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Medical Complications of anorexia nervosa

It is impossible to cover the medical complications of anorexia nervosa more than superficially and for more detailed information we would suggest the following resources: American Psychiatric Association, 1993; Bhanji & Mattingly, 1988; Kaplan and Garfinkel, 1993; Sharpe and Freeman, 1993; Treasure and Szukler, 1995.

Skin and hair changes

The skin is dry, and fine downy hair – so-called lanugo hair – develops. There is often loss of head hair, and this will appear thin and lifeless.

Musculoskeletal problems

Muscles: Individuals with severe anorexia nervosa have poor muscle strength and a decrease in stamina. Eventually, proximal myopathy develops with difficulty standing from a crouch or lifting the arms above the head to comb the hair. The poor muscle strength also leads to an impairment in respiratory function (Murciamo et al, 1984).

Bones: Osteoporosis and pathological fractures are one of the commonest causes of pain and disability in anorexia nervosa (Treasure and Szmulker, 1995; Herzog et al 1990). The annual incidence of non-spine fractures of 0.05 per person year in anorexia nervosa is seven fold higher than the rate reported from a community sample of women aged 15-34 (Rigotti et al, 1991). Risk factors for this complication are a long duration and an increased severity of illness (Serpell and Treasure, 1997). Refeeding alone produces a rapid rise in bone turnover (Stephanis et al, 1997) and an increase in bone mineral content (Orphanidou et al, 1997). Insulin growth factor also increases bone turnover (Grinspoon et al, 1996). The value of hormone replacement therapy is uncertain, overall it produced no effect although it may protect against further bone loss in the subgroup who remain chronically unwell (Klibinski et al 1995). It is uncertain whether it is possible to restore bone mass to normal levels. Patients who have gained weight and have had a return of menses over many years had persistent osteopenia (Ward et al, 1997). Duration of amenorrhoea/illness was the best predictor of osteopenia, but also an index of the duration of recovery was highly correlated with outcome.

Dental changes

The commonest stigma of persistent vomiting is erosion of dental enamel in particular from the inner surfaces of the front teeth. Eventually, dentine is exposed and the teeth become over sensitive to temperature and caries develops. Dental complications such as abnormal tooth wear are not limited to the group which vomit (Robb et al, 1995). The other causes of poor dental health are over-consumption of acidic foods such as fruit and carbonated drinks, grinding and loosening of the teeth due to osteoporosis of the jaw.

Effects on the CNS

Brain substance decreases in anorexia nervosa and the ventricular spaces and the sulci increase in size (Dolan et al, 1988; Katzman et al, 1996; Krieg et al, 1988). The increased resolution offered by MRI has also shown that the pituitary is smaller (Dolan et al, 1988; Katzman et al, 1996). To a degree these structural abnormalities, such as loss of grey matter persist despite weight recovery for over a year, which suggests that there may be a degree of irreversible damage even in adolescents with a short history (Lambe et al, 1997). The cause of the cerebral atrophy is uncertain. It may be a general effect of starvation or may result from the high level of cortisol which is present in anorexia nervosa and which is known to be toxic to dendrites (Sapolsky, 1992, 1996). A post mortem study of a 13 year old girl who died of anorexia found that the dendrites showed evidence both of stunting of growth and of neuronal repair (Schönheit et al, 1996). In addition, women with anorexia nervosa may be at greater risk for Alzheimer's disease because of their prolonged state of oestrogen deficiency, if it is possible to extrapolate from the findings in post menopausal women in which it was found that oestrogen treatment appeared to delay the onset of dementia (Tang et al, 1996).

Neurophysiological abnormalities such as vertex transients are more common in adolescents with anorexia nervosa (56%) than in a control population (14%) (Rothenberger et al, 1995). Also auditory brain stem response has been found to be abnormal (Rothenberger, 1991; Miyamoto et al, 1992). Functional imaging such as Single positron emission CT (SPECT) studies have shown decreased flow both in the active state and after recovery (Gordon et al, 1997; Rastam et al, 1997). Functional MRI scanning has shown an abnormal response to images of high calorie drinks with increased activation in the insula and amygdala (Ellison & Foong, 1998).

Functional cognitive impairment is seen with deficits in memory tasks, flexibility and inhibitory tasks persisting despite weight recovery (Kingston et al, 1996). Women who have recovered from anorexia nervosa have average IQ scores but have poorer scores on the object assembly subtest (Gilberg et al, 1996).

Cardiovascular problems

The heart becomes smaller and less powerful, because muscle is lost and the blood pressure and heart rate is lowered. This can lead to faints. There is poor circulation in the periphery and this leads to cold blue hands, feet and nose. At its extreme, this results in chilblains and even gangrene, in particular in children.

There have been reports of cardiac valvular problems (Johnson et al, 1986) although many of the murmurs that are heard are flow murmurs. Sudden death occurs in anorexia nervosa and may result from arrhythmias (Isner et al, 1985). QT prolongation is common in anorexia nervosa (Cooke et al, 1994). Low potassium which results from many of the methods of weight loss can exacerbate this problem.

Fertility and reproductive function

Fertility is reduced in women with anorexia nervosa. In part this is due to suboptimal physical recovery. In a follow up of 12.5 years in Denmark, the fertility rate was a third of that expected and the perinatal mortality rate was six fold higher (Brinch et al, 1988). The birth weight of children born to anorexic mothers is lower than average (Treasure and Russell, 1988). Women with anorexia nervosa may also have difficulties in feeding their children who may become malnourished and stunted in growth (Russell et al, 1998).

Endocrine system

The hypothalamic-pituitary-gonadal axis regresses to that of a prepubertal child. The pituitary does not secrete FSH and LH and the ovaries decrease in size. The ovarian follicles remain small and do not produce oestrogens or progesterone (Treasure, 1988). By contrast the hypothalamic pituitary adrenal axis is overactive, probably driven by excess CRF, with high levels of cortisol which are not constrained by any feedback (Gwirtsman et al, 1989; Licinio et al, 1996).

Gastrointestinal tract

Residual gastrointestinal problems such as irritable bowel syndrome are common after recovery from anorexia nervosa (Herzog et al, 1992). Functional abnormalities such as delayed gastric emptying and generalised poor motility are related to the degree of undernutrition (Szmukler et al, 1990). Anatomical abnormalities as a result of the trauma of vomiting and overeating or loss of mesenteric fat occur. Structural abnormalities such as ulcers etc. are common (Hall et al, 1989). It is important not to overlook the effects of sorbitol present in sugar free gums and sweets, which can cause abdominal distension, cramps and diarrhoea (Ohrlich, 1989).

Salivary glands hypertrophy and produce increased levels of amylase (Kinzl et al, 1993). Pancreatitis is an extremely rare complication (Gavish et al, 1987). In cases of severe emaciation, fatty infiltration of the liver occurs and liver enzymes increase (Hall et al, 1989).

Haematology

All components of the bone marrow are diminished but the order in which this is discernible in the peripheral blood is white cells, red cells and finally platelets. The level of marrow dysfunction relates to the total body fat mass (Lambert et al, 1997). The immune system is compromised with a decrease in CD8 T cells (Mustafa et al, 1997).

Blood chemistry

In restricting anorexia nervosa, the most common abnormality is a low urea level which is a function of a low protein intake. Low potassium levels result from vomiting or laxative and diuretic abuse. Usually, this is associated with raised levels of bicarbonate but some laxatives can produce a metabolic acidosis. Many other salts and metabolites are reduced, for example magnesium, phosphate, calcium sodium and glucose.

Comorbidity with physical problems

One of the most important areas of difficulties is when eating disorders develop in the context of somatic illness. This is a particular problem with diabetes mellitus: approximately a third of adolescent girls with diabetes have some form of eating disorder (Fairburn & Steele, 1980; Rydall et al, 1997; Williams & Gill, 1997) and these tend to persist. It is common for these women to omit their insulin as a means of losing weight (Szmukler & Russell, 1983). The combination of diabetes and an eating disorder leads to the development of early and severe neurovascular complications (Steel et al, 1987; Colas et al, 1991; Ward et al, 1995), retinopathy (three times as prevalent in patients with highly disordered eating, Rydall et al, 1997), osteoporosis (Vila et al, 1995) and a higher mortality (5 times that of anorexia alone and 15 times that of diabetes, Nielsen et al, 1998; Nielsen & Molback, 1998). Eating disorders also lead to difficulties in the management of Crohns disease (Meadows & Treasure, 1986) and thyroid disease (Tiller et al, 1995).

Diabetes was more common in women with bulimia nervosa (OR 2.3) and binge eating disorders (OR 3.6) (Johnson, 2001).

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