated with advanced cancer [7]. Intravascular extension and tumor thrombi are most commonly seen in patients with renal cell carcinoma (RCC), occurring in 4% to 10% of RCC cases [8]. Intravascular extension of lymphoma is a rare clinical finding. In our case, no IVC thrombosis was seen on imaging.

4. CONCLUSION

We are describing a case of Fisherman Wader's sign and stressing on the clinical importance of early detection due to therapeutic implications. This sign can be found incidentally on bone scan and high level of awareness by the nuclear medicine physicians may alert the treating team for the underlying diagnosis.

CONSENT

Patient gave her informed consent before she gets involved in this study.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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