Ketogenic diet to alleviate symptoms of gout
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Madam, gout, historically known as the “disease of the kings” or “rich man’s disease”, is a rheumatic complaint with chronic inflammatory arthritis. It is a disorder of purine metabolism characterized by deposition of monosodium urate (MSU) crystals in joints in the setting of persistent hyperuricaemia. The disease has a decided male preponderance and usually presents between 30 and 50 years of age. The estrogen, exerting a protective effect in premenopausal women through increasing the uric acid excretion in renal tubules. The typical manifestation is one of acute peripheral synovitis presenting with erythema, tenderness and swelling of the tortuous joint which resolves within 1-2 weeks.1 Though, frequent bouts can lead to chronic arthropathy, renal disease and cardiovascular dysfunction.

Treatment alternatives for gout are limited focusing mainly on prophylaxis based on urate lowering therapy (ULT) like Xanthine oxidase inhibitors (allopurinol, febuxostat). On the other hand, adherence to ULT is often subpar owing to precipitation of acute flares on intensive use2 which highlights the incipient need to devise alternate treatment possibilities for gout. Lately, restriction of carbohydrate intake with reduction in total calorie consumption has revealed promising outcomes in taming the rate of recurrence of gouty flares3 which makes dietary interventions a prospective contender against the current management options of gout.

A study by Goldberg et al has precisely proposed the practice of renowned ketogenic diet to alleviate the symptoms of gout.4 Ketogenic diet (KD) gained reputation in the 1990s as a potent weight loss regime and for its role in diabetes management. It is a high fat, moderate protein and low carbohydrate diet in which the body undergoes “dietary ketosis” with an equal production and consumption of ketones without causing the symptoms of ketonaemia.5 Already considered a long-term effective therapy for refractory paediatric epilepsy6 KD can be a prospective treatment modality for acute gout flare without compromising the immune status of the body.

Gout is prompted by the MSU crystal stimulation of macrophages which by the NLRP3 inflammasome mechanism causes the release of proinflammatory cytokine IL-1β which drives the acute inflammatory mechanism responsible for gouty flares. KD increases beta-hydroxybutyrate levels in the body that inhibit NLRP3 inflammasome, thus lessening the frequency and severity of these attacks.3 These verdicts have remarkable implications for reshaping the current standard of care in gout, if tested and found effective in clinical trials.

Even though some adverse effects of ketogenic diet including non-diabetic ketoacidosis, acute kidney injury and arrhythmias have been reported6 its latent as a cost-effective management option for gout cannot be ignored. Well-designed randomized controlled trials should be led to inaugurate its safety and effectiveness.

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References

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