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The "Fisherman's Waders" sign in a bone scan of inferior vena cava thrombosis associated with nephrotic syndrome

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This paper presents a 61-year-old male patient with nephrotic syndrome, hypercoagulability and IVC thrombosis. Increased soft tissue uptake below the level of the mid chest was seen in his bone scan. The term "Fisherman's Waders" sign is suggested for this finding, whose recognition may permit the identification of inferior vena cava obstruction in bone scans. The existence of a cavo-portal shunt was also confirmed by dynamic scintigraphy.

Key words: inferior vena cava, thrombosis, cavo-portal shunt, nephrotic syndrome, ^{99m}Tc-Medronate, radionuclide imaging

INTRODUCTION

IT HAS BEEN WELL DOCUMENTED that the nephrotic syndrome is associated with a hypercoagulable state and a risk of thromboembolism.[1,2] Herein we present a nephrotic syndrome patient complicated by hypercoagulability and inferior vena cava (IVC) thrombosis, in whom a bone scan showed increased soft tissue uptake below the level of the mid chest due to severe generalized edema. The nature of the scintigraphic image (Fig. 1) suggests that the "Fisherman's Waders" sign would be an appropriate name for this phenomenon. Awareness of this sign, even if observed in a bone scan performed for other reasons, can provide great benefit to the patient.

CASE REPORT

A 61-year-old man, who was previously in good health, presented with right flank pain and progressive swelling of his legs. On admission, physical examination revealed a blood pressure of 130/70 mmHg, tortuous superficial veins over the anterior chest wall and the upper abdominal area, right costovertebral angle tenderness, and severe pitting edema in both legs and in the lower back region. The cardiac examination was unremarkable. Results of laboratory investigations were: serum creatinine 1.1 mg/dl (normal, 0.6–1.4 mg/dl); BUN 22 mg/dl (normal, 7–20 mg/dl); serum sodium 140 mEq/l (normal, 137–147 mEq/l); serum potassium 3.7 mEq/l (normal, 3.4–4.7 mEq/l); serum chloride 104 mEq/l (normal, 101–114 mEq/l); serum cholesterol 487 mg/dl (normal, 150–245 mg/dl); triglycerides 238 mg/dl (normal, 20–200 mg/dl); fasting blood glucose 105 mg/dl (normal, 65–125 mg/dl); total protein 4.6 g/dl (normal, 6.4–8.4 g/dl); albumin 2.0 g/dl (normal, 3.7–5.3 g/dl); hemoglobin 12.1 g/dl; hematocrit 37.5%; and white cell count 7,600/mm^3. Dipstick urinalysis revealed 4+ protein and 3+ blood and the 24-hr urine protein content was 25 g. Both the antinuclear antibody and serum hepatitis B surface antigen tests were negative. A comprehensive workup for a hypercoagulable state revealed a platelet count of 362,000/mm^3 (normal range, 130,000–400,000/mm^3), prothrombin time 10.1 seconds (control, 13.4 seconds), activated partial thromboplastin time 36.2 seconds (control, 39.4 seconds), factor VIII 189.5% (normal range, 70–140%), factor V 58.1% (normal range, 70–140%), factor VII 24% (normal range, 60–150%), factor II 32% (normal range, 60–150%), factor X 17% (normal range, 60–150%), antithrombin III 75% (normal range, 70–140%), protein C 41.2% (normal range,

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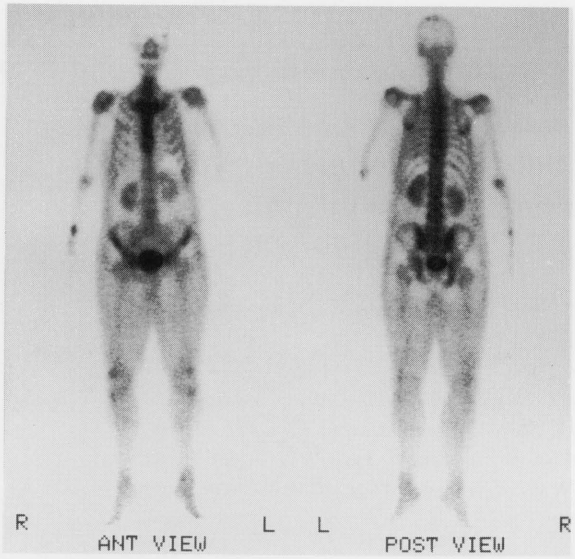
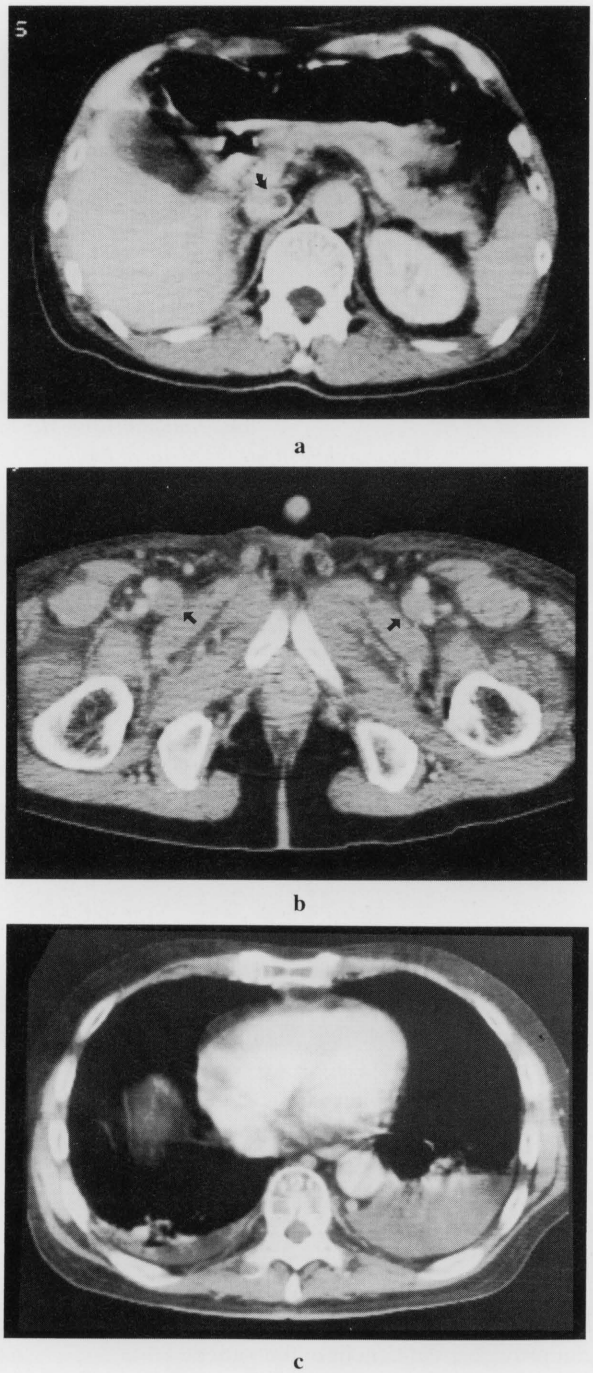


Fig. 1 ^{99m}Tc MDP whole body scan, obtained 4 hours post injection, in the anterior and posterior projections, showing soft tissue uptake of radiotracer in the distribution of "Fisherman's Waders" (below the mid chest level).

70–140%), and fibrinogen 889 mg/d*l* (control, 280 mg/d*l*). A chest radiograph demonstrated left-sided pleural effusion. Intravenous urography revealed no structural or functional anomality. A computed tomographic (CT) scan of the lower chest, abdomen, and pelvis showed an extensive thrombus extending from the subhepatic portion of the IVC (Fig. 2a) down to the bilateral femoral veins (Fig. 2b) as well as suspected thrombosis of the right renal vein. Subcutaneous edema was also noted (Fig. 2c). The liver, spleen, pancreas and kidneys were normal. Magnetic resonance imaging revealed extensive IVC thrombosis (Fig. 3a) extending into the bilateral iliac veins (Fig. 3b), but unfortunately the bilateral renal veins and femoral veins were not imaged. Suspecting tumor-induced IVC obstruction, ^{99m}Tc-methylene diphosphonate (MDP) bone imaging was performed, which clearly revealed increased soft tissue activity below the mid chest level similar to the appearance of "Fisherman's Waders" (Fig. 1). For a better evaluation of the venous obstruction, a radionuclide venogram was performed by injecting 3 mCi (111 MBq) ^{99m}Tc-macroaggregated albumin (MAA) into the dorsal veins of both feet. There was evidence of complete obstruction of both the IVC and the right proximal femoral vein with collaterals (Fig. 4) as well as homogeneous radionuclide uptake in the liver (Figs. 4 and 5). The lung perfusion was normal without any defect suggesting pulmonary emboli. Under suspicion of a cavo-portal shunt, a dynamic study was performed the next day with 3 mCi of ^{99m}Tc-phytate injected into a right dorsal pedal vein. The IVC was not visualized, but ill-defined collateral veins could be seen. Subsequently there was uniform trapping of the radionuclide in the liver (Fig. 6). A series of examinations, including a physical examination, various radiological studies, gastrointestinal endoscopy, tumor markers, and bone and gallium scans, failed to find any evidence of thrombophlebitis in the lower extremities, pelvic inflammatory disease, or malignancy.



a

b

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Fig. 2 a. Contrast-enhanced CT scan immediately inferior to the level of the liver reveals a thrombus in the lumen of the IVC (arrow). b. Contrast-enhanced CT scan through the lower pelvis shows bilateral femoral veins thrombosis (arrows). c. Contrast-enhanced CT scan of lower chest reveals subcutaneous edema.

Based on the clinical findings and the biochemical,

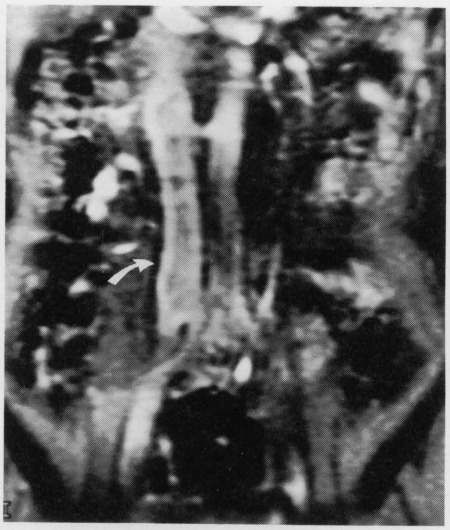

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Fig. 3 a. Coronal gradient refocused echo image (TR/TE 50/4 ms, flip angle 60°) clearly shows extensive IVC thrombosis (arrow). b. Coronal gradient refocused echo image (TR/TE 46/6.7 ms, flip angle 60°) demonstrates extension of the thrombus into the bilateral iliac veins (arrows).

radiological and radionuclide examinations, nephrotic syndrome associated with a hypercoagulable state and IVC thrombosis was diagnosed. During hospitalization, the patient was managed with intravenous furosemide, albumin, and anticoagulant therapy. A kidney biopsy was not performed because he was on anticoagulant therapy. After being discharged from the hospital, he was maintained on warfarin and prednisolone. Six months later, a follow-up CT scan showed residual small thrombi in the IVC.

DISCUSSION

Thromboembolism is one of the main complications in patients with the nephrotic syndrome, with an incidence ranging from 10 to 30%.[3] Deep vein thrombosis in the leg, renal vein thrombosis, and pulmonary embolism are the most common thromboembolic complications,[4] although

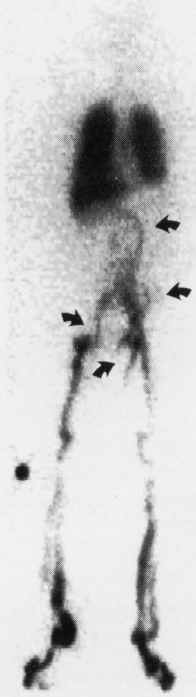

Fig. 4 The radionuclide venogram shows occlusion of the IVC with hang-up of radioactivity, truncated right proximal femoral vein, and collateral channels (arrows), as well as homogeneous uptake of ^{99m}Tc MAA in the lungs and liver.

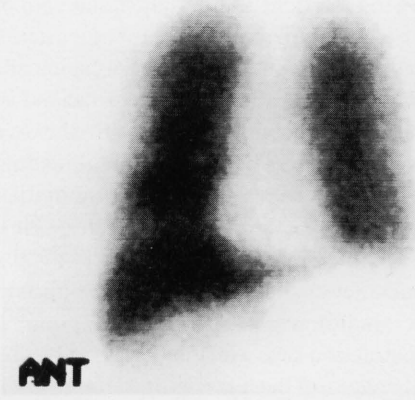

Fig. 5 Anterior view of both lungs and upper abdomen after injection of ^{99m}Tc MAA in the dorsal pedal veins showing homogeneous uptake in both liver and lungs.

IVC thrombosis has also been reported.[5] The nephrotic syndrome can change the turnover and concentrations of most plasma proteins, including those that take part in the coagulation cascades, and the resulting coagulation disorders tend to induce blood hypercoagulability,[4] as was indicated in the results of some of this patient's blood studies. The association between hypercoagulability and

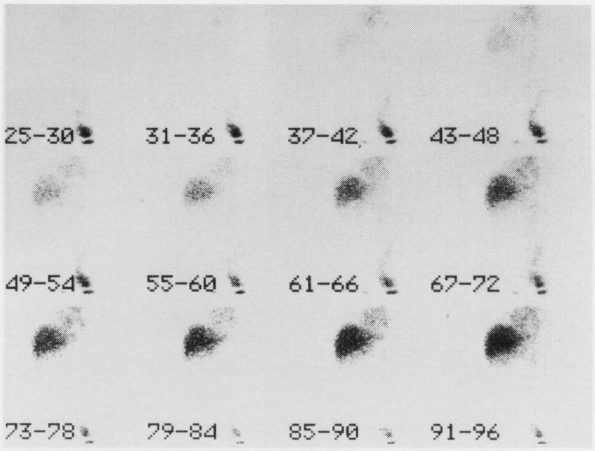

Fig. 6 ^{99m}Tc phytate was injected in a right dorsal pedal vein. Initially the liver and heart are seen simultaneously, then gradually there is uniform trapping of the radionuclide in the liver. Note the pericaval collaterals, and non-visualization of the IVC, the spleen and kidneys.

thromboembolic complications in the nephrotic syndrome has been well established. The nephrotic syndrome patient presented in this paper was clearly in a hypercoagulable state and developed extensive IVC thrombosis extending into the bilateral femoral veins.

In the presence of IVC occlusion, the collateral venous channels can be categorized into four major pathways:[6,7] (a) Deep pathway: The ascending lumbar veins can communicate with the azygous-hemiazygous system. (b) Intermediate pathway: The periureteric veins drain into the ipsilateral renal vein; the gonadal veins drain into the infrarenal IVC on the right and into the renal vein on the left. (c) Superficial pathway: The inferior epigastric veins communicate with the superior epigastric veins and the internal mammary veins; the circumflex iliac and superficial epigastric veins communicate with the axillary veins. (d) Portal pathway: The internal iliac veins communicate with the inferior mesenteric vein via the hemorrhoidal venous plexuses; the inferior epigastric veins may anastomose with the umbilical and para-umbilical veins and communicate with the left branch of the portal vein. Scintigraphic studies which show a cavo-portal shunt after IVC obstruction are well documented.[8–12] In this case, the existence of the cavo-portal shunt could be confirmed by dynamic scintigraphy, which clearly showed collateral veins and early hepatic uptake. The absence of splenic uptake rules out reticuloendothelial cell uptake of colloidal particles and the absence of renal uptake rules out a right to left shunt.

The mechanisms responsible for soft tissue uptake on bone-scanning include expanded interstitial volume, malignant new bone formation, dystrophic calcification, metastatic calcification, transchelation with metals, radiopharmaceutical factors, and abnormal retention of activity in the intravascular space.[13] In this patient, the diffuse increased soft tissue uptake of ^{99m}Tc-MDP below the mid chest level can be explained by the mechanism of expanded interstitial volume. Normally there is a dynamic equilibrium between osseous uptake of bone-seeking agents, and their presence in the intravascular volume, and interstitial volume. The case presented in this paper had severe edema rising up to the mid chest due to loss of protein in the urine and IVC obstruction, thus resulting in soft tissue accumulation of bone-imaging radiopharmaceuticals. Many disorders can cause edema formation,[14] including venous thrombosis and the nephrotic syndrome. 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. In this patient, the soft tissue clearance above the mid chest level was normal (Fig. 1).

In conclusion this is the first report of a scintigraphic recognition of IVC thrombosis in a patient with nephrotic syndrome. Only one previous report,[15] in a patient with hypernephroma and tumorous IVC, showed just a ^{99m}Tc-MDP bone scan finding similar to this report. Recognition of the "Fisherman's Waders" sign in a bone scan, a frequently practised study, may allow identification of IVC obstruction.

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