LETTERS TO THE EDITOR

Disappearance of a Hyperfunctioning Thyroid Nodule

The case report by Kammer and Loveless (/), describing the disappearance of a hyperfunctioning thyroid adenoma following TSH stimulation, is of considerable interest. Recently we had the opportunity to evaluate and treat a patient with a toxic autonomous nodule that disappeared after 4 weeks of therapy with propylthiouracil (PTU). The 24-year-old patient was found to have a prominent thyroid by her general physician. She complained of frequent headaches of 2 months' duration, which had been relieved somewhat by antidepressant medication. She also noted hair loss, easy fatigability upon exertion, intolerance to heat, and increased nervousness.

On physical examination, the thyroid gland was generally enlarged to 1½ times normal size, with a prominent 2-cm nodule palpable in the upper pole of the right lobe. The gland was slightly tender in both lobes, more so on the left. The remainder of the physical examination was essentially normal.

Laboratory tests showed a T4 RIA to be 8.6 µg/dl (normals 5.2-13.0 µg/dl), T3 uptake was 34.7% (normals 22-46%), free thyroxine index was 2.98 (normals 1.72-5.98), and the T3 RIA was 215 ng/dl (normals 110-190 ng/dl). The serum antithyroglobulin antibody was negative by indirect immunofluorescent staining procedure and by indirect hemagglutination test. The 24-hr thyroidal radioiodine uptake was 12%. The thyroid scan (see figure) confirmed the presence of a functioning 2-cm nodule in the upper pole of the right lobe. The nodule was hyperfunctioning relative to the remainder of the gland, which was functionally suppressed.

The patient was started on sodium salicylate because of the tenderness of the gland. Propylthiouracil (PTU) was started because of the clinical and laboratory findings of T3 thyrotoxicosis resulting from a toxic autonomous nodule. TSH stimulation and T3 suppression tests were not performed.

The patient was re-examined after 1 month of this therapy. The 2-cm nodule was completely nonpalpable. The overall size of the gland remained 1½ times normal size to palpation, and was nontender. The patient had improved greatly symptomatically.

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FIG. 1. Rectilinear thyroid scan demonstrates hyperfunctioning nodule in upper pole of right lobe.
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Two possible explanations are suggested for the disappearance of the toxic autonomous nodule during therapy with sodium salicylate and PTU. One is that the nodule was found early in its development, and the inhibitory effects of PTU caused complete regression before formation of fibrous stroma, cystic degeneration and other irreversible changes could occur. Another possibility is that the extranodular, partially suppressed, thyroid tissue was involved with subacute thyroiditis at the time of the initial studies. The right upper-pole nodule may have been the first area undergoing regeneration and recovery from earlier thyroiditis, whereas the suppressed thyroid tissue was still inflamed. A biopsy was not performed. This hypothesized inflammatory process may have been beneficially affected by the sodium salicylate, PTU, or spontaneous remission over the period of a month.

In the article by Kammer and Loveless, we note that a T3 RIA was not reported on the patient's initial workup. Perhaps at that time there was a T3 thyrotoxicosis present. The patient then received an injection of TSH on each of three consecutive days. One week following the TSH injections, the thyroid nodule had shown no change. The patient was then placed on antithyroid drugs and the nodule disappeared. Our cases seem very similar. The autonomous nodule in both patients disappeared while on antithyroid drugs in a period of several weeks. To resolve the above questions, it might be helpful to examine similar cases of early autonomous nodule with needle biopsy of both the autonomous and suppressed regions in a small number of selected cases.

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Bone Scintigraphy: Radiation-induced Cartilaginous Exostosis

The deleterious effects of radiation upon differentiating tissue are recognized. Radiation therapy in children may produce damage in the lungs, intestine, kidney, liver, salivary glands, eye, spinal cord, heart, and bone (1). Damage to bone may result in scoliosis, deformities of the vertebral body and pedicle, necrosis, or pathologic fracture, as well as malignant and benign tumors (2-4). The following case illustrates a radiation-induced cartilaginous exostosis.

A 2-year-old child was first seen in October of 1974. A retinoblastoma of the right eye was found, and the eye was enucleated. Bone scintigraphy, radiographic skeletal survey, and bone-marrow aspiration were normal. In August of 1975, pain developed in the right fibula. Bone scintigraphy, skeletal radiograms, and marrow aspiration revealed a metastasis to the distal right fibula. A total exposure of 3000 R over a 6-wk period was delivered in a 22- by 7-cm field that included the distal femur and the entire tibia and fibula. Figure 1 shows a bone scintigram and radiograph 2 years following radiation therapy. Figure 2 shows an interval study a year later. There is a focal area of increased activity lateral to the distal femoral metaphysis.

Bone abnormalities produced in children who have undergone therapeutic radiation are primarily related to: (a) patient's age, (b) total dose delivered, and (c) area irradiated. In general, the younger the child and the larger the radiation dose, the higher the incidence of bone abnormality. Regional effects differ somewhat: impairment of chondrogenesis with epiphyseal irradiation; abnormal resorption with metaphyseal irradiation; and altered periosteal bone deposition with diaphyseal irradiation. The maximum effect is usually to the epiphysis (5).

In animal studies, it has been proved that benign cartilaginous tumors can be produced by irradiation (6). The mechanism by which radiation produces cartilaginous exostosis in children is not clear. These benign cartilaginous tumors are probably secondary to the associated metaphyseal-diaphyseal growth impairment combined with the aberrant epiphyseal growth pattern. The radiographic and scintigraphic appearances are illustrated in Fig. 2.

To date, 35 such cases have been reported (4), with only radiologic documentation. We have not heard of malignant degeneration of these exostoses despite the 1-5% incidence of spontaneous malignant degeneration of a solitary cartilaginous exostosis (7-10).

In scintigraphic appearance, a radiation-induced exostosis looks like other exostoses (11). Several views may be needed to
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