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Tc-99m dextran scintigraphy in the diagnosis of protein losing enteropathy

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Tc-99m dextran scintigraphy was performed on a 16-year-old male patient with protein losing enteropathy. Abnormal leakage of the radiotracer was observed in the right upper lumbar area that moved over time and excreted in the stool which was suggestive of protein loss. It is concluded that Tc-99m dextran is useful in the detection of protein losing enteropathy which is not detected by extensive radiological and endoscopic examination.

Key words: protein losing enteropathy, Tc-99m dextran scintigraphy

INTRODUCTION

PROTEIN LOSING ENTEROPATHY (PLE) is a gastrointestinal disorder observed in various diseases such as Crohn's disease, ileocecal tuberculosis and ulcerative colitis which are characterized by excessive loss of plasma protein into the gut.[1,2] Alpha-l antitripsin clearance, random stool alpha-l antitripsin concentration and radiolabeled macromolecules have been used as part of the diagnosis. The localization of which segment in the gut where the protein loss occurs is clinically important. For this reason radiolabeled macromolecules are preferred for their ability to detect the amount and location of the protein loss. Initially, radioactive agents such as Cr-51, I-131, I-125 and In-111 tagged with serum proteins were used and the diagnosis was based on gradual decrease in blood activity with fecal excretion for several days. Recently, technetium-99m labeled human serum albumin and dextran have been used in the diagnosis of protein losing enteropathy.[3,4]

We present a patient with protein losing enteropathy and discuss the recent advances in imaging methods in the diagnosis of this disorder.

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CASE REPORT

A 16-year-old male with a month's history of swelling of the face and dorsum of the feet and abdominal pain was hospitalized with a diagnosis of nephrotic syndrome in another medical center and was treated with intravenous human albumin which made some improvement in his clinical status. When the patient was referred to our institution, he was a normally developed boy with an unremarkable past history. His physical examination was normal except for pitting edema on the lower extremities. The kidneys were normal on ultrasonographic examination and no ascites was detected. He had hypochromic microcyter anemia and occult blood in his stools. The total protein and albumin values were 3–7 g/dl and 1.9 g/dl, respectively. Liver function tests were normal and no significant proteinuria was present. Antigliadin antibodies IgA, IgG and anti-endomysium antibody were negative.

The upper gastrointestinal series showed edematous small intestine and normal terminal ileum. Gastroduodenal endoscopy was normal and histologic examination of duodenal biopsy samples revealed minimal lymphoplasmocytic cell infiltration with normal villi. The barium study of the colon and colonoscopy showed normal findings whereas the histologic examination revealed moderate lymphoplasmocyter and minimal polymorphonuclear cell infiltration with rare crypt ulceration.

The patient underwent lymphoscintigraphy with Tc-99m dextran. After 555 MBq Tc-99m dextran was

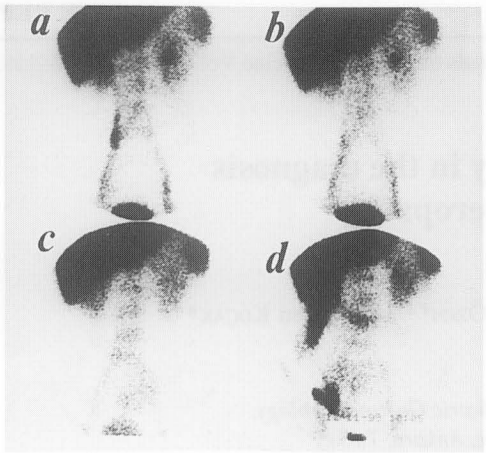

Fig. 1 Anterior static images are shown at 30 minutes (a), 60 minutes (b), 90 minutes (c) and 4 hours (d). Although no radioactivity was observed until 90 minutes, accumulation of the tracer was observed in the hepatic flexura at 4 hours.

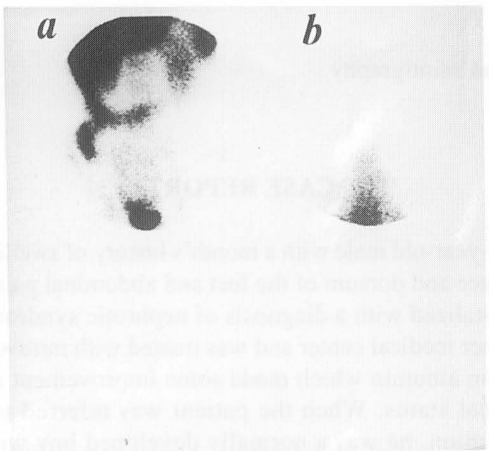

Fig. 2 The static images of the abdomen (a) and the stool (b) were taken at 24 hours show accumulation of the activity in the right upper quadrant of the abdomen in the hepatic flexura and excreted activity in the stool, respectively.

injected intravenously dynamic images of the patient was taken with a gamma camera (Toshiba 501) and low energy all purpose collimator. Although no tracer was observed in the abdomen during serial imaging up to 90 minutes, in a 4 hour image there was gradual accumulation of the tracer in the right upper abdomen in the ileocecal and hepatic flexura of the ascending colon suggestive of leakage of the tracer in bowel loops (Fig. 1). Images of the abdomen and the stools were taken at 24 hours as well as at the same location in which the suspected area of protein loss showed persistent accumulation of the tracer and the stools contained excreted radioactivity (Fig. 2).

The patient was prescribed oral mesalazine with a diagnosis of indeterminate colitis. His response to treatment was poor and he was readmitted to the hospital five

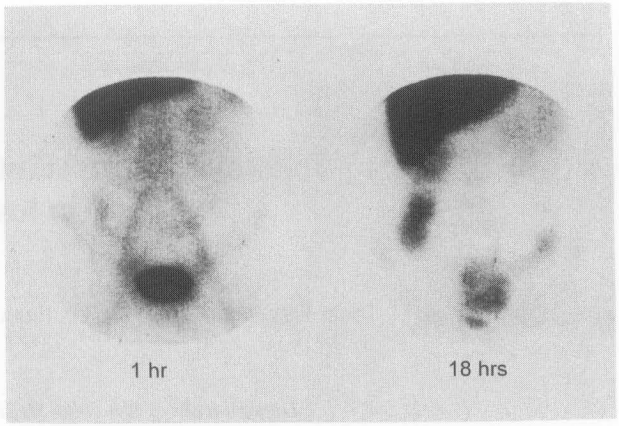

Fig. 3 A repeat scintigraphy performed 5 months later, revealed intense accumulation of the radiotracer in the ascending and the sigmoid colon at 18 hours, which was suggestive of progress in his clinical status.

months later with the same symptoms and findings. A repeat scintigraphy showed accumulation of the radiotracer in the ascending and the sigmoid colon which was suggestive of progress in his status (Fig. 3).

DISCUSSION

Gastrointestinal protein loss can occur in any condition resulting in mucosal inflammation, ulceration, abnormal mucosal permeability and destruction of the lymphatic channels.[1] This disorder is seen in several diseases including gastritis, gastric carcinoma, intestinal lymphangiectasia, celiac disease and autoimmune inflammatory diseases.[1] Endoscopic examination and biopsy, serum and stool levels of alpha-1 antitripsin and radiolabeled macromolecules were used as part of the diagnosis. Radiolabeled macromolecules such as Cr-51, I-131 and I-125 tagged with serum proteins were preferred for their ability to detect the amount and localization of protein loss but, since this was a count based method and no images were taken, it did not help in detecting the site of the leak. Later, Technetium-99m, a tracer with better physical characteristics which make it ideal for most nuclear medicine studies, is used in the localization of protein loss in gut labeled with human serum albumin.[2] But difficulty in labeling and the problem of a free or reduced form of radioactivity up to 30%, reduced the specificity of this radiopharmaceutical.[3] Also its being an anionic macromolecule and vascular endothelium being anionic itself repels such molecules, which may lead to an increase in the false negative rate. Later, human immunoglobulin G labeled with Tc-99m, in kit form, was tried. Although it had high labeling efficiency, because of its being an antigenic protein, it had the potential for immunologic hazards.[4] Recently, Bahatnagar et al.[5] reported the use of Tc-99m Dextran (Tc-99m-D) in the diagnosis of protein

losing enteropathy.

Dextran is a polysaccharide, nonprotein macromolecule with a molecular weight of 60000–90000, available both in neutral and cationic forms. It was first used as a plasma expander and then a blood-pool and lymphoscintigraphic agent when tagged with Tc-99m.[6,7] It has a long intravascular half life, is degraded slowly by the liver and is excreted from the kidneys. Its normal biodistribution does not include intestinal excretion.[8] Four to twenty-four percent of the tracer is directly excreted through the kidneys, resulting in a smaller radiation burden and better background clearance than Tc-99m albumin.[8] Since it does not bind to plasma proteins, stays in the blood pool and does not leak through normal intestinal mucosa,[7] it is regarded as an ideal agent for protein losing enteropathy imaging.

We have used this agent to detect the site of protein loss in a 16-year-old male patient with peripheral edema, hypoalbuminemia and abdominal pain, and it showed where the intestinal protein loss occurred. In conclusion, Tc-99m dextran is useful in the detection of protein losing enteropathy which is not detected by extensive radiological or endoscopic examination.

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