Fluid Retention Syndrome in Women

Abstract

The term Fluid Retention Syndrome (abbreviated; FRS) is more accurate than the commonly used term “idiopathic edema” or “cyclical edema”, as it substantiates the view that the disorder has multifactorial etiology. FRS of women may be defined as fluid retention occurring in the absence of well-defined hydrostatic or oncotic mechanism as a result of congestive cardiac failure, hypoproteinemia or local venous or lymphatic obstruction.

FRS may result from humoral, metabolic, autonomic and iatrogenic mechanisms. The syndrome comprises a triad of fluid retention, autonomic disturbance and affective disorder; the latter represents the cornerstone of the syndrome and the condition is a distinctive psychosomatic disorder.

The symptoms of fluid retention are variable and many patients are subjected to multiple hospital referrals for unnecessary investigations, medication or surgery which can be avoided once the unified scope of the syndrome is well appreciated.

From November 1991 – Nov. 1994 we could study 52 cases of typical triad symptoms of FRS between the ages of 16-52 years (mean 36.5 years). These cases represent our ongoing workup to find out more cases to establish the syndrome as a true and existing entity in our daily medical practice.

Introduction

Fluid Retention Syndrome in Women (FRS)

The commonly used term for FRS “Idiopathic edema” is misleading, because sufferers rarely show pitting or dependent edema and contributory risk factors can be defined, the descriptive term “FRS” is more precise as it distinguishes the perspective that the condition has multifactorial etiology.

The majority of general practitioners think that “FRS” doesn’t exist as distinguished condition, but as indefinite or rather nebulous entity associated with women who demand diuretic for “bloating” or “swelling”. The condition is frequently, confused with premenstrual tension and some deny its existence dismissing as imaginary the complaints of women who insist that they swell during the day have to spend the evening in loose dressings [1].

Definition

“FRS” may be defined as a fluid retention in the absence of well-defined hydrostatic or oncotic mechanism as a result of congestive cardiac failure, hypoproteinemia or local venous or lymphatic obstruction. Most patients are between 20-50 years but the onset may follow menopause. It is rare in children, although the syndrome was found between 2-10 years of age where there is strong family history of the syndrome. FRS is extremely rare in men [1].

Etiology

FRS may result from humoral, metabolic, autonomic and iatrogenic mechanisms [2]. Although the definitive etiology is uncertain, increased permeability of capillary walls accentuated by arteriolar vasodilation appears to be a primary culprit in etiology [3,4], both mechanisms result in the transfer of fluid from the intravascular to the extravascular compartment with the resulting hypovolemia leading to sodium and water retention through activation of Renin-Angiotensin-Aldosterone system (RAAS).

Clinical Features of FRS

These comprise a triad of symptoms of fluid retention, autonomic disturbance, and affective disorder [2]. The second and the third components of the triad are present to a variable degree in the individual patients. Fluid retention leads to complaints of swelling of the face, hands, breasts, abdomen and legs, which becomes worse as the day advances. The patient frequently changes into loose dressings, and the time at which this occurs is a useful assessment of the entity and the severity of this disorder. The patient’s face may appear puffy and bloated, rings may be discarded and a distended abdomen may lead to accusations of pregnancy!

Most women retain fluid to some extent, during premenstrual period, and there is no clear dividing line between normal and abnormal fluid retention. Fluid retention is usually accompanied by diurnal weight variation of between 1.4 and 6.0 kg (3-14lbs.) daily. A diurnal weight variation of more than 1.4 daily is considered abnormal [5]. Longer term fluctuation weight with a periodicity of 1-2 weeks is not uncommon see Table 1.
Table 1: Common symptoms of FRS in Clinical Cases.

<table>
<thead>
<tr>
<th>Weight Gain From Fluid Retention</th>
<th>Breast Tenderness</th>
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<tbody>
<tr>
<td>Mood Swing and Irritability or Anger</td>
<td>Tension or Anxiety</td>
</tr>
<tr>
<td>Appetite Changes and Food Cravings</td>
<td>Depressed Mood</td>
</tr>
<tr>
<td>Insomnia</td>
<td>Crying Spells</td>
</tr>
<tr>
<td>Joint or Muscle’s Pains</td>
<td>Headaches</td>
</tr>
<tr>
<td>Abdominal Bloating</td>
<td>Fatigue</td>
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Parasympathetic Overactivity

That includes hyperactivity of the bowel, bladder and vascular system, a history of irritable bowel; with complaint of abdominal pain, intermittent diarrhea and constipation is common and the patients may be referred to gastroenterologist or gynecologist.

Complaints of urge frequency of micturition amounting to urge incontinence and in severe cases may lead to unnecessary antibiotics treatment for urinary “infections” or referral to an urologist.

Intermittent vasovagal attacks, in forms of syncope or dizziness spells are not uncommon and lead to a mistaken diagnosis of epilepsy and neurological referral as well!

Most fluid retention patients show symptoms of varying degree of affective disorder; this range from minor degree of fatigue, irritability anxiety and depression. Affective symptoms form an integral part of FRS and the condition is a true psychosomatic disorder [6].

Visual blurring caused by retinal edema is common in fluid retaining patients; and lead to consult the ophthalmologist. Tension headaches commonly accompany affective symptoms, in few patients headaches is severe and worse in the morning resembling headache of increased intracranial pressure, presumably caused by a degree of cerebral edema. Polydipsia result from hypovolemic stimulus following extravasation of fluid from intravascular to extravascular compartment leads to consumption of large quantities of fluid with nocturnal polyuria. If thirst is intense the patient may be suspected of having diabetes mellitus or hysterical water drinking. In summary, the symptoms of FRS are variable and many patients are subjected to multiple hospital referrals for unnecessary investigations, medications or surgery which can be avoided once the scope of the syndrome is appreciated.

Risk Factors of FRS

Recognition of the contributory risk factors in fluid retention is essential for rational and effective management.

Metabolic factors

Obesity, or a history of weight gain is found in most fluid-retaining patients. A family history of diabetes is common, and acute fluid retention may occur in the both sexes with the institution of insulin therapy for diabetes mellitus (‘insulin edema’) [7]. Subacute and chronic forms of diabetic oedema are seen in young women with unstable diabetes [8].

Endocrine factors

Thyrotoxicosis, hypothyroidism [9] and estrogens may be associated with fluid retention. More subtle endocrinopathies may be involved in the etiology of fluid retention. In one study, urinary dopamine levels were low in patients with fluid retention [10] and, in another, gonadotrophin levels were raised after stimulation with releasing hormones compared with control patients.

Psychiatric factors

Affective symptoms are common in fluid-retaining patients and onset may follow emotional stress related to life events.

Short-term Precipitants

Prolonged standing, high ambient temperatures, a febrile illness and acute emotional stress, liberal dietary intake (Alcohol, excessive carbohydrate) may accentuate pre-existing fluid retention (Table 2).

Table 2: Major Risk factors for the FRS.

<table>
<thead>
<tr>
<th>Female Sex</th>
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<tbody>
<tr>
<td>Metabolic Factors</td>
</tr>
<tr>
<td>Obesity (20% Over Ideal Weight Reactive Fluid Retention).</td>
</tr>
<tr>
<td>Weight Gain In The Normal Weight Range For Age Height (Weight Reactive Fluid Retention).</td>
</tr>
<tr>
<td>Diabetic Family History</td>
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<tr>
<td>Diabetes Mellitus (Diabetic Oedema)</td>
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<tr>
<td>Endocrine Factors</td>
</tr>
<tr>
<td>Estrogens (Premenstrual Fluid Retention)</td>
</tr>
<tr>
<td>Thyroxine (Hypo-And Hyperthyroidism)</td>
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<tr>
<td>Psychiatric Factors</td>
</tr>
<tr>
<td>Reactive/Endogenous Depression</td>
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<tr>
<td>Anxiety States-Depression</td>
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<tr>
<td>Iatrogenic Factors</td>
</tr>
<tr>
<td>Steroid Hormones (Estrogens As Oral Contraceptive Or As Hormone-Replacement Therapy, Fludrocortisone, Danazol).</td>
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<tr>
<td>Aldosterone-Like Compounds Carbenoxolone, Liquorice)</td>
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<tr>
<td>NSAID's (Including Aspirin).</td>
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<tr>
<td>Hypotensive Drugs (Guanethidine, Hydralazine, Prazosin, Calcium Channel Antagonists).</td>
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<td>TZDs; Glitazones</td>
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Hypothetical Links between the Risk Factors and the Fluid Retention Syndrome

The nature of the endocrine or metabolic defect responsible for abnormal fluid retention in women is unknown. Increased sympathetic activity may diminish renal sodium and water excretion via diminished renal dopamine synthesis [10]. Active
neurogenic vasodilation with increased bowel and bladder contractility leading to fluid retention, the irritable bowel syndrome and urge frequency of micturition may have a common basis; increased parasympathetic (cholinergic) activity. In fluid-retaining patients, increased autonomic activity appears to be driven centrally via the connections of the hypothalamus with limbic system; the putative site of affective disorder.

The association of fluid retention with obesity, diabetes mellitus or a diabetic family history may result from the absolute or relative hyperinsulinemia that commonly accompanies these metabolic disorders. Insulin is known to induce renal retention of sodium and water, and insulin-mediated upregulation of cellular sodium/potassium ATPase results in efflux of sodium from the intracellular compartment and fall in intracellular sodium and calcium that will lead to fall in resistance vessel resulting in arteriolar vasodilation increases capillary ultrafiltration and augments fluid retention via arterial hypovolemia. As in T2 diabetes mellitus, insulin responsiveness may be restored by dietary restriction, leading to effective weight loss.

The mechanisms of vasodilating precipitants of fluid retention (such as thyrotoxicosis, fever and high ambient temperature) and of iatrogenic precipitants such as estrogens, danazol and NSAID’s are well recognized and require little comment [12,13].

**Management of the Fluid Retention Syndrome**

It is important to reach an accurate diagnosis of the condition based on the exclusion of cardiac, hypoproteinemic and obstructive cases of edema. This may be achieved by appropriate screening investigations, a characteristic history and the demonstration of the abnormal diurnal weight variation by means of weight chart kept by the patient over a period of 2-4 weeks. Most cases respond to abolishing or modification of the contributory risk factors (Table 3).

**Weight-reactive Fluid Retention**

Where fluid retention is weight-reactive, the important aspect of management is to return the patient to a target weight. This may be defined either as the patient’s weight in early adult life or, less ambitiously, body weight prior to the onset of fluid retention. It is important to set realistic goals for weight reduction. Long-term weight loss of 0.5-1.5 kg (1-2 lbs.) weekly is satisfactory but losses of up to 2.3-2.7 kg (5-6 lbs.) may occur in the first weeks of dieting as a result of fluid loss. Salt restriction is unnecessary beyond that imposed by reduced caloric intake. Fluid intake should not be restricted.

Symptoms of bloating and swelling subside, accompanying affective symptoms improve, and in many cases, the patient describes a sense of well-being and positive health that may have been absent for many years. In most cases of weight-reactive fluid retention, dietary means are sufficient to control symptoms, diuretics are usually unnecessary and not be prescribed initially.

A proportion of non-obese fluid-retaining patients give no history of weight gain before the onset of symptoms. In these patients, the dominant risk factors are usually emotional, and diuretics are effective in controlling symptoms in inverse proportion to the degree of psychiatric abnormality present.

Symptoms of autonomic hyperactivity often improve with treatment of fluid-retaining symptoms by dietary means. Severe symptoms by irritable bowel or urge frequency of micturition may require the prescription of an anticholinergic drug. In patients with severe urge frequency and incontinence of micturition, a referral to urologist for urodynamic studies and bladder retaining capacity may be necessary. Affective symptoms often improve with the treatment of fluid retention, but patients with severe anxiety and depression remain unwell and occasionally require psychiatric referral.

**Iatrogenic fluid Retention**

This ceases when the offending drug is stopped. Fluid retention related to thyroid disorder responds to specific treatment. Diabetic oedema is best managed by dietary measures to achieve ideal weight and the establishment of optimal metabolic control; diuretics may be required in some cases.

**Role of Diuretics**

The place of diuretics in the management of fluid retention is controversial. On one hand, the only treatment many patients receive is a diuretic prescribed by general practitioner for ‘bloating’, while on the other, it has been claimed that fluid retention is diuretic-induced illness [15].

According to the author’s experience, fluid-retaining symptoms have invariably preceded the prescription of a diuretic; women are reluctant to take diuretics and anxious to stop them. However, it is possible that diuretics abuse is seen in a few patients referred to specialized centers.

Diuretics are most effective in non-obese fluid retaining women with no history of weight gain or severe affective illness.

As weight increases or psychiatric illness becomes more severe, they become less effective. This may lead to the consumption of increasing doses of diuretic (especially loop diuretics) with the consequent risk of electrolyte depletion. Diuretics are usually ineffective in obese fluid-retaining patients or in patients with severe psychiatric disorder regardless of weight.

A thiazide diuretic with either a potassium supplement or potassium sparing diuretic should be prescribed if required. Loop diuretics should be avoided and spironolactone is no longer recommended for the treatment of fluid retention syndrome.

Many women find that diuretics are required only intermittently to control exacerbation of fluid retention related to the risk factors described above. If these removed or subside spontaneously, diuretics may be gradually reduced and discontinued. However, patients who discontinue diuretics should be warned that withdrawal may be followed by 1-2 weeks of self-limiting rebound edema.

**Role of other drugs**

Several other drugs have been used in the management of the fluid retention in small numbers of patients. These include bromocriptine, levodopa, dexamphetamine, propranolol,
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We will continue our ongoing work up to find our more cases to establish the syndrome as existing true entity in our current medical practice.

Acknowledgements

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References