Year 2003 Paper two: Questions supplied by Tricia

Question 79

The arrow points to sternal notch marker

The above thyroid technetium scintiscan is least consistent with thyrotoxicosis due to which one of the following clinical conditions?

A. Subacute thyroiditis
B. Thyroxine therapy
C. Amiodarone therapy
D. Graves' disease
E. Postpartum thyroiditis

The scan looks to me like it is not taking up any iodine except in the marker, suggesting a low uptake of iodine. I thinks its the wrong test also as it is generally used to evaluate a nodule.

Thyroid scan

The thyroid scan uses a small amount of a radioactive substance, usually radioactive iodine, to obtain a picture of the thyroid gland. Because thyroid cancer cells do not take up radioactive iodine as easily as normal thyroid cells do, this test is used to determine the likelihood that a thyroid nodule contains a cancer. If done as the first test, the thyroid scan is used to determine those patients who most need a biopsy. The scan usually gives the following results.

1. The nodule is cold. In other words, the nodule is not taking up radioactive iodine normally. This patient is referred for a fine needle biopsy of the nodule.

2. The nodule is functioning. Its uptake of radioactive iodine is similar to that of normal cells. A biopsy is not needed right away since the likelihood of cancer is very low.

3. The nodule is hot. Its uptake of radioactive iodine is greater than that of normal cells. The likelihood of cancer is extremely rare, and so biopsy is usually not necessary.
If the fine needle biopsy was done as the first test, then a scan is usually ordered to evaluate a suspicious biopsy result. In this case, patients with a "cold" nodule result should have their nodule removed. Patients with "functioning" or "hot" nodules on a scan and a suspicious biopsy can be watched, and surgery is not immediately necessary.

Subacute thyroiditis

**Thyroiditis may cause transient thyrotoxicosis, with a characteristic low or undetectable thyroid radioiodine uptake.** Painless lymphocytic thyroiditis occurs in up to 10% of women after giving birth. This is an inflammatory autoimmune disorder in which lymphocytic infiltration results in thyroid destruction and leads to transient mild thyrotoxicosis as thyroid hormone stores are released from the damaged thyroid. As the gland becomes depleted of thyroid hormone, progression to hypothyroidism occurs. Thyroid function returns to normal within 12-18 months in 80% of patients.

Painful subacute thyroiditis, the most common cause of thyroid pain, is a self limiting inflammatory disorder of possible viral aetiology. Patients typically present acutely with fever and severe neck pain or swelling, or both. About half will describe symptoms of thyrotoxicosis. After several weeks of thyrotoxicosis, most patients will develop hypothyroidism, similar to postpartum thyroiditis. Thyroid function eventually returns to normal in almost all patients. The hallmark of the laboratory evaluation of painful subacute thyroiditis is a markedly elevated erythrocyte sedimentation rate and C reactive protein.

**Thyroxine therapy**
Excess exogenous thyroid hormone is often associated with thyrotoxicosis. This may be iatrogenic, either intentional, when TSH suppressive doses of thyroid hormone are prescribed to suppress the growth of thyroid cancer or decrease the size, or unintentional, when overly vigorous treatment with thyroid hormone is prescribed for hypothyroidism. Thyrotoxicosis factitia may also result from patients' surreptitious use of thyroid hormones or from inadvertent ingestion. Serum thyroglobulin values are low to undetectable in thyrotoxicosis factitia but are raised in all other causes of thyrotoxicosis.

**Amiodarone therapy**
- Amiodarone may cause both hypothyroidism (AIH) and thyrotoxicosis (AIT1 and 2)
- AIT1 iodine excess
- AIT2 destructive thyroiditis
- Clinically a combination of AIT1 and 2 often occurs

Amiodarone induced thyrotoxicosis (AIT)

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<thead>
<tr>
<th></th>
<th>AIT1</th>
<th>AIT2</th>
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<tbody>
<tr>
<td>Thyroid</td>
<td>Goiter +</td>
<td>Normal</td>
</tr>
<tr>
<td>Ultrasound imaging</td>
<td>MNG/diffuse goiter</td>
<td>normal</td>
</tr>
<tr>
<td>Colour flow Doppler scan</td>
<td>Normal or increased flow</td>
<td>Decreased flow</td>
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<td>Thyroid antibodies</td>
<td>Absent/present</td>
<td>Absent generally</td>
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<tr>
<td>Interleukin 6</td>
<td>Normal or high</td>
<td>Very high</td>
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<tr>
<td>RAI uptake</td>
<td>Low or normal</td>
<td>Very low / absent</td>
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<tr>
<td>Preferred treatment</td>
<td>Carbimazole/PTU potassium perchlorate</td>
<td>glucocorticoids</td>
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**Graves’ disease**
Graves’ disease is an autoimmune disorder in which thyroid stimulating immunoglobulin (TSI) binds to and stimulates the thyroid stimulating hormone (TSH) receptor on the thyroid cell membrane, resulting in excessive synthesis and secretion of thyroid hormone. Patients with Graves’ disease usually have diffuse, nontender, symmetrical enlargement of the thyroid gland. Ophthalmopathy, consisting of protrusion of the eyes with periorbital soft tissue swelling and inflammation, and inflammatory changes in the extraocular muscles resulting in diplopia and muscle imbalance, is clinically evident in 30% of patients with Graves’ disease

Graves disease is the only choice from the list below of hyperthyroidism with a high radioiodine uptake
Postpartum thyroiditis

Postpartum thyroiditis is a common thyroid disorder that presents during the first postpartum year. It is the occurrence of either transient hyperthyroidism, transient hypothyroidism, or transient hyperthyroidism followed by transient hypothyroidism. Most, but not all, women are euthyroid 1 yr postpartum.

Postpartum thyroiditis is an exacerbation of an underlying autoimmune thyroiditis, aggravated by the immunological rebound that follows the partial immunosuppression of pregnancy.

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Postpartum Thyroiditis Alex Stagnaro-Green
http://jcem.endojournals.org/cgi/content/full/87/9/4042

Hyperthyroidism with a high radioiodine uptake
Autoimmune thyroid disease
Graves’ disease
Hashitoxicosis
Autonomous thyroid tissue (uptake may be low if recent iodine load led to iodine-induced hyperthyroidism)
Toxic adenoma
Toxic multinodular goiter
TSH-mediated hyperthyroidism
TSH-producing pituitary adenoma
Non-neoplastic TSH-mediated hyperthyroidism
Human chorionic gonadotropin-mediated hyperthyroidism
Hyperemesis gravidarum
Trophoblastic disease

Hyperthyroidism with a low radioiodine uptake

Subacute thyroiditis
Subacute granulomatous (de Quervain’s) thyroiditis
Subacute lymphocytic thyroiditis (painless, silent)

Postpartum thyroiditis
Amiodarone (also may cause iodine-induced hyperthyroidism)
Radiation thyroiditis
Palpation thyroiditis
Exogenous thyroid hormone intake

Excessive replacement therapy
Intentional suppressive therapy
Factitious hyperthyroidism
Ectopic hyperthyroidism
Struma ovari
Metastatic follicular thyroid cancer