The diagnosis and localization of parathyroid adenomas is still a challenging problem. Various techniques have been used in the past to localize parathyroid adenomas with \(^{75}\text{Se}\)-selenomethionine (1,2) and selective venous catheterization and radio-immunoassay (3). All these techniques were partially successful but were difficult to perform, and the search continued for an easy method for localizing the parathyroid adenomas.

We have succeeded in localizing the parathyroid adenomas with the use of \(^{75}\text{Se}\)-selenomethionine and the scintillation camera after glucagon stimulation in a simple and high yielding technique.

**METHOD**

The patient (BM), a 48-year-old negro female with hypercalcemia, was admitted to Jackson Memorial Hospital, Miami, Florida, for workup and treatment. She was first admitted to the gynecology service in August 1969 where she was found to have an elevated serum calcium of 12.4 mg\%. She was readmitted to the hospital in April 1970 with acute onset of right hemiparesis and was found to have a left basal ganglion hematoma. During this admission a workup for hypercalcemia was undergone. An immunoelectrophoresis showed a monoclonal peak. This finding suggested that a gammopathy associated with hypercalcemia was the explanation for this patient's clinical picture, but hyperparathyroidism could not be ruled out and she was studied accordingly. The patient was discharged for rehabilitation of her cerebral vascular accident on an outpatient basis and was finally readmitted to the hospital on March 1, 1971, for final testing and treatment. The patient always denied any symptoms related to hypercalcemia and her past medical history did not include any other findings related to it.

**Physical examination.** At the time of admission the patient had a blood pressure of 120/80 mmHg, a pulse rate of 74 per min, and a weight of 148 lb. She was afebrile. Respiration was within normal limits. Her general appearance was that of a 48-year-old negro female with a right hemiplegia in no acute distress. Head, eyes, ears, nose, and throat examination was unremarkable, other than for neurological findings. Neck was soft, trachea in midline. There were no lymphadenopathies. Thyroid was palpable without nodules. Lungs were clear to percussion and auscultation. The heart had a normal sinus rhythm—S1 normal, S2 normal with Grade II/VI systolic ejection murmur at the base without gallops. Abdomen: liver was palpable two finger-breathths below the costal margin; no splenomegaly. Extremities revealed 1+ ankle edema. Neurological examination revealed right facial weakness and right hemiparesis with spasticity and a positive Babinski on the right. Roentgenograms showed no changes compatible with hyperparathyroidism or multiple myeloma.

**Laboratory data.** Her representative CBC showed a hemoglobin of 12.6, hematocrit 37.5, reticulocyte count 0.5\%, WBC 4,750, segs 67, stabs 1, lymphs 20, monos 9, eosinophils 3, and platelet count 263,000. Slight anisocytosis was reported. Urinalysis showed 20–30 WBC and many bacteria. Urine culture and sensitivity showed growth of Proteus species and Klebsiella, and the patient was treated for a urinary tract infection.

The patient's biochemical profile showed all values within normal limits, except for hypercalcemia. The alkaline phosphatase was minimally elevated. An immunoelectrophoresis showed a monoclonal peak. A bone marrow aspiration showed relative erythroid hyperplasia and plasmocytosis. The VDRL determination was nonrelative. Some of the patient's sequential determinations of calcium and phosphorus

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in the serum and urine, the tubular reabsorption of phosphorus test and the steroid suppression test results are shown in Table 1.

**Radioactive and nonradioactive pharmaceuticals.** Normal thyroid metabolic activity of the patient was suppressed by administration of 15 drops of SSKI and 400 mg potassium perchlorate by mouth 30 min before intravenous injection of 250 μCi of $^{75}$Se-L-selenomethionine (E. R. Squibb & Sons), which was followed by the injection of 2 mg of glucagon intramuscularly (E. Lilly and Co.).

**Scanning.** Images of neck and mediastinum were obtained using the Anger camera (Nuclear-Chicago Pho/Gamma III) 30 min, 1 hr, and 2 hr postinjection. The 4,000-hole diverging collimator was used and 30,000–40,000 counts were collected per image. Serum calcium was drawn before the glucagon injection and at 1 and 2 hr postinjection to monitor the calcium changes.

### Table 1. Laboratory Data on the Patient with Hyperparathyroidism Before and After Surgery

<table>
<thead>
<tr>
<th>Date</th>
<th>Serum calcium (mg%)</th>
<th>Serum phosphorus (mg%)</th>
<th>Alkaline phosphatase (KAU)</th>
<th>24-hr urine calcium (mg%)</th>
<th>24-hr urine phosphorus (mg%)</th>
<th>Other tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>8–69</td>
<td>12.4</td>
<td>2.1</td>
<td>13</td>
<td>11.4</td>
<td>17</td>
<td>Tubular reabsorption of phosphorus: 62%</td>
</tr>
<tr>
<td>6–70</td>
<td>13.7</td>
<td>2.4</td>
<td>20</td>
<td>17</td>
<td></td>
<td>Steroid suppression test: (40 mg every 8 hr for 10 days of hydrocortisone) resulted in no change in serum calcium.</td>
</tr>
<tr>
<td>3–71</td>
<td>13.3</td>
<td>1.9</td>
<td>19</td>
<td>13.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4–1–71</td>
<td>9.9</td>
<td>1.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4–3–71</td>
<td>9.5</td>
<td>2.7</td>
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</tr>
<tr>
<td>4–6–71</td>
<td>8.9</td>
<td>3.7</td>
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</tbody>
</table>

**RESULTS**

Scintillation scans and camera pictures (Fig. 1) revealed the concentration of $^{75}$Se in the area of the inferior right pole of the thyroid at 1 and 2 hr postinjection of the radiopharmaceutical. This same finding was shown on a repeat scan 1 month later. Serial calcium levels were 12.5 mg% before the glucagon injection; 11.1 mg% in 1 hr; and 11.5 mg% in 2 hr post glucagon stimulation. After the workup was completed, a neck exploration was performed on April 1, 1971. A parathyroid adenoma was found in the right inferior pole of the thyroid.

Microscopic sections of the adenoma showed cells with small, dark, round, uniform nuclei and clear or eosinophilic cytoplasm. A fibrous capsule was noted. The patient did well postoperatively, and her calcium level dropped to normal levels with no evidence of tetany.

**FIG. 1.** Scintigrams of neck showing concentration of $^{75}$Se in area of inferior right pole of thyroid at 1 and 2 hr postinjection of radiopharmaceutical.

**COMMENT**

The camera images at 1 and 2 hr clearly showed the parathyroid adenoma in the inferior right pole of the thyroid in this patient. In eight out of ten hypercalcemic patients with suspected hyperparathyroidism we were able to accurately localize the position of parathyroid adenomas in the neck and chest. The mean drop in serum calcium in these patients following the use of 2 mg glucagon was 1.2 mg in 1 hr and 1.0 mg in 2 hr. Glucagon in the doses used appears to lower serum calcium transiently by its effect on calcitonin release (4). The resulting hypocalcemia was an effective stimulant to parathormone release and incorporation of $^{75}$Se-L-selenomethionine, resulting in better parathyroid visualization. No morbidity was noted in this procedure, except for transient hyperglycemia after the use of glucagon. The addition of glucagon stimulation to $^{75}$Se-L-selenomethionine parathyroid scanning resulted in better localization of parathyroid adenomas.
REFERENCES


