LETTER TO THE EDITOR

Incidental Demonstration of Hyperthyroidism on Cerebral Blood-Flow Study with Glucoheptonate

A 39-yr-old Hispanic male was admitted to the hospital after having sustained a grand-mal seizure. In the emergency room the patient was disoriented and combative and had a sinus tachycardia of 120. There were no focal neurological findings. Hyper-reflexia with ankle clonus was elicited bilaterally. Cerebrospinal fluid examination was normal, and routine chemistries (which did not include a serum thyroxine) were not remarkable.

The initial impression was that the patient was having an alcohol-withdrawal seizure. On phenobarbital and diphenylhydantoin there were no further seizures. Over the next 2 days the patient became reoriented and cooperative. He later admitted to a 2-mo history of weakness, weight loss, and heat intolerance, but denied any change in appetite or any alcohol or narcotic abuse. Urine toxicology was negative. There was no history of prior seizures.

A brain scan was performed with 20 mCi of technetium-99m gluceptate (glucoheptonate) sodium* and showed significant thyroid uptake by 3 sec, and intense thyroid uptake on the remainder of the flow study and subsequent static images (Fig. 1). This was interpreted as suggesting hyperthyroidism.

An I-131 thyroid scan demonstrated diffuse enlargement of both lobes, consistent with Graves' disease. The 4-hr and 24-hr I-131 uptakes were 89% and 97%. The T_4 was 30.6 μg/dl; T_3 resin uptake 1.67; free-T_4 index 51.1 (normal values: T_4 5.5-14.4 μg/dl; T_3 resin uptake 0.88-1.15; free-T_4 index 4.8-16.1). The patient was treated with propylthiouracil and propranolol. He was discharged with the diagnosis of Graves' disease and seizure of unknown origin, to be followed in Endocrine Clinic.

The uptake of pertechnetate in the thyroid has been proposed as a measure of thyroid function, and an approximate relationship has been established between the 20-min pertechnetate (Tc-99m) thyroidal uptake and the 24-hr I-131 thyroidal uptake, with a correlation coefficient of 0.70 (*1*).

In the reconstituted Tc gluceptate kit, free pertechnetate is present in a concentration of less than 2%, according to the manufacturer. In our case it is unlikely that an excessive amount of free pertechnetate was present, since neither the salivary glands nor the choroid plexus was visualized on the blood-flow study or on the delayed static scans of this patient. In addition, on the same day the identical batch of gluceptate was used to perform a brain scan on another patient, and there was no uptake of tracer in the thyroid, salivary glands, or choroid plexus.

While thyroid trapping of pertechnetate is well understood, similar evidence for trapping of the gluceptate molecule is not known to the manufacturer. However, a recent review of the chemistry and tissue distribution of Tc glucoheptonate in rats showed that it is concentrated in the thyroid at 1 hr after injection (*2*).

The thyroid imaging in our case may therefore depend on extremely rapid trapping of the gluceptate and/or the pertechnetate molecule by the gland.

The observation of early and intense thyroid imaging during a cerebral blood-flow study serves principally as a reminder that thyrotoxicosis may masquerade as central nervous system disease, and that serendipity may play a useful role in medical imaging. We are not aware of a previous report of thyrotoxicosis first suspected on the basis of rapid thyroidal uptake during a cerebral blood-flow study.

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FOOTNOTE

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REFERENCES

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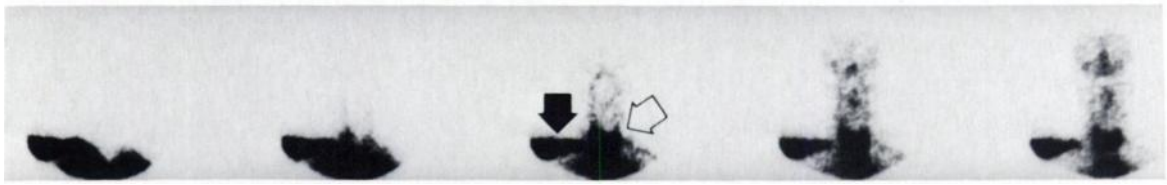

FIG. 1. Cerebral blood-flow study with Tc gluceptate. Images were taken at 1.5-sec intervals. Subclavian vein (black arrow) and thyroid gland (open arrow) are identified.