For people who have complex disability or multiple medical problems, respite at a day hospital or in a hospital bed might be more appropriate than care in their own home or a residential home. This can be arranged by the local hospital department of elderly medicine after referral by a general practitioner.

Types of respite care provided by social services departments

- Sitting service
- Day centre attendance
- Family placement schemes
- Respite in residential or nursing home

Services for elderly people with sensory impairment

Local social services departments hold a register of blind and partially sighted people. Registration is voluntary, and follows referral to and examination by an ophthalmologist.

Rehabilitation officers for visually handicapped people work as members of the disability services team and can give advice to help a person with impaired vision cope with the activities of daily living. This may

include training in communication methods and techniques to improve mobility, and special equipment (for example clocks, telephones) may be provided.

Social services rehabilitation officers also act as a link with voluntary services for visually impaired or deaf people. Social services departments or voluntary bodies may run day centres for partially sighted or blind people and deaf people; local authority homes that specialise in the care of visually impaired people are found in some areas.

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Lesson of the Week

Adrenal mass with virilisation: importance of endocrine investigation

Joohi Nasir, Christopher Walton

To prevent unnecessary surgery, all patients with adrenal masses should undergo a full endocrine evaluation irrespective of the apparent hormonal activity of the mass

The combination of virilisation, increased androgen activity, and an adrenal mass on imaging in a female patient suggests a diagnosis of adrenal virilising tumour, for which appropriate treatment is surgical removal. Adrenal "incidentalomas," however, are comparatively common, and this combination of problems can also be due to congenital adrenal hyperplasia. We describe a case of virilisation and left sided adrenal tumour in a 16 year old woman in whom a full endocrinological investigation would have avoided unnecessary surgery.

Case report

A 16 year old woman was referred with primary amenorrhoea, poor breast development, hirsutism, acne, and excessive sweating. She had been investigated for being "above average height" at 8 years of age, for which no cause had been found, although details of investigations performed were not available.

Examination showed that she was 155 cm in height. Facial acne, greasy skin, generalised hirsutism and increased muscle bulk were noted. There was no breast development. Her blood pressure was 135/80 mm Hg. The findings on genital examination were not recorded. Chromosomal analysis confirmed a normal female karyotype (46XX), but testosterone and androstenedione concentrations were greatly increased at 14.0 nmol/l (normal female range 0-2.8 nmol/l) and 68.8 nmol/l (normal female range 3-12 nmol/l), respectively. An ultrasound scan of the abdomen and pelvis showed normal ovaries, uterus, and right adrenal gland, while the left adrenal gland appeared enlarged. Subsequent

computed tomography of the abdomen showed a well circumscribed left adrenal mass 3 cm by 2 cm in size.

On the basis of the raised androgen concentrations and scan appearances a left virilising adrenal tumour was diagnosed, and the patient underwent an uneventful ipsilateral adrenalectomy. Histological examination confirmed a well circumscribed, benign, adrenocortical adenoma, while the adjoining zona glomerulosa and reticularis were described as slightly prominent. The histopathology report included a comment that "in light of the clinical features, this is likely to be functional."

Four weeks after surgery the patient's testosterone and androstenedione concentrations remained greatly increased at 9.3 nmol/l and 51.6 nmol/l, respectively. Selective venous sampling was performed, the results from which suggested that the source of the continuing excessive androgens was the right adrenal gland. Further adrenalectomy was planned, but she was also referred for a full evaluation of her endocrine function. The presence of virilising clinical features was confirmed, although she declined examination of the genitalia, which she said were normal.

Investigations showed that in addition to the raised basal serum testosterone and androstenedione concentrations, basal 17 α -hydroxyprogesterone concentration was also increased at 16.0 nmol/l (normal range 1.4-12.0 nmol/l), suggesting congenital adrenal hyperplasia. This was confirmed by administration of dexamethasone 0.5 mg at night, which returned the androgen and 17 α -hydroxyprogesterone concentrations to within the normal ranges. Measurement of 11-deoxycortisol concentration, which was also grossly

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increased at 289 nmol/l (normal range 18–51 nmol/l), showed that this was due to 11 β -hydroxylase deficiency. Subsequent regular steroid treatment led to onset of menarche, breast development, some regression of hirsutism, a reduction in acne and sweating, and an improvement in muscular appearance.

Discussion

Adrenal masses are fairly common, being found incidentally in 2–11% of abdominal computed tomograms or during necropsy.^{1,2} Management depends on the cause, which includes hormonally active tumours (glucocorticoid, aldosterone, or catecholamine producing adenoma or carcinoma), hormonally inactive tumours (non-functional adenoma, neural tumour, metastases), infections, and haemorrhage. In recent years there have also been several case reports of adrenal masses occurring in association with congenital adrenal hyperplasia.^{2,4} These enzymatic defects of cortisol synthesis result in loss of negative feedback of pituitary adrenocorticotrophin secretion with unremitting stimulation of all three zones of the adrenal cortex; stimulation of the zona reticularis results in excessive androgen production. In the other two zones accumulation of precursor steroids in the cortisol (11-deoxycortisol, zona fasciculata) and aldosterone (11-deoxycorticosterone, zona glomerulosa) pathways is diagnostic if measured in blood and urine. Classic congenital adrenal hyperplasia, which has an overall incidence of 1 in 14 000 live births, is often diagnosed in infancy or early childhood after presentation with ambiguous genitalia or salt wasting crisis. Non-classic forms are surprisingly common, with an overall prevalence of 1 in 100. They are an important differential diagnosis of primary amenorrhoea and virilisation in adolescence, and boys may present at this age with precocious puberty or adrenarche.⁵ Later in life, presentation may occur with varying degrees of virilisation, secondary amenorrhoea, the polycystic ovarian syndrome, or infertility.⁵ Ninety five per cent of cases are due to 21-hydroxylase deficiency; most of the remaining 5% are due to 11 β -hydroxylase deficiency, which is associated with sodium retention rather than salt wasting and may therefore lead to hypertension.

In our patient the presence of virilisation and raised androgen concentrations, along with an adrenal mass,

led to the incorrect diagnosis of a virilising tumour. Initial measurement of concentrations of androgen precursors and metabolites at baseline and after dexamethasone administration would probably have deferred or obviated the need for surgery as most cases can be satisfactorily treated with steroids, which correct the feedback mechanism and suppress abnormal androgen production. Selective venous sampling was helpful in this case, localising the source of excess androgens to the remaining adrenal gland, and this should be considered in the investigation of any unilateral adrenal mass before adrenalectomy, particularly when the diagnosis remains in doubt after endocrine evaluation. If our patient had been medically treated follow up computed tomography of her adrenal mass, which measured 3 cm by 2 cm, would have been considered to monitor changes in size. Some people think that masses measuring 3.0 cm or more in diameter should be considered for early removal in view of the risk of malignancy,^{6,7} but others have suggested that a cut off point of 6.0 cm is more appropriate.^{1,8} Possible decrease in size or at least no further increase could be anticipated after partial suppression of adrenocorticotrophin secretion.^{3,9}

This case illustrates the need for all patients with adrenal masses—irrespective of their apparent hormonal activity—to undergo complete endocrine evaluation before adrenalectomy.

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WHEN I USE A WORD...

Abbreviations can be confusing. Is MI myocardial infarction or mitral incompetence? Is ESM an ejection systolic, an early systolic, or an end systolic murmur? Is SOB shortness of breath or an insult to the patient?

The other day one of the students told me that omeprazole was an H₂ receptor antagonist. I could have simply corrected her error, but the imp of the perverse seized me and instead I asked her what H₂ stood for. Hydrogen, she replied, and then looked a bit confused. I could not in all honesty say that she was wrong, but her answer made the source of her original error clear. I wrote down “O₂, N₂, H₂” and asked her what they meant. Oxygen, nitrogen, and hydrogen, came the reply. So far so good. What about “H₂ receptors”? Ah, yes, the light started to dawn—histamine. Then for the final demonstration: “H⁺/K⁺-ATPase.” And so the link between H (the hydrogen in hydrochloric acid) and H (histamine) receptors was established, and the mode of action of omeprazole could be revealed. Of course she knew that it switched off the secretion of hydrogen chloride by inhibiting the so called proton pump, but she had

confounded the H of hydrogen and the H of histamine.

On another occasion, some years ago, I was discussing the action of nitrates in angina pectoris, and started, as so often, with the history of the subject. Nitroglycerin (or trinitrin) was synthesised by the French chemist Ascanio Sobrero. When he tasted it, as all good nineteenth century chemists did with a new compound, it gave him what he called a migraine, because of its vasodilatory effect. This was later harnessed in the treatment of angina by William Murrell, following the experience of Lauder Brunton with amyl nitrite. For medical purposes it was subsequently renamed glyceryl trinitrate to hide the fact that it was literally, as well as metaphorically, dynamite, the “safety powder” that Alfred Nobel had invented in 1866. “Is that why it’s called TNT?” one of the students asked. Fortunately, the term trinitrin has gone out of fashion, so that nowadays I do not have to explain that TNT (trinitrin) and TNT (trinitrotoluene) are two completely different types of explosive.—JEFF ARONSON is a clinical pharmacologist in Oxford