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3823 John D. Firth

ESSENTIALS Idiopathic oedema is an unsatisfactory label that is applied to women who complain of swelling, typically variable, with diag- nosis requiring exclusion of known causes of oedema and (most authors would agree) demonstration of weight gain, from morning to evening, of more than 1.4 kg. The cause of idiopathic oedema is (by definition) unknown:
hypotheses include abnormal capillary permeability/leakage, re- feeding oedema, and diuretic-induced oedema. There is no clear relationship to the menstrual cycle. Even if not a primary cause, the use and abuse of diuretics can complicate and exacerbate the problem. Management is difficult, but patients can be helped by a sympa- thetic approach from the physician and
(1) encouragement to lose weight if they are obese; (2) avoidance of excess dietary salt; and
(3) weaning from consumption of high doses of diuretics that can cause or exacerbate the tiredness, lethargy, weakness, and dizziness that are suffered by many. Definition and diagnosis In some women fluid retention occurs in the absence of any clear explanation and is termed idiopathic oedema. Since the condi- tion typically fluctuates in severity from one time to another it is sometimes called cyclical or periodic oedema, but these terms mis- lead; first, because there is rarely any recognizable periodicity, and second, because the condition is not related to menstrual periods. Most women retain fluid just before the menses and lose this fluid immediately afterwards. Idiopathic oedema occurs most com- monly in women aged 20 to 40 years, but has no clear relationship with the menstrual cycle and can persist after the menopause or oophorectomy. The diagnosis of idiopathic oedema depends on the exclusion of other causes of oedema, including cardiac, hepatic, renal, allergic, or hypoproteinaemic disease, venous or lymphatic obstruction, and use of some medications. The role of diuretics, causally or in treatment, is contentious, as discussed next. However, it is always unsatisfactory when a diagnosis is made by exclusion of other con- ditions rather than on the basis of ‘positive’ criteria. Such criteria for the diagnosis of idiopathic oedema have not been universally agreed, although both Thorn and McKendry (see Kay

et al. for discussion) have made proposals that (1) require evidence of substantial weight gain during the course of the day from morning to evening, with a figure of more than 1.4 kg often quoted, although this does not provide a clear-cut separation from normal, and (2) demand the presence of (loosely specified) emotional or psychological factors. Many authors comment on the aggravation of swelling by prolonged sitting or standing, but this does not feature in the diagnostic criteria mentioned. Clinical features The patient's complaint is of swelling, which usually waxes and wanes but can be constant. In the morning the face and eyelids feel swollen and heavy. By the end of the day the areas worst affected are the hands, breasts, trunk, abdomen, thighs, ankles, and feet. Rings no longer fit swollen fingers, and undergarments and clothes can feel so uncomfortably tight that they have to be removed or replaced with something larger. The feet and ankles may be relatively spared, hence the disposition of oedema tends to be different from that in most other oedematous states, where it begins distally in the feet and ankles and progresses proximally. Episodes or exacerbations of fluid retention often occur unpre- dictably, but obesity, emotional stress, and consumption of high- carbohydrate food are thought to be triggers in some. Sufferers are often mentally and physically lethargic during periods of fluid re- tention, frequently expressing the view that they feel bloated and ugly, even though this may not be apparent to the observer. Many appear to be emotionally labile or anxious and some are depressed, invariably (and perhaps correctly) claiming that this is secondary to the fluid retention. Other common symptoms include carpal tunnel syndrome, nonarticular rheumatism, palpitations, nonulcer dys- pepsia, and headaches, and idiopathic oedema may be a factor in women troubled by severe cellulite. Women with idiopathic oedema are often very concerned about their weight. One study showed an association between symptoms 16.19 Idiopathic oedema of women John D. Firth

section 16 Cardiovascular disorders 3824 of this condition and abnormal attitudes to eating. Some patients will report that they episodically severely reduce their food intake, perhaps making them susceptible to the phenomenon of re-feeding oedema when they start eating again. Aside from oedema, which may or may not be present at the time of medical assessment, examination is unremarkable, as are routine in- vestigations for the cause of oedema. Those patients that have used di- uretics may have a hypokalaemic hypochloraemic metabolic alkalosis. Idiopathic oedema has been reported to be associated with a range of conditions including obesity, diabetes, and psychiatric disorders. Apart from depression, the latter may include purging behaviours (self-induced vomiting, laxative abuse, diuretic abuse). Making the diagnosis of an additional 'idiopathic' condition in such circum- stances is fraught with difficulties. Pathophysiology The cause of idiopathic oedema is not known (by definition). Because of pooling of extracellular fluid in the legs when standing, normal weight gain during the day is typically around 1 kg, and di- urnal weight fluctuation of more than 1.4 kg is required for diag- nosis, but weight may fluctuate from day to day by up to 4 or 5 kg. During periods of weight gain the patient may be oliguric, passing low volumes of urine in which there is little sodium (<20 mmol/ litre). There are three main pathophysiological hypotheses. Abnormal capillary permeability The blood vessels of women with idiopathic oedema are more perme- able to albumin, the fractional catabolic rate of albumin is increased, both intravascular and total body albumin pools are smaller, and the plasma volume decreases by more on standing than in normal controls. Activation of the sympathetic nervous system, renin– angiotensin–aldosterone system, and high levels of antidiuretic hor- mone in the plasma that are consistent with intravascular volume depletion have all been reported, and these changes provide a plaus- ible explanation for why the kidney retains salt and water in idiopathic oedema. They also form the background to postural water-loading or sodium-loading tests that

have been advocated as diagnostic tools, al- though these are not used routinely in clinical practice. After similar loading on two separate occasions, patients with idiopathic oedema who remain upright throughout the test excrete less water or sodium than they do if they remain supine. However, the prime mover re- mains uncertain: decreased release of dopamine has been reported, as has generalized impairment of hypothalamic function. Re-feeding oedema If women concerned about their weight engage in ‘crash dieting’ followed by binge eating, then they may induce re-feeding oedema. This has led some authors to the conclusion that idiopathic oedema may be a presentation of eating disorder. Why re-feeding should pre- cipitate oedema is not clear, but in a study of malnourished patients with anorexia nervosa, those re-fed with a low-sodium diet were less susceptible to oedema than those re-fed with a normal-sodium diet. Diuretic-induced oedema Many patients seen in hospital practice will already be taking diur- etics or have taken them in the past, and some will be consuming large doses of loop agents every day. One influential study re- ported 10 such patients who started to take diuretics because of concern about swelling or their body weight and who continued to take them because cessation provoked rapid weight gain, facial bloating, and abdominal distension. When prevailed upon to stop diuretics they each gained weight (up to 5 kg), reaching a max- imum in 4 to 10 days, but by 20 days 7 of the 10 had fallen to below their previous weight, and 9 of the 10 remained free of oedema over a long period of follow-up without taking diuretics. This led the authors to suggest that diuretic abuse might be the cause of all cases of idiopathic oedema. This view is not held by most with ex- perience in the field, but rebound oedema on diuretic withdrawal can undoubtedly be an exacerbating feature, and it is appropriate to look for evidence of diuretic abuse if the patient denies taking such drugs and yet routine biochemical testing of blood and urine suggests the possibility. Management Women with idiopathic oedema frequently complain that doc- tors have not taken their condition seriously, and there is no doubt that it is a frustrating disorder for both patients and their phys- icians. Sympathetic explanation of the nature of the problem helps management. A patient who is obese should be given advice as to how to lose weight, and—independent of any effect on weight—some find that reducing dietary sodium and carbohydrate intake helps. They should be advised to avoid long periods of standing or sitting and to wear loose-fitting clothing, although most will have discovered these things for themselves. Avoidance of an excessive dietary intake of so- dium is a sensible recommendation. On theoretical grounds the use of elastic stockings would also seem appropriate, since these might reduce the postural reduction in plasma volume seen in idiopathic oedema. However, few find that the benefits of elastic stockings out- weigh their disadvantages and it is difficult to get most patients to persist with them for long enough to see whether or not they really would be of help. Diuretics are a real problem. It seems intuitively obvious to most patients and to many doctors that someone who is retaining fluid would benefit from a diuretic, hence many patients with idiopathic oedema end up on very large doses of loop agents, often combined with amiloride or spironolactone. Rather than helping, these may worsen symptoms of tiredness, lethargy, weakness, and dizziness by exacerbating intravascular volume depletion, and attempts to stop typically lead to rebound oedema. Long-term use of high doses of furosemide can also cause medullary nephrocalcinosis and variable degrees of renal insufficiency. Explanation is the key here, in that if patients recognize rebound oedema for what it is and relieve oedema with supine rest rather than renewed consumption of high doses of diuretics, then there is a reasonable chance that they can be weaned off diuretics with benefit. A range of agents including levodopa, carbidopa, bromocrip- tine, captopril, metformin, calcium dobesilate, and ephedrine have been tried in idiopathic oedema. None is of widely proven benefit. There is a single report that aminaphtone (aminophenazone) pro-

duced improvement in 70% of cases in a small series, but this drug (formerly used as an antipyretic and analgesic, but which can cause

16.19 Idiopathic oedema of women 3825 leucocytopenia) is not widely available. A controlled study suggested that non-surgical periodontal therapy led to improvement in idio- pathic oedema and hypothesized that this might be by reducing a source of systemic inflammation. However, the duration of follow- up was very short (4 weeks), and further studies of adequate dur- ation are needed to know whether or not such treatment should be recommended for this chronic condition.
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