



**FIGURE 65-3** Diagnostic evaluation for causes of gynecomastia based on measurements of serum human chorionic gonadotropin (HCG), luteinizing hormone (LH), testosterone (T), and estradiol ( $E_2$ ). ↑, Increased; ↓, decreased; CT, computed tomography; MRI, magnetic resonance imaging;  $T_4$ , thyroxine; TSH, thyroid-stimulating hormone. (From Braunstein GD: Gynecomastia, *N Engl J Med* 328:490–495, 1993.)

use; liver, lung, or kidney dysfunction; and signs and symptoms of hypogonadism or hyperthyroidism. If these conditions are not present, only follow-up is required. In contrast, in an adult with recent onset of progressive painful gynecomastia, thyroid, liver, and renal function should be determined. If test results are normal, serum concentrations of HCG, LH, testosterone, and estradiol should be measured. Further evaluation should be carried out according to the schema outlined in [Figure 65-3](#).

Removal of the offending drug or correction of the underlying condition causing the gynecomastia may result in regression of the breast glandular tissue. If the gynecomastia persists, a trial of antiestrogens (e.g., tamoxifen) may be given for 3 months to see whether regression occurs. Gynecomastia that has been present for longer than 1 year usually contains a fibrotic component that does not respond to medications. In these cases, correction usually requires surgical removal of the tissue.

For a deeper discussion on this topic, please see [Chapter 236, "Reproductive Endocrinology and Infertility,"](#) in *Goldman-Cecil Medicine, 25th Edition*.

#### SUGGESTED READINGS

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