exceeding 200 rads, the administration of more than 300 mCi or the retention of more than 150 mCi at 48 hr.

With intense ^{131}I uptake throughout the marrow due to diffuse metastatic infiltration, the usual dosimetric assumptions clearly do not apply in our patient. As can be seen from the ^{131}I whole-body scan, the bone marrow scan and the histology of the marrow biopsy, there is ^{131}I-avid thyroid cancer intimately mixed with the red hematopoietic marrow throughout the entire normal marrow space. Thus, the red marrow radiation exposure would include that from beta irradiation from ^{131}I in perfusing the blood and in contact with the marrow, gamma irradiation from focal distant metastatic tumor deposits (e.g., skull, vertebra, humerus) and, most importantly, tumor present in and in direct contact with the marrow. The exact radiation dose to the bone marrow in these circumstances cannot be calculated but is clearly far greater than that normally encountered.

The patient received modest doses of ^{131}I for distant metastatic thyroid cancer (150 and 92 mCi) but developed significant myelosuppression which would not normally be anticipated even if ^{131}I labeled thyroid hormones were synthesized in substantial quantities. The myelosuppression resulted, at least in part, from the intense, specific uptake of ^{131}I by thyroid cancer intimately admixed with the bone marrow. The tumorous infiltration of the bone marrow may have also caused part of the myelosuppression, as is suggested by the abnormal hemogram prior to ^{131}I therapy.

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Pleuroperitoneal Migration of Intraperitoneal Phosphorus-32-Chromic Phosphate Therapy for Stage I Ovarian Carcinoma

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A patient with postoperative Stage I ovarian carcinoma received 15 mCi of ^{32}P-chromic phosphate suspension in normal saline intraperitoneally as part of her therapy. The following day, a portion of the infused radiopharmaceutical and normal saline had passed transdiaphragmatically into the patient's right pleural cavity. Thoracentesis removed as much fluid as possible and this fluid contained radioactive material. In the ensuing 4 yr, the patient has not manifested any detectable pleural or pulmonary abnormalities attributable to the radioactivity. Retrospective review of 100 consecutive patients receiving ^{32}P-chromic phosphate intraperitoneal therapy resulted in 43 patients in whom the hemithoraces could be evaluated scintigraphically. Three of the 43 patients (7%) had right pleural fluid radioactivity. This is similar to the percentages reported in patients with cirrhosis with ascites in whom hepatic hydrothorax is identified.

Key Words: ovarian carcinoma; phosphorus-32-phosphate pleural radioactivity; radiopharmaceutical therapy

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The unexpected finding of ^{32}P-chromic phosphate suspension within the right pleural cavity 24 hr after therapeutic intraperitoneal instillation of the radiopharmaceutical is reported. This led to close observation of this patient and the subsequent review of our similarly treated patient population about the frequency of this phenomenon.

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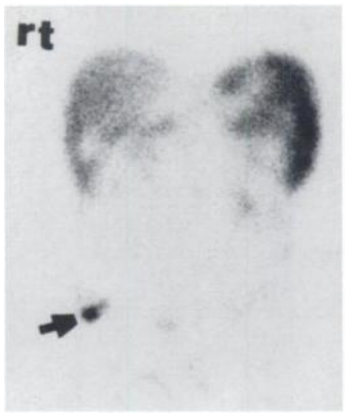

FIGURE 1. Pretreatment image with 1 mCi ^{99m}Tc-sulfur colloid and 600 ml normal saline in the peritoneal cavity. Arrow indicates injection site. Note absence of tracer in the thorax.

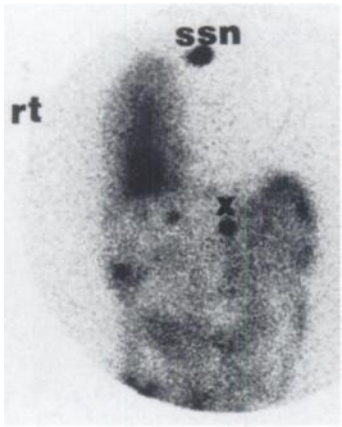

FIGURE 2. Twenty-four hours post-^{32}P-chromic phosphate intraperitoneal therapy, bremsstrahlung radiation imaged. Note radiopharmaceutical in right pleural cavity. The "X" is at the xiphoid. "SSN" is at the suprasternal notch.

CASE REPORT

A 56-yr-old woman with Stage I ovarian carcinoma underwent a hysterectomy, bilateral oophorectomy, salpingectomy and omentectomy. It was elected to treat her with intraperitoneal ^{32}P-chromic phosphate suspension. The most recent chest radiographs obtained prior to the radiopharmaceutical therapy were taken 2 days postoperatively (4 days pretherapy). The chest radiographs revealed small bilateral pleural effusions, left greater than right, and clear lungs. On the day of the treatment and before therapeutic radiopharmaceutical administration, scintillation camera imaging following the intraperitoneal instillation of 1.1 mCi of ^{99m}Tc-sulfur colloid and 600 ml of normal saline established satisfactory distribution (Fig. 1). The patient received 15 mCi of ^{32}P-chromic phosphate suspension mixed in 250 ml of normal saline, and following this, an additional 100 ml of normal saline. The patient was returned to her room on a routine schedule of periodic position changes and directed to the nuclear medicine imaging area approximately 24 hr later for routine post-treatment abdominal imaging using the bremsstrahlung radiation for delineating the therapeutic radiopharmaceutical distribution. The initial abdominal image incorporated the lower one-third of the thorax. Tracer activity within the right hemithorax as well as the peritoneal cavity was noted. Images of the thorax revealed the radiopharmaceutical to be within the right pleural cavity (Fig. 2). Upright and recumbent radiographs confirmed the presence of fluid within the right pleural cavity (Fig. 3). It was decided to perform a thoracentesis using ultrasonographic guidance. Approximately 250 ml of fluid was removed. Post-thoracentesis radiographs revealed only minimal residual pleural fluid. It was estimated that there was approximately 1.9 mCi of ^{32}P-chromic phosphate in the fluid which was removed. The patient was encouraged to remain in the upright or semiupright position.

There have not been any clinical or laboratory findings to indicate the presence of liver disease (e.g., cirrhosis) or other possible causes for ascites. The patient has been carefully monitored for more than 4 yr and there have been no clinical or radiographic findings suggestive of pulmonary or pleural abnormalities.

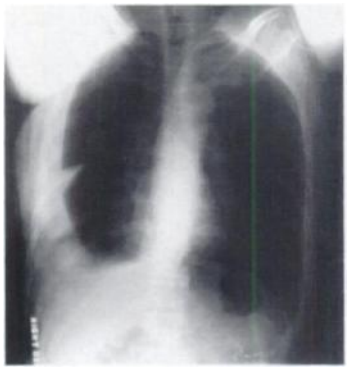

FIGURE 3. Right decubitus chest radiograph demonstrating pleural fluid.

Intraperitoneal Phosphorus-32 Therapy

Our procedure is essentially as described in the Case Report.

We usually attain a liter of intraperitoneal fluid incorporating the radiopharmaceutical. The patient was encouraged to alter their position for several hours post-therapy to enhance distribution of the radiopharmaceutical throughout the peritoneal cavity. Twenty-four hr later, the patient is imaged to determine ^{32}P-chromic phosphate distribution as reflected by the bremsstrahlung radiation. We use an image acquisition technique similar but not identical to that reported by Kaplan et al. (*1*); either technique appears satisfactory. We, however, use a large field of view single detector scintillation camera. Pulse-height analyzers were centered at 120 keV with a 50% window; Kaplan et al. (*1*) centered the analyzers at 81 keV with a 90% window. We obtain 300,000-500,000 counts per anterior abdominal image. Kaplan et al. (*2*) found that the proximity of their 81-keV peak to the 78 keV lead x-ray did not appreciably compromise image resolution. They also reported significant image degradation from 200 keV to 500 keV energy levels (*1*). We use either low- or medium-energy collimators. Modification of this technique may be dictated by the imaging instrument used.

We use 24-hr images to help determine whether there was loculation of ^{32}P-chromic phosphate not evident on the pretherapy radiotracer peritoneal distribution image. If there is loculation, subsequent patient follow-up should look for developing obstruction or perforation of the bowel. This is of particular importance in those patients also receiving external beam radiation therapy.

To determine the frequency of fluid transfer between the peritoneal and pleural space in this clinical setting, the nuclear medicine film folders of 100 consecutive patients receiving intraperitoneal ^{32}P-chromic phosphate suspension therapy between August 1984 and February 1995 were retrospectively reviewed. We reviewed all pretherapy ^{99m}Tc-sulfur colloid images and bremsstrahlung images obtained approximately 24 hr post-therapy. The images were evaluated for sufficient thoracic incorporation to determine the presence or absence of tracer in pleural fluid. Of the 100 patients reviewed, 15 did not have 24-hr ^{32}P post-therapy images. These exceptions to the protocol were generally at the request of a referring physician. Of the remaining 85 patients, 43 (47%) had sufficient thoracic incorporation on the image to warrant inclusion in the analysis. Of those 43 patients, two patients demonstrated pleural fluid with radioactivity on the 24-hr image and one patient demonstrated tracer in pleural fluid on the initial pretherapy ^{99m}Tc-sulfur colloid images. Thus, 3 of the 43 patients (7%) with thoracic regions in the field of view had tracer in the pleural fluid. A review of our teaching files, which extend to about

1978, revealed one additional case of post-therapy pleural fluid tracer.

DISCUSSION

No chest radiographs were obtained immediately before radiopharmaceutical therapy in this patient. There were, however, small bilateral pleural effusions, left greater than right, on the chest radiographs obtained 2 days postoperatively and 4 days before therapy. Although it is possible that the right pleural fluid present on the post-therapy radiographs reflected an increase in the previous pleural effusion, it is difficult to explain the activity in the right pleural fluid unless there was a pleuro-peritoneal communication. It is highly unlikely that there was sufficient absorption of the radiopharmaceutical or of unbound radionuclide from the peritoneal cavity to result in the degree of activity in the right hemithorax when compared with peritoneal cavity tracer activity.

Intraperitoneal treatment of Stage I and II ovarian carcinoma using ^{32}P-chromic phosphate suspension has been used for about four decades. Colloidal ^{198}Au was initially used, but ^{32}P-chromic phosphate was advocated because it is a pure beta emitter, whereas colloidal ^{198}Au is both a gamma and beta emitter. While the 412-keV gamma emission from ^{198}Au permits imaging, it also poses personnel radiation exposure problems. An objection to the use of ^{32}P-chromic phosphate is that it is a suspension rather than a true colloid, which makes heterogeneous distribution more likely (*1,2*). When compared with earlier reports using colloidal ^{198}Au, clinical series using ^{32}P-chromic phosphate have shown comparably improved survival in patients with earlier stages of ovarian cancer and in second-look laparotomy patients with minimal or no evidence of residual disease (*3–11*). Colloidal ^{198}Au is reported to have a higher complication rate than does ^{32}P-chromic phosphate (*9*).

The use of a small (≤250 ml) versus large volume of intraperitoneally instilled fluid with the therapeutic tracer has its advocates. We favor the use of about one liter of normal saline with the therapeutic dose. Other investigators have had similar experiences. For example, Tulchinsky and Eggli (*12*) reported their findings on a potentially false-negative interpretation of fluid loculation using a small volume of fluid. The large volume of intraperitoneal fluid was a factor in the transdiaphragmatic passage of tracer.

Although not a clinical consideration in our patient, a similar image pattern was seen in hepatic hydrothorax after intraperitoneal introduction of a tracer (usually ^{99m}Tc-sulfur colloid), and subsequent thoracic and abdominal imaging.

Usually seen in the right pleural cavity, hepatic hydrothorax is occasionally bilateral and on rare occasions solely in the left pleural cavity. This phenomenon in patients with cirrhosis is seen with detectable ascites but occasionally without detectable ascites, presumably due to rapid passage of tracer from the peritoneal to the pleural cavity (*13,14*). The most common postulated mechanisms for ascites passage in the pleural cavity have been: (a) anatomic presence of diaghragmatic defects and/or (b) drainage through the diaphragmatic lymphatics (*15–17*). Lieberman et al. (*17*) rejected transdiaphragmatic lymphatic passage of ascites is the primary mechanism for this phenomenon. A pneumoperitoneum procedure strongly suggested that functional diaphragmatic defects are the conduit through which ascites flow into the pleural cavity. Autopsy in two patients revealed millimeter size fenestrations in the tendinous aspects of the diaphragm and mesothelial limed bleb formation at a diaphragmatic defect in one. It was postulated that the increased intra-abdominal pressure from the ascites encouraged this ascitic flow (*17*). Rubinstein et al. (*18*) studied two patients with hepatic hydrothorax without detectable abdominal ascites and found no evidence for flow of the tracer back into the peritoneal cavity once it was in the pleural cavity. Thus, normal tendinous fenestrations in the diaphragm would be a reasonable explanation for the finding in our patient. Although we, as well as others, have shown lymphatic passage of intraperitoneal radiotracer into mediastinal lymph nodes, this is unlikely to be the primary mechanism (*19*). As noted earlier, 3 of the 43 patients (7%) with thoracic regions imaged demonstrated right pleural fluid with radioactivity. Interestingly, although perhaps not surprisingly, this 7% figure is similar to the 5.5% incidence of ascitic hydrothorax in 330 consecutive cases of cirrhosis reported by Lieberman et al. (*17*), given the presumably congenital nature of diaphragmatic defects in most patients.

One may question if radioactivity in the pleural cavity is potentially beneficial or detrimental. Kaplan et al. (*2*) postulated that there may be some benefit due to potential passage of malignant cells through the same lymphatic drainage route. There can be dissemination of tumor cells in the peritoneal cavity when an ovarian carcinoma cyst bursts during surgery and the passage of fluid into the pleural cavity may be analogous. Fortunately, our patient has not manifested signs of pleural seeding of tumor to date. One might postulate that the presence of the ^{32}P-chromic phosphate in the pleural fluid reduced this risk. Because there is a potential route for malignant cell passage into the pleural cavity, treatment of this space might be desirable. It may be argued that early removal of the fluid might be undesirable if tumor cells are introduced into the pleural cavity because of insufficient time for absorption of ^{32}P-chromic phosphate on the seeded pleural surface. Deposition of most of the suspension on the pleural surface reportedly occurs within the first 24 hr. Conversely, localization of the radiopharmaceutical within the pleural cavity could raise the hypothetical concern of long-term formation of pleural adhesions or conceivably radiation changes to the lung tissues adjacent to the pulmonary pleura. Such changes have not been detected in the three patients in the series. In the past, either this radiopharmaceutical or colloidal ^{198}Au was used in the treatment of otherwise uncontrolled malignant pleural effusions, but the control was transient and solely palliative. The long-term therapeutic effects have not been well characterized.

CONCLUSION

The most likely mechanism for saline and ^{32}P-chromic phosphate passage into the right pleural cavity is transit of fluid from the peritoneal cavity into the pleural cavity through diaphragmatic tendinous fenestrations. While uncommon, the potential for this phenomenon should be kept in mind by those administering this treatment. Although not evident in our patient, transdiaphragmatic flow of fluid may be detectable on the pretherapy distribution image and was found in one of our patients with right pleural fluid tracer activity. Whether it is deleterious, inconsequential or potentially beneficial to have the intraperitoneal therapeutic ^{32}P-chromic phosphate suspension migrate into the pleural cavity is not established. To date, there are no demonstrable untoward effects directly attributable to this phenomenon in the three patients after limited temporal follow-up.

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Intestinal Leakage of Technetium-99m-MDP in Primary Intestinal Lymphangiectasia

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We present a case in which a patient with primary intestinal lymphangiectasia demonstrated abnormal intestinal accumulation of tracer during ^{99m}Tc-methylene diphosphonate (MDP) skeletal scintigraphy. Early intestinal leakage with gradual colonic migration and concentration was confirmed by repeat bone scan with serial acquisitions. The mechanism for the intestinal localization of ^{99m}Tc-MDP seen in this patient is not clear. Thus, intestinal lymphangiectasia can be a cause for extra-osseous localization of bone scan agents in the intestine.

Key Words: intestinal lymphangiectasia; technetium-99m-MDP

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Intestinal lymphangiectasia is characterized by a generalized disorder of the lymphatic channels causing dilated intestinal lymphatics, enteric protein loss, edema, hypoalbuminemia and lymphopenia (*1*). It is usually diagnosed on the basis of a characteristic small bowel mucosal histology along with methods demonstrating enteric protein loss (*2*). We report a patient with primary intestinal lymphangiectasia in whom abnormal intestinal leakage of ^{99m}Tc-methylene diphosphonate was demonstrated unexpectantly during bone scintigraphy.

CASE REPORT

A 23-yr-old woman presented with generalized edema, recurrent tetanic attacks, multiple bone pain, tingling sensation and diarrhea. She had experienced episodes of generalized edema and easy fatigue that had waxed and waned for 2 yr before admission. Physical examination revealed pitting edema of both legs and sclerotic degenerative fingernails. Initial laboratory tests showed severe hypoalbuminemia (15 g/liter), hypocalcemia (total calcium 1.2 mmole/liter; ionized calcium 0.15 mmole/liter), hypokalemia (3.7 mmole/liter), lymphopenia (400/mm^3), increased serum alkaline phosphatase level (316 IU/liter) and an abnormally elevated α_1-antitrypsin clearance rate (249 ml/day). Renal and liver function tests were normal. A small bowel series disclosed diffuse wall thickening of the small intestine, while computed tomography showed no specific abnormalities. Bone scintigraphy with ^{99m}Tc-methylene diphosphonate (MDP) was performed to evaluate multiple bone pain. The bone scan showed diffuse increased skeletal uptake with a focal tibial lesion, poor soft-tissue and renal activity and an unexpected abnormal accumulation of activity in the upper abdomen conforming to the transverse colon (Fig. 1).

The general pattern of tracer distribution that was seen in the patient was compatible with metabolic bone change due to secondary hyperparathyroidism from hypocalcemia, which was attributed to malabsorption. Since the abdominal activity could not be explained, a bone scan was repeated 17 days later with serial images of 30-min intervals. In the repeat bone scan, early images demonstrated diffuse abdominal activity in the small intestinal region. The delayed images confirmed gradual colonic accumulation of the activity (Fig. 2). Lymphoscintigraphy with ^{99m}Tc-antimony colloid disclosed dilated lymphatic channels in the lower extremities and abnormal abdominal tracer activity that later localized in the intestinal region (Fig. 3). The diagnosis of primary intestinal lymphangiectasia was established by clinical findings, laboratory results and pathology from intestinal biopsy (Fig. 4).

DISCUSSION

Primary intestinal lymphangiectasia is a relatively uncommon entity in which congenital central or peripheral lymphatic dysplasia produces functional lymphatic obstruction (*1*). The disease is characterized by dilated intestinal submucosal and subserosal lymphatics, protein losing enteropathy, hypoalbuminemia, hypoproteinemic edema and lymphopenia. Excessive enteric loss of proteins has been shown to be the cause for hypoalbuminemia in this disease (*2*).

In this patient, hypoalbuminemia could be attributed to

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