Clinical Image

Bone Scan in Graves Disease

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CLINICAL IMAGE

43-year-old Hispanic woman was seen in clinic for thyrotoxicosis. She was hospitalized for hyperthyroidism a few months prior to the clinic visit and was started on methimazole and propranolol. She had a moderately enlarged thyroid gland. The biochemical evaluation showed TSH 0.20 uIU/mL (0.34-5.60), Free T4 0.60 ng/dL (0.58-1.64), T3 1.9 ng/mL (0.7-2.0) on methimazole. TSH receptor antibodies were greater than 40.00 IU/L (< =1.75) and Thyroid Stimulating Immunoglobulins were 435 % (< 122). The Alkaline phosphatase was 499 IU/L (32-91), GGT 102 IU/L (7-50), Bone Alkaline Phosphatase 125.6 ug/L (4.5 - 16.9 ug/L), PTH 29.6 pg/mL (12.0-88), Calcium 9.3 mg/dL (8.6-10.2), Glycoprotein 99 mg/dL (2.5-4.5), Urine N-telopeptide 199 nM BCE/mM creatinine (17-94) and Vitamin 25-OHD 17 ng/mL (30-80). A whole body nuclear bone scan was done because of significantly elevated alkaline phosphatase and showed diffuse symmetric increased radiotracer uptake within both distal lower extremities, especially surrounding the knee and ankle joints with distinctive visualization of both tibias.

There were no foci of increased radiotracer activity suggestive of osseous metastatic disease (Figure 1). All the therapeutic options including radioiodine therapy, anti-thyroid medications and thyroidecmy were reviewed with the patient. She wanted total thyroidectomy. The most recent Bone Alkaline Phosphatase decreased to 15.8 ug/L (4.5 - 16.9) nine months after achieving euthyroid status.

DISCUSSION

The Table 1 shows diffuse active osseous remodeling mainly in the distal femur and tibia, axial skeleton which is characteristic of thyroid bone disease [1]. Hyperthyroidism is a well-recognized risk factor for secondary osteoporosis and is associated with increased bone turnover with net bone resorption. The bone involvement can be assessed by the markers of the bone formation like bone alkaline phosphatase and the bone resorption markers like N- and C-terminal cross-linking telopeptides of type-I collagen. Furthermore, whole body scan and Dual-energy X-ray absorptiometry can be used to evaluate bone involvement. The accelerated bone turnover has been shown to be higher in Graves disease than in other causes of thyrotoxicosis [1].

Table 1: TSH and Bone Alkaline Phosphatase levels before and after thyroidectomy.

<table>
<thead>
<tr>
<th>Timeline for thyroid function tests</th>
<th>TSH (0.34-5.60 UI/mL)</th>
<th>Bone Alkaline phosphatase (4.5-16.9 mcg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline on initial evaluation</td>
<td>0.20</td>
<td>125.6</td>
</tr>
<tr>
<td>One month post Thyroidectomy</td>
<td>4.45</td>
<td>-</td>
</tr>
<tr>
<td>Four months post Thyroidectomy</td>
<td>7.71</td>
<td>40.90</td>
</tr>
<tr>
<td>Nine months post Thyroidectomy</td>
<td>3.56</td>
<td>15.8</td>
</tr>
</tbody>
</table>

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Triiodo-L-Thyronine (T3) induced mRNA expression of Receptor Activator of Nuclear factor–B Ligand (RANKL), which results in osteoclast formation, has been implicated in thyroid
bone disease [2]. Furthermore, the thyroxine (T4) prohormone is converted to T3 by the type-2 iodothyronine (d2) which is also expressed in the bone [2]. The osteopenia induced by hyperthyroidism can be reversed after achievement of euthyroid state [3]. The improvement in bone alkaline phosphatase reflects reduced bone turnover after achieving euthyroid state.

REFERENCES