

# IDIOPATHIC EDEMA IN THE ATHLETIC WOMAN

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## SYNOPSIS

**Idiopathic edema is a poorly understood disorder of unknown etiology occurring almost exclusively in females of reproductive age.**

**A case is described in a competitive female runner, possibly related to the sustained fall in blood pressure which occurred on assuming serious straining. The possible etiology of this condition is discussed, and therapy for the athletic female, based on general management, and intermittent, bolus diuretic treatment, is suggested.**

## INTRODUCTION

Fluid retention occurring at apparently irregular intervals is seen occasionally in women, and has been called idiopathic edema. The mechanism is unknown, although several theories have been formulated, and treatment is unsatisfactory. Here is a report of this condition occurring in an otherwise healthy female competitive runner.

## CASE REPORT

A 37-year-old married woman, had been having bouts of irregular and rapid weight increase and facial, leg and hand edema for approximately 3 years. At 59 kilos, 1.5 metres at age 18 she had been dieting sensibly all her life and engaged in only light exercise until at age 32, she began jogging, and a year later, serious training and competing in local distance races, up to 10 kilometres. Her weight had gradually decreased to 54 kilos. She was also a regular blood donor. Consequently daily weights and regular 3-4 monthly blood pressures were available. She was not on birth control tablets or any medication. The edema appeared insidiously and would involve weight gains of up to 4.5 kilos over a period of 12 hours. This would then take 3 or 4 days to subside. It tended to occur more often in the premenstrual period, and was especially severe after plane journeys lasting more than 2 hours. Hot weather markedly increased the frequency and severity of these episodes. No other reliable precipitating factors could be found in spite of careful investigation, except possible an occasional relationship to some foods, such as mangoes, and some pollens, suggesting an allergic component. The increased weight was associated with mild incoordination, tiredness, depression and mood alteration and poor racing and training performances. Exertional dyspnoea occurred much sooner, suggesting a decreased aerobic capacity. Blood counts, enzyme and electrolyte profiles and urinalyses performed during the between such bouts never showed any abnormalities. However, there had been a sustained and moderate blood pressure decrease, which occurred during the first few months of serious training, from 120/80 to 105/60. This was also unaffected by the weight gains.

Management of this has been difficult. Mild edematous periods have been untreated, especially if near menstruation. Swimming and caffeine-containing drinks help. However, more pronounced episodes, especially if near a race, have responded well to a short course of chlorothiazide (500 mg), usually two doses. A single dose of 25 mg of spironolactone taken with 500 mg of chlorothiazide has been even more effective. Antihistamines have not seemed helpful.

## DISCUSSION

Many female runners appear to be familiar with this disorder, but apparently few seek professional advice, and if they do, few physicians appear knowledgeable about this condition. It is thought to be fairly common, and to affect almost exclusively women of reproductive age. An excellent recent review has been given by Marks in *Postgraduate Medicine*. (1) Typically, stress, premenstrual timing and prolonged standing appear to be precipitating or intensifying factors, and the fluid accumulates in dependant areas, and the face, hands and breasts.

The diagnosis can be made by the history of excessive daytime weight gain (in excess of 1 kg) together with a positive water-loading test. However, in an athletic woman with the typical history, and no physical or chemical evidence of renal dysfunction, the confirmative test is probably unnecessary.

The pathophysiology is still unknown. The final common pathway is very possibly a secondary increase in aldosterone production, with salt and water retention and volume expansion. Many factors are known to influence the renin-angiotension-aldosterone reaction, especially posture, and upright posture does appear to

be the most frequent precipitating factor in this disorder. (2) Presumably plasma volume contraction is accentuated by upright posture, with decreased renal perfusion. The mechanism may be a capillary basement membrane abnormality, allowing leakage of plasma water and protein into the interstitial space, (3) and other hormonal abnormalities have been described. There may also be hypothalamic defect. (4)

The subject of this case report shows two interesting features. Long periods of time spent in aeroplanes was especially likely to exacerbate the problem. This is probably due to the upright posture and lack of movement, and also possibly the lowered oxygen tension, which in itself is known to stimulate the renin-aldosterone reaction. The only other physiological change has been the moderate and sustained fall in blood pressure. This may result in mild hypoperfusion of the kidneys with the resulting stimulation of the renin-aldosterone pathway, although this cannot be the causative factor, as the kidney in normal people can easily autoregulate renal blood flow in response to a gradual reduction in blood pressure secondary to physical training.

Treatment has not been entirely satisfactory. An essentially non-pathologic condition which is often only uncomfortable to a spectator can be debilitating in an athlete. Physical activity in athletes may limit the edema somewhat, and swimming should be considered as part of the exercise program as it gives exercise in the horizontal position. Sodium restriction is usually advised, and this should probably be recommended for athletes also. Fluid restriction is also suggested, but I feel this is too dangerous in athletes who will sweat large amounts, especially in hot and humid conditions, which as we have seen, intensifies the disorder. The discomfort of the edema is vastly outweighed by the possible effects of dehydration. Leg elevation and weight reduction are suggested, and should be tried, but other therapies, such as support hose, are really impractical in the tropics. Sympathomimetics, especially dextroamphetamine, have been used with some success, (5) and bromocriptine has been recommended, but with doubtful efficacy. (6) Many of these patients in the West are already on diuretics. This is less likely in Singapore, but if found, attempts should probably be made to wean the patient off regular usage of these drugs.

The main drug therapy will rest on diuretics, but these should be used sparingly. It is most important to avoid long term, continuous diuretic treatment in athletes, as this will be highly likely to lead to some electrolyte imbalances, and the loop diuretics especially are liable to aggravate the underlying secondary aldosteronism. In the present case the single bolus effect of 500mg of chlorothiazide, together with 250mg of spironolactone, was extremely effective, and appeared to last for several days. The stronger diuretics, such as frusemide, did not appear to work as well, and I feel should be avoided. Also spironolactone on its own was not very effective. Antihistamines are worth trying if there seems to be an allergic component. Probably each patient will have to be individually assessed as to their response to short courses of thiazides and spironolactone. Diuretics should probably not be used within 12 hours or so of a strenuous training session or a competitive event, especially in hot weather, in case electrolyte imbalances occur.

Above all sympathy and understanding is most important. As is frequently the case where no pathology can be demonstrated, many of these sufferers are considered to be either emotionally unstable or to be greatly exaggerating the subjective feelings of discomfort. (7) They must be convinced that their condition does have an organic basis, and to a large degree they are responsible for the monitoring and treatment of it.

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