Lung and renal uptake of technetium Tc 99m sulphur colloid related to disseminated intravascular coagulation

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Abstract. In addition to a recently published case study, we present another three cases in which we observed both lung and renal uptake of technetium Tc 99m sulphur colloid which was related to a period of disseminated intravascular coagulation. Being familiar with this relationship may influence the diagnosis and course of the illness in certain patients.

An abnormal lung uptake of technetium Tc 99m sulphur colloid (TSC) during liver scintigraphy has been described in patients with liver transplants [3, 13], spleen and bone-marrow transplants [12], malignancy [10, 16, 24], lymphoma [10, 12], liver disease [8, 10, 16, 24], intra-abdominal abscess [10], infection [5, 8, 16, 18, 25], haematologic diseases [10, 16], collagen-vascular diseases [16], mucopolysaccharidoses [14], histiocytosis-X [2] and in two patients after an injury to the thorax [9].

The renal uptake of TSC is well known in chronic renal-transplant rejection [23] and is occasionally seen after liver transplantation [17] and with congestive heart failure [4, 6, 21]. One newborn patient has been described who exhibited a diffuse lung, renal and splenic (but not hepatic) uptake of TSC during a viral infection [7]. Smith et al. [22] have described a patient who showed an accumulation of TSC by the lungs and kidneys following disseminated intravascular coagulation.

In this paper, we describe another three patients who, during liver scintigraphy showed both lung and renal uptake of TSC, which was related to a period of disseminated intravascular coagulation.

Case reports

Case 1

A 43-year-old woman was admitted to the hospital because of malaise, loss of weight and pain in the upper abdomen and back. On examination, it was clear that she was very ill. A tumour with a diameter of about 4 cm was palpated in the lateral part of the left mammary gland, and enlarged lymph nodes were felt in the left armpit. The liver was enlarged and could be palpated 1 cm below the costal margin. Laboratory investigation showed hypercalcaemia (3.35 mmol/l) and raised levels of liver enzymes in the serum [alkaline phosphatase (AP), 507 IU/l; lactic dehydrogenase (LDH), 1,920 IU/l; SGOT, 412 IU/l; SGPT, 670 IU/l]. The reference values in our laboratory were as follows: AP: P95 = 310 IU/l; LDH: P95 = 275 IU/l; SGOT P95 = 21 IU/l; SGPT P95 = 28 IU/l.

The sedimentation rate was 20 mm in the 1st hour. The haemoglobin level was 8.6 mmol/l, and chest radiography revealed no abnormalities. Mammography showed a very dense parenchyma in the left mammary gland with thickening of the skin but without a circumscribed tumour or microcalcifications.

The right mammary gland showed no abnormalities, but the pattern of the left mammary gland had changed dramatically since mammography performed 2 months before. Liver scintigraphy revealed a diffuse, irregular uptake of TSC in the enlarged liver and increased uptake by the enlarged spleen. There was also marked uptake by the lungs and kidneys, and some uptake by the spine (Fig. 1). A cytologic puncture was done in the enlarged lymph nodes in the left armpit. Tumour cells were found and, in addition, the palpable mammary tumour was punctured. Cytology demonstrated a carcinoma. One day after the liver scan, haematological investigation was performed because of the patient's carcinoma and low sedimentation rate. As a result, a diagnosis of diffuse intravascular coagulation was made [ethanol-gelation test, positive; partial thromboplastin time (PTT), 20.9 s; activated partial thromboplastin time (APTT), 57.5 s; bleeding time, more than 15 min; clotting time, 7 min 5 s; fibrin degradation products (FDPs), more than 15 mg; antithrombin III (AT III), 30%].

The patient was heparinized, and chemotherapy was started. Because of the hypercalcaemia, she was also treated with aminohydroxypropanediphosphonate (APD). In the following days, she developed adult respiratory-distress syndrome and left-heart failure, together with liver insufficiency and ascites. She gradually deteriorated and died in intractable shock.

Case 2

A 69-year-old woman was re-admitted to the hospital because of malaise, anorexia and loss of weight. Two months earlier, a cholecystectomy had been performed because of cholecystolithiasis.
She had a history of a myocardial infarction and suffered from angina pectoris. On examination, slightly elevated venous pressure was found, and there were crackles at the base of the lungs. A hepatomegaly in the liver was found which reached 2 cm below the right costal margin, and there was pitting oedema in both legs. Laboratory examination showed elevated levels of LDH (225 IU/l, mainly factor V), AP (6.0 U/l; normal 1.2–3 Bessey units/l, liver and bile fraction). Chest radiography showed consolidation of the right middle lobe. After admission, heparin therapy was started for suspected lung embolism, but as a lung scan did not support this diagnosis, the medication was stopped. Liver scintigraphy showed a patchy uptake of TSC in the liver and a marked uptake by the lungs and kidneys (Fig. 2). When the examination was repeated 2 days later, the same abnormalities were found. During her stay in the hospital, she developed thrombocytopenia (77 x 10⁹/1), and 6 days after the second liver scan, a further haematological examination showed decreased activity of the factors of the prothrombine complex, a positive ethanol-gelation test, a markedly decreased fibrinogen level and a slightly elevated FDP level. These findings demonstrated a low-grade disseminated intravascular coagulation. Therapy with low doses of heparin was started, but for no apparent reason, the patient gradually deteriorated and died 6 days later. Obduction showed old and fresh myocardial infarcts and generalized atherosclerosis. Microscopy of the lungs and kidneys showed neither signs of intravascular coagulation nor any apparent increase of cells of the reticuloendothelial system.

Case 3

A 35-year-old woman with a left-sided medullary sponge kidney and positive urine cultures was admitted to hospital with left-sided colic. Urography revealed an obstructing calculus in the middle portion of the left ureter. On the 4th day after admission, she developed urosepsis that was treated with intravenously administered antibiotics. Ultrasound revealed no marked hydronephrosis, but a cystic mass was seen which could not clearly be related to the left kidney or the spleen. Therefore, liver scintigraphy was performed which showed an uptake of TSC in the enlarged liver and a round process in the spleen, as well as marked uptake of TSC by the lungs and kidneys (Fig. 3). Differential diagnosis of the cause of these findings included disseminated intravascular coagulation. Haematological examination was performed on the same day as the liver scan.