Lipid Cell Tumor of Ovary

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CASE REPORT

Twenty-one-year-old unmarried female came with complaints of (a) amenorrhea since 1 year. Amenorrhea was not preceded by oligomenorrhea or hypomenorrhea, (b) abnormal facial and body hair growth since 1 year, (c) abnormal weight gain and (d) male type baldness since last 6 months. Menstrual history—Menarche was at 14 years, past and present menstrual cycles were regular with average blood loss. Examination—Ht 153 cm, Wt 74 kg, BMI 31.6 kg/m. She had a masculine built with moon face. Male type of baldness was present. There was abnormal hair growth on the face (Fig. 1), breast around the areola, sternum, anterior abdominal wall from the xipisternum till umbilicus and on the legs. Modified Ferriman Gallwey score was 22 (more than 6-8 suggests marked hirsutism). Pulse 84/min, BP 130/80 mmHg. There was no thyroid swelling and systemic examination was normal. Local examination—she had moderate clitoromegaly. On per rectal examination—uterus was normal in size and no mass was palpable in the adnexa. Hence a provisional diagnosis of (1) Cushing’s syndrome (2). Androgen producing ovarian tumor was made.

Investigations:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Patients values</th>
<th>Normal values</th>
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<tbody>
<tr>
<td>FSH</td>
<td>2.11 mIU/ml</td>
<td>2.50-10.20</td>
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<tr>
<td>LH</td>
<td>1.27 mIU/ml</td>
<td>1.90-12.5</td>
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<tr>
<td>Prolactin</td>
<td>7.47 ng/ml</td>
<td>2.8-29.2</td>
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<tr>
<td>Testosterone</td>
<td>5.98 pg/ml</td>
<td>1-2</td>
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<tr>
<td>DHEA-S</td>
<td>269.40 ug/dl</td>
<td>134.4-407.7</td>
</tr>
<tr>
<td>Inhibin</td>
<td>5.72 pg/ml</td>
<td>10-15 (early follicle) 30-40 (mid-late)</td>
</tr>
<tr>
<td>Cortisol</td>
<td>15.53 μg/dl</td>
<td>4.30-22.4</td>
</tr>
<tr>
<td>ACTH</td>
<td>23.20 pg/ml</td>
<td>0-46</td>
</tr>
<tr>
<td>Cortisol after Dexamethasone</td>
<td>0.68 μg/dl</td>
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She had normal Cortisol and ACTH levels. Abnormal DHEA-S levels had ruled out any adrenocortical tumor. Normal Cortisol after dexamethasone suggests no evidence of any pituitary adenoma or ectopic ACTH production. So Cushing’s syndrome was ruled out. So the only possibility was ovarian tumor secreting androgen. As the inhibin levels were low, possibility of Sertoli cell tumor was ruled out. All investigations were in favor of ledig cell or stromal cell tumor. Ultrasonography showed 5 × 6 cm mass arising from left ovary which was solid, other ovary, uterus and rest of abdomen showed normal study. CT scan confirmed the USG findings.

Exploratory laparotomy was done. Left ovary was 6 × 8 cm, solid, pearly white appearance, smooth surface (Fig. 2). Right ovary was normal. Left sided Ovariotomy was performed, frozen section of which revealed sex cord stromal tumor without any evidence of malignancy. Histopathology revealed polygonal...
tumor cells arranged in sheets with clear vacuolated cytoplasm and centrally placed nuclei. Few cells showed granular cytoplasm (Fig. 3).

**Final Diagnosis: Lipid Cell Tumor: Adrenocortical Type**

Postoperative follow-up, she resumed her menstruation on day 37 after surgery. There was marked regression in male type hair distribution (Figs 4 and 5), and there was reduction in weight by 15 kg in 2 months. Her free testosterone levels returned to normal at end of 3 months. Patient is on cosmetic therapy. Follow-up for the last one year showed no recurrence.

**DISCUSSION**

Ovarian steroid cell tumors account for less than 0.1% of all ovarian tumors.1 This term applies to a group of rare tumors which are usually virilizing, characterized by endocrine type architecture and are formed of large oval polygonal cells which resemble lutein, leydig or adrenocortical cells. These tumors are derived from ovarian stromal cells, which differentiate in variety of pathways to produce a range of tumors of common histogenesis. There is controversy over the genesis of lipid cell tumors as to whether they are primarily ovarian or adrenal in origin. Adrenal and ovarian steroid producing cells are derived from common primitive mesenchymal cell. When the stromal cell in the ovary becomes neoplastic they have the potential to function like adrenocortical tissue. Thus ovarian stromal cells have the potential to function like adrenocortical tissue.

They form a subtype of sex-chord stromal tumors. They are usually benign and usually unilateral.1,2 These tumors can originate from any normal steroid hormone producing cell and hence are further divided into three subtypes: stromal leutoma, Leydig cell tumor and Steroid cell tumor (not otherwise specified). Steroid cell tumors account for approximately 60% of these.1,2

These tumors have varied presentation due to their functional nature and secretion of adrenocortical and virilizing hormones. Steroid cell tumors are associated with androgenic changes with variable frequency ranging from 12% to over 50%.1,3 According to study by Taylor and Norris4 they are most often virilizing (77%) and may be associated with Cushing’s syndrome (10%) or estrogenic activity (23%).

Historically these tumors have been referred as Lipid cell tumors, adrenal-like tumors, masculinovoblastomas, and adrenal rest tumors. They are mostly unilateral, a study of 63 cases in Massachusetts Gen Hospital showed 94% unilateral tumors.2 They are usually benign and only 25-45% are clinically malignant.6 Hayes and Scully2 defined five pathologic features suggestive of malignancy: size of 7 cm or more, more than two mitosis per high power field, necrosis, hemorrhage and nuclear atypia.

The primary treatment is surgical. As the frequency of bilateral tumor is only 6%1,2 removal of the affected ovary is
adequate. There are no reports of effective chemotherapy or radiotherapy for these tumors.\textsuperscript{5}

A clinical and pathologic analysis of 30 lipid cell tumors by Taylor et al.\textsuperscript{4} shows that both adrenocortical-like and hilus-like cells are present and in most instances the attempts to separate them objectively is often impossible. These tumors have immunohistochemistry staining positive for gonadotrophin receptors and vimentin.\textsuperscript{5}

This case is presented due to dilemma in diagnosis requiring an array of investigations to come to a conclusion. In a case of secondary amenorrhea with hirsutism a diagnosis of lipid cell tumor should be kept in mind. Prompt surgical management leads to rapid reversal in functional and morphological disfigurement.

REFERENCES
