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Child abuse or copper deficiency? A radiological view

Copper deficiency has been a recognised cause of hypochromic, microcytic anaemia and neutropenia in infants for over 30 years.¹⁻⁴ It also causes skeletal abnormalities and fractures, and recently copper deficiency has been offered to the courts (and given wide publicity) as an explanation of the radiological findings in children whose parents have been charged with abuse. The case of one such child with multiple rib, long bone, and metaphyseal fractures was recently heard by the Court of Appeal. In dismissing the appeal the Lord Chief Justice rejected the defence's evidence on copper deficiency and suggested that it actually strengthened the prosecution case.

Should copper deficiency be considered in the differential diagnosis of non-accidental injury? The skeletal manifestations of copper deficiency occur late⁴ and comprise retardation of bone age, osteoporosis (which is often severe), metaphyseal cupping, increased density of the zone of provisional calcification, and metaphyseal sickle shaped spurs. These changes are distributed symmetrically throughout the skeleton.⁵ Copper deficiency is rare—only about 100 cases have been reported—and fractures caused by copper deficiency are even rarer: there have been 16 documented cases, and only five in term infants.^{3,5-7} Full term infants are born with high body stores of copper that are sufficient for at least the first six months of life. Modern British infant feeding formulas containing 40 µg copper per 100 ml are not associated with copper deficiency in term infants, and no breast fed infant has ever developed copper deficiency. The five full term infants with fractures were all having an abnormal diet or total parenteral nutrition. Rib fractures have been recorded only in premature infants, and skull fractures have never been described. All the children with fractures had obviously abnormal bones.

Metaphyseal fractures are highly suggestive of non-accidental injury and are the most valuable single diagnostic radiographic feature.⁸ They are undoubtedly traumatic: some parents admit to injuring their child, and similar lesions are seen in newborn infants who have suffered a traumatic breech delivery. The new bone formation that accompanies fracture healing is often not present with intra-articular fractures,⁹ and the fractures themselves often do not produce local pain, swelling, and heat.^{8,10} Those who are unfamiliar with this picture may discount a traumatic origin and ascribe the radiological signs to another cause of metaphyseal abnormalities—such as copper deficiency.

The differentiation of metaphyseal abnormalities caused by copper deficiency from metaphyseal fractures caused by child abuse and from normal developmental variants, such as

cupping of the anterior ends of ribs and metaphyseal breaking, is not difficult for the experienced. Bennani-Smires *et al*⁷ described a term infant with multiple metaphyseal fractures caused by copper deficiency that mimicked non-accidental injury, pointing out that it might be possible to confuse the two conditions. The fractures were, however, symmetrical, and other signs of copper deficiency—that is, osteoporosis, metaphyseal cupping, and sickle shaped spurs—were also present. The presence of otherwise normal bones with a normal bone age excludes copper deficiency of a severity sufficient to cause fractures. When other features of copper deficiency (low serum copper and caeruloplasmin concentrations, neutropenia, and anaemia) and predisposing factors (prematurity, total parenteral nutrition, or copper deficient milk) are also absent we can be even more confident.

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Assessment before prostatectomy

Prostatectomy is best performed by the transurethral method. Since most patients will be over 60 preoperative cardiopulmonary assessment is required, and in some cases spinal rather than general anaesthesia may be essential. Preoperative intravenous urography used to be performed routinely, but this policy has been questioned.¹ Plain abdominal radiography and ultrasonography may give the same information more easily and cheaply.² Others have concluded, however, that preprostatectomy urography "remains a sound principle of good urological care."³

Urography may show an intravesical prostatic impression, but prostatic size bears no relation to the symptoms or degree of bladder outlet obstruction. Prostatic size or malignant infiltration are in any case better shown by transrectal ultrasonography or computed tomography.^{4,5} Nor does the unmodified urogram give quantifiable data on total or split renal function; these require additional clearance studies and radionuclide techniques.^{6,7} Dilatation of the upper urinary tract in high pressure chronic retention or ureteric invasion by prostatic cancer will be apparent, but the former condition can be diagnosed on clinical criteria⁸ and the latter condition will be shown by histological examination. Showing dilatation preoperatively is unlikely to affect primary management. An empty bladder on urography does not exclude obstruction—

the detrusor muscle may compensate for appreciable obstruction at the bladder neck; conversely, large residual amounts of urographic media may be found in the absence of true obstruction.⁹ Bladder calculi will be seen on the plain film and diverticula and tumours at cystoscopy—so that the only justification for routine urography becomes detection of incidental lesions. For no other condition is an investigation recommended simply because it might result in the chance finding of disease distant from the focus of clinical attention.

The investigation is positively indicated if a patient has haematuria, unexplained uraemia (up to 15 mmol/l), urinary tract sepsis, or a history of renal disease or stones. Nevertheless, not surprisingly doctors are sometimes reluctant to condemn a patient to a long waiting list without some objective radiographic reassurance that a serious alternative explanation for the symptoms—such as a bladder tumour or early high pressure chronic retention—has not been overlooked.

Formal urodynamic evaluation is unnecessary in patients with uncomplicated prostatic hypertrophy. If a neurologically normal man over 60 with an enlarged prostate complains of hesitancy, frequency, and nocturia and has a poor independent urinary flow rate then he needs a transurethral resection. When irritative symptoms (frequency, urgency, urge incontinence) predominate, however, urodynamic assessment may be vital to avoid removing the prostate from a man with non-obstructive detrusor instability. Flow rate and pressure flow studies will usually suffice, and complex videocystometry should be reserved for equivocal cases.⁹

In most specialist urological units routine cross matching of blood for patients undergoing transurethral prostatectomy is no longer considered necessary. Grouping and saving the serum will suffice if an arrangement is made with the laboratory whereby blood can be made available at short notice. Blood should be cross matched for high risk groups—the very frail, patients with anaemia or blood dyscrasias, or those with prostates estimated to weigh over 50 g.

Clinical evaluation, examination for fitness for anaesthesia, basic blood tests, grouping and saving of serum, a plain abdominal radiograph, and bacteriological examination of the urine are all that should be required preoperatively in most patients admitted with acute urinary retention. What these patients need is a prompt return to life without a catheter. If malignancy is suspected, serum acid and alkaline phosphatase concentrations should be measured. Transrectal ultrasonography may be required, but bone scanning can wait until histological examination has confirmed malignancy. The same applies to patients with chronic retention, but renal function studies and urography or ultrasound should be conducted if upper tract dilatation is suspected and urodynamic studies if low pressure chronic retention is suspected. The man presenting with chronic symptoms of outlet obstruction requires the same assessment as the patient with acute retention. Urography, urodynamics, transrectal or abdominal ultrasonography, and computed tomography should be reserved for specific indications rather than used routinely as part of an irrationally overcomprehensive investigation programme.

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The search for a hormonal switch for obesity

Very rarely is obesity caused by a primary endocrine disturbance such as hypothyroidism or hypercortisolism, and most patients do not have any obvious cause, hormonal or otherwise.¹ Feeding behaviour in man is controlled by a multiplicity of signals, but the search has continued for a peptide that might finally control appetite. We do not seem to be near to finding such a peptide.

The regulation of feeding behaviour is highly complex since the conscious appreciation of hunger and satiety is dependent on so many factors including genetic predisposition,² all the senses, mood, cultural habits, socioeconomic status, intercurrent illness, and, last but not least, food availability.³ The functional distinction between a ventromedial hypothalamic satiety centre and a ventrolateral hypothalamic feeding centre has served as a valuable experimental model⁴ and may be relevant in certain types of genetically obese mice as well as in the rare cases of hypothalamic obesity in man.⁵ Other brain areas, however, are also important in feeding behaviour, especially the paraventricular nucleus of the hypothalamus, the globus pallidus, and the amygdala.³ These hypothalamic centres may sort and integrate afferent information from both the periphery and other brain regions. The centres produce an efferent autonomic response as well as a conscious appreciation of the need to eat.

Many hypotheses have tried to provide an all embracing explanation for appetite regulation and its disorders, but we are not as simple as the mollusc pleurobranchia, which eats everything in sight until its appetite is switched off by its egg laying hormone.⁶ In man we must consider the afferent input from taste, smell, and sometimes even auditory and visual stimuli. Additionally there are important autonomic afferent signals from stomach, gut, and perhaps liver that may interact with messages (dependent on specific hypothalamic receptors) from certain nutrients and metabolites such as glucose, glycerol, amino acids, and possibly free fatty acids.^{3,7-9}

Against this complex background are feedback signals mediated by classical hormones both old and new, and since many of the peptide hormones are synthesised in both the brain and the periphery (especially the gut and pancreas) there is much speculation on the specific production and target sites in relation to appetite control. Important among these are thyroid hormones¹⁰ and glucocorticoids,¹¹ which stimulate appetite, and cholecystokinin, bombesin, somatostatin, and pancreatic glucagon, all of which can produce some degree of satiety.¹² Although in man

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