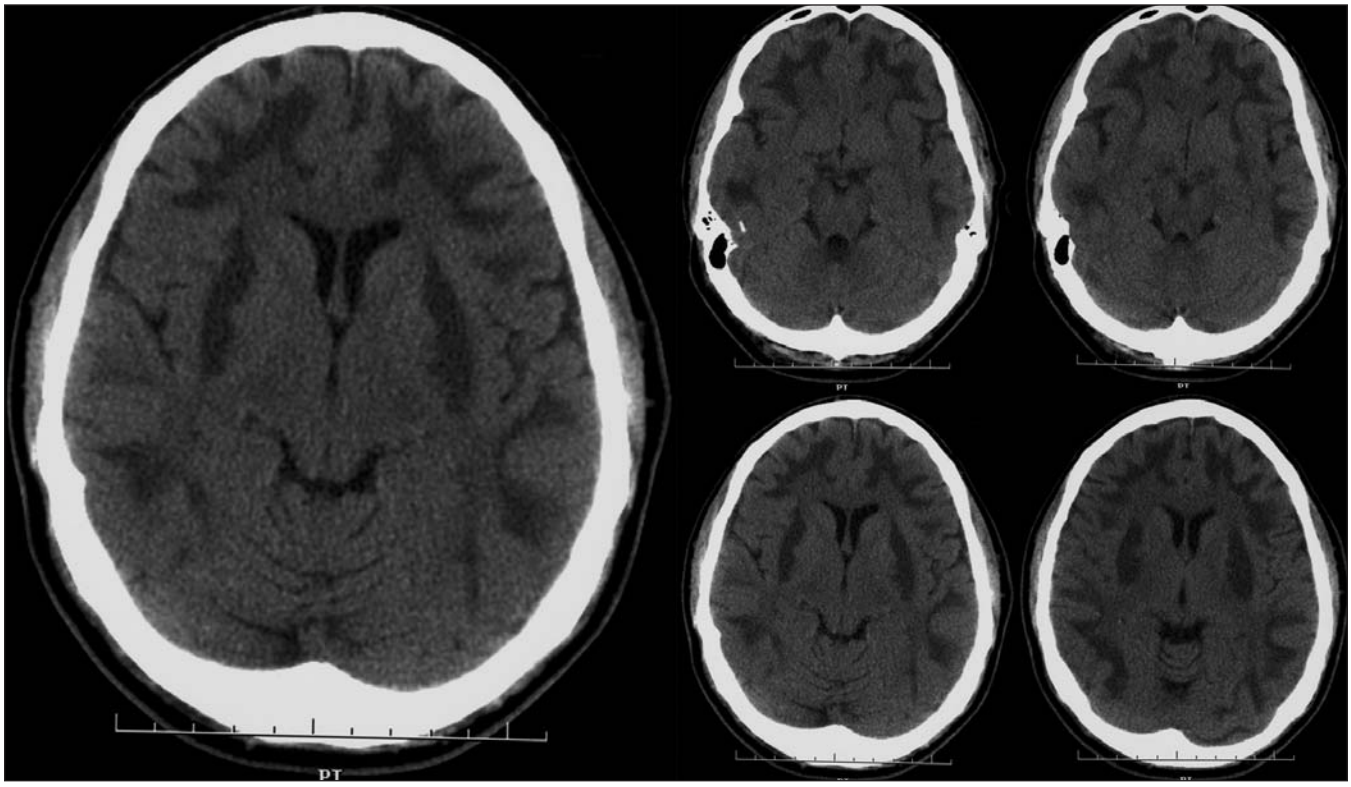
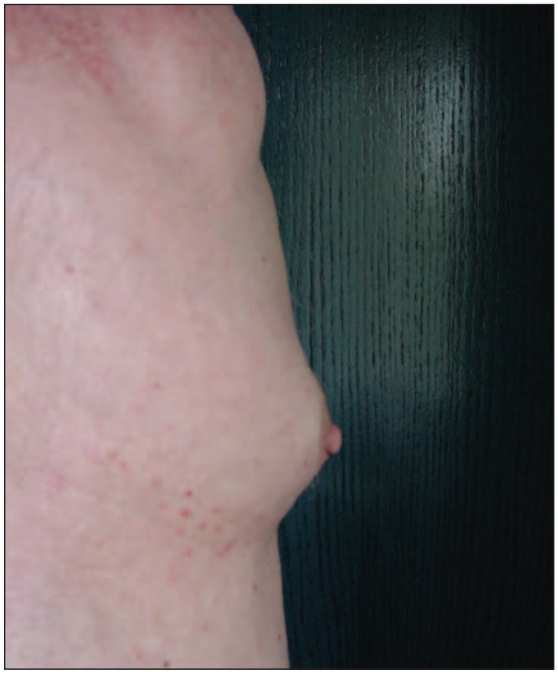
Clinical Vistas Briefs

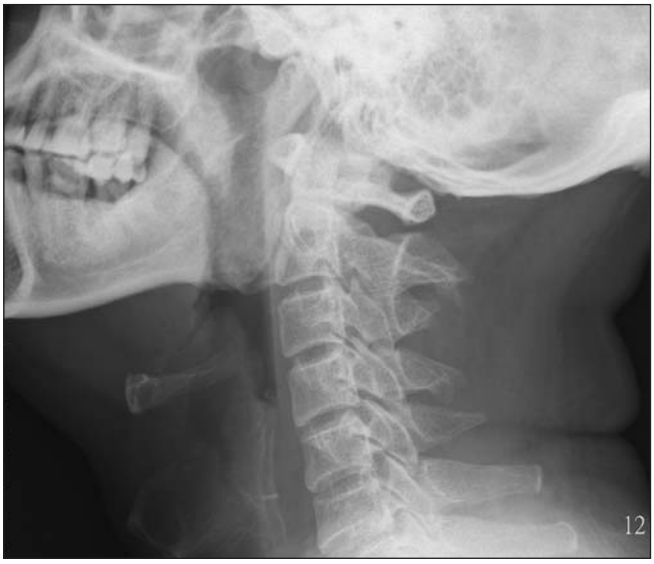
What's your call?



Head CT scans of an unconscious 42-year-old man, taken 48 hours after admission to hospital after an attempted suicide.



Bilateral breast tenderness and enlargement in a 76-year-old man with congestive heart failure.



Soft-tissue radiograph of the neck of a 46-year-old man who presented to the emergency department with a 3-day history of fever, sore throat, dysphagia, odynophagia, drooling and a muffled voice.

See page 623 for diagnoses.

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CT findings in methanol intoxication

The patient had attempted suicide by consuming methanol. On admission, his methanol level was 46 (normal < 2) mmol/L. He was unconscious, with generalized rigidity, exaggerated muscle stretch reflexes, and bilateral clonus and extensor plantar responses. CT scans of the brain obtained 48 hours after admission to hospital showed symmetric areas of low attenuation in the peripheral white matter of both cerebral hemispheres bordered by the grey-white junction, involving both frontal, parietal occipital and temporal lobes (Fig 1, small arrows). Symmetric hypointensities were also noted in both lentiform nuclei, centred within the putamen and extending into the external capsules (Fig. 1, bold arrow).

Mechanical ventilation, hemodialysis and intravenous ethanol therapy were begun, with no significant improvement in clinical status. Nine days after admission, the patient died of respiratory failure after his family decided to withdraw ventilatory support.

Methanol has a toxic effect on the central nervous system, especially the optic nerves and the basal ganglia. Symmetric involvement of the basal ganglia is the most characteristic radiologic feature of methanol poisoning.[1] However, certain other conditions must be kept in mind in the differential diagnosis, such as carbon monoxide intoxication,[2] striatonigral degeneration, anoxic stroke and Wilson's disease.[3] Although bilateral involvement of the basal ganglia on CT scans is a characteristic finding of methanol intoxication, it is infrequently seen in clinical practice.

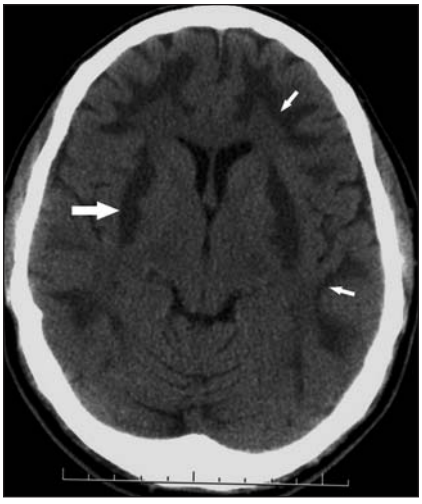

Fig. 1: CT scan showing symmetrical areas of low attenuation in the peripheral white matter (small arrows) and symmetrical hypointesities in both lentiform nuclei (bold arrow).

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Spironolactone-induced gynecomastia

This patient reported bilateral increasing breast size and tenderness. Examination of his breasts revealed gynecomastia (Tanner stage III). The patient had an implantable cardioverter defibrillator for sustained ventricular tachycardias and a history of hypertension and heart failure. His medications included phenprocoumon, enalapril, nebivolol, spironolactone (25 mg once a day), torasemide, digoxin, amiodarone and finasteride. His plasma levels of testosterone and estradiol were within normal limits.

Although gynecomastia is common in aging men and may be due to extraglandular conversion of androgens to estrogen, a variety of pathological causes can underlie the condition. These include deficient production or action of testosterone (from testicular failure) or renal failure, or increased estrogen production (from testicular tumours, carcinoma of the lung or other tumours producing human chorionic gonadotropin, adrenal disease, liver disease, malnutrition or hyperthyroidism). Many medications have been associated with gynecomastia, including phytoestrogens, estrogens and drugs with estrogen-like properties (e.g., digitalis), inhibitors of testosterone synthesis or action (e.g., ketoconazole, metronidazole, cimetidine, alkylating agents, finasteride) and other agents with unknown mechanisms (isoniazid, methyldopa, tricyclic antidepressants, penicillamine, diazepam, omeprazole, calcium-channel blockers, angiotensin-converting-enzyme inhibitors, marijuana and heroin).

Spironolactone is a well-known cause of gynecomastia and may act by displacing androgen from the androgen receptor and sexual-hormone–binding globulin, and by causing increased metabolic clearance of testosterone and higher estradiol production.[1,2] The patient's spironolactone was replaced with eplenerone, a new aldosterone-receptor blocker that has greater selectivity for the aldosterone receptor and a lower incidence of gynecomastia, mastodynia, abnormal vaginal bleeding and sexual impotence than spironolactone has.

At follow-up 3 months later, the patient was stable and his mastodynia and gynecomastia had resolved.

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