

# Unusual mediastinal lymph node uptake and peritoneopleural fistula demonstrated on Technetium-99m macro-aggregated human serum albumin (Tc-99m MAA) peritoneal scintigraphy in a patient with portal hypertension

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## SUMMARY

Peritoneal radionuclide scan is an established imaging modality for evaluating peritoneopleural communications. In this case report, unusual mediastinal lymph node radiotracer uptake is seen in a patient with portal hypertension on peritoneal scintigraphy. This was suspected to be due to marked lymphatic enlargement from longstanding portal hypertension since childhood, permitting passage of the large Tc-99m MAA particle. The nodes were morphologically benign on CT. Mediastinal lymph node uptake on peritoneal scintigraphy is rare but should not raise undue clinical concern, particularly in a patient with chronic portal hypertension. Anatomic correlation with SPECT-CT can provide reassurance.

## INTRODUCTION

The peritoneal radionuclide scan is an established imaging modality in evaluating for peritoneopleural communications, particularly in the setting of a patient with a large, unilateral pleural effusion, accompanied by risk factors of chronic ascites or known peritoneal dialysis. In our institution, we use Technetium-99m macro-aggregated human serum albumin (Tc-99m MAA) as the radiotracer of choice. This is due to its large particle size of 10-100µm, which typically precludes absorption by intra-abdominal/transdiaphragmatic capillaries and lymphatics, causing it to remain in the peritoneal cavity except in the presence of a significant diaphragmatic defect or other communication. Extra-peritoneal/pleural uptake is rare.

In this case report, we describe a patient with peritoneopleural fistula diagnosed on peritoneal scintigraphy, but with unexpected tracer uptake in mediastinal lymph nodes.

## CASE REPORT

Our patient is a 35-year-old male of Chinese ethnicity. He had a significant past medical history of chronic portal and superior mesenteric vein thrombosis secondary to umbilical vein catheterisation and neonatal sepsis. This resulted in

chronic non-cirrhotic, extra-hepatic, portal hypertension. Hepatic venous pressure gradient measurement confirmed the pre-hepatic aetiology of portal hypertension, with corrected sinusoidal pressure measured at 4.3mmHg. The portal hypertension was complicated by ascites, as well as oesophageal varices.

The patient presented to the Emergency Department with a 4-day history of shortness of breath associated with non-productive cough. Clinical examination revealed reduced breath sounds over the whole right chest with dullness to percussion. The patient was afebrile and plasma total white cell counts were normal. Chest radiograph confirmed the finding of a large right pleural effusion (Figure 1). Diagnostic pleural and therapeutic abdominal taps revealed that fluids from both cavities were transudates.

Peritoneal scintigraphy was performed as part of the diagnostic work-up. Four mCi of Tc-99m MAA was diluted and infused passively into the peritoneal cavity via an abdominal drainage catheter. Radiopharmaceutical purity was verified beforehand by routine quality control assessment. Sequential static images were acquired for 30 minutes, followed by non-contrast-enhanced single photon emission computed tomography (SPECT-CT), as well as delayed static images.

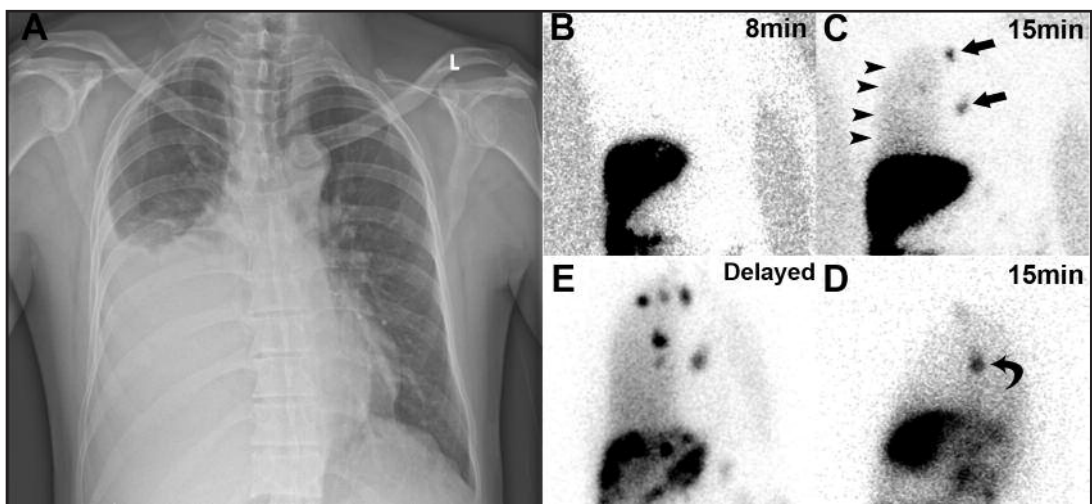
Planar images showed prompt, diffuse accumulation of radiotracer throughout the right hemithorax over the course of 30 minutes, suspicious for peritoneopleural fistula (Figure 1). However, from the 15-minute mark onwards, multiple discrete foci of radiotracer uptake were seen in the mediastinum. Correlative SPECT-CT revealed these to represent subcarinal, right internal mammary, subpectoral, and right paratracheal lymph nodes (Figure 2). These demonstrated benign morphology on CT with small size and preservation of fatty hilum. No overtly suspicious lesion was detected on CT.

The patient was treated with spironolactone and low sodium diet, with clinical improvement in the amount of ascites, and radiographic reduction in the size of the pleural effusion. The

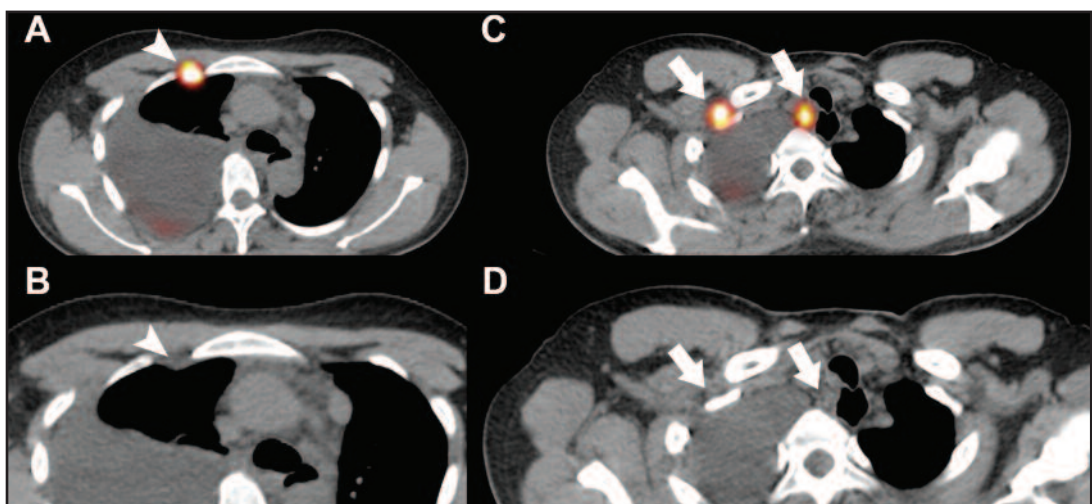
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**Fig. 1:** (clockwise from left) A) PA chest radiograph of the patient for reference showing the large right pleural effusion. Planar peritoneal scintigraphic images of the patient taken at 8min (B, anterior projection with flood), 15min (C, anterior with flood and D, lateral), 165min delayed image (E, anterior). Note the presence of radiotracer in the right hemithorax from 15min indicative of a pleuroperitoneal fistula (arrowheads). Abnormal focal uptake is seen in the central chest (arrows) that is localised to the middle mediastinum on the lateral projection (curved arrow). The delayed image shows further uptake in the central and upper mediastinum.



**Fig. 2:** Fused SPECT-CT images (A and C) showing intense tracer uptake at the right internal mammary (arrowheads), and right subpectoral and paratracheal stations (arrows) respectively. Magnified, unfused, CT images at the corresponding slices (B and D) showing that the tracer uptake corresponds to tiny lymph nodes without suspicious features. These were deemed non-specific and unlikely of clinical significance.

mediastinal/upper abdominal lymph node uptake was deemed non-specific and no further investigation or treatment was necessary.

**DISCUSSION**

There are three possible mechanisms for the large pleural effusion in our patient<sup>1</sup>: first, the leakage of fluid via a diaphragmatic defect; second, azygos venous hypertension; third, transdiaphragmatic leakage via lymphatic channels. A fourth possible mechanism (seen in hepatic hydrothorax), that of hypoalbuminemia, is not applicable as the patient is not cirrhotic and has normal serum albumin.

The rapid visualisation of tracer in the thoracic cavity in our patient indicates that the first mechanism, a diaphragmatic defect, is likely the dominant cause for the pleural effusion. Multiple studies have shown the presence of discontinuities in the tendinous portion of the diaphragm. In the setting of raised intra-abdominal pressure such as in chronic ascites, subcentimetre blebs<sup>2</sup> may herniate through these defects and subsequently rupture, forming a peritoneopleural fistula. This is right sided in up to 85% of cases<sup>1</sup> as the left hemidiaphragm is more muscular and resistant to bleb formation.

However, it is unusual for radiotracer uptake to be seen along the mediastinal/internal mammary lymph node chains. The large size of the Tc-99m MAA particle typically prevents it from entering lymphatic channels in the abdomen. We suspect that the nodal uptake is due to the presence of markedly dilated lymphatic drainage channels permitting Tc-99m MAA uptake, likely secondary to longstanding portal hypertension.

Portal hypertension is known to cause splanchnic arterial vasodilation, which results in abnormal sodium reabsorption via the activation of vasoconstrictor and anti-natriuretic homeostatic mechanisms. This results in increased extracellular fluid volume and thus ascites. With the development of porto-systemic collaterals, there is also an increase in number and size of hepatic and intestinal lymphatics, as well as diameter of the thoracic duct. This has been seen with direct surgical observation<sup>3</sup> and lymphangiography. Endoscopic ultrasound<sup>4</sup> has been used to evaluate the distal end of the thoracic duct in healthy volunteers and patients with cirrhosis/portal hypertension, with a statistically significant difference in average diameter of 1.9mm and 3.1mm respectively. More recently, a feasibility study of non-contrast magnetic resonance lymphangiography<sup>5</sup> also revealed a statistically significant difference in the maximum diameter of the thoracic duct in normal patients (3.74mm) versus those with alcoholic cirrhosis (6.98mm).

A literature search did not reveal a similar pattern of lymph node uptake in published reports, nor have we previously encountered such uptake in our local experience in patients with portal hypertension. We hypothesise that the long duration of the patient's portal hypertension since childhood (a period of growth), resulted in lymphatic enlargement to a greater degree than patients with portal hypertension/chronic liver disease starting in adulthood. The correlative CT appearance in this case provided reassurance, with morphologically benign appearance of the lymph nodes, which would otherwise raise concern for pathological uptake on planar imaging alone.

## CONCLUSION

We report on a case of unusual mediastinal lymph node radiotracer uptake in a patient with portal hypertension on peritoneal scintigraphy. This was suspected to be due to marked lymphatic enlargement from longstanding portal hypertension beginning in childhood, permitting passage of the large Tc-99m MAA particle. The nodes were morphologically benign on CT and not clinically significant. Mediastinal lymph node uptake on peritoneal scintigraphy is rare but should not raise undue clinical concern, particularly in a patient with chronic portal hypertension. Anatomic correlation with SPECT-CT can provide reassurance.

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