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Short communication

Keto Flu: A Friend or Foe?

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Introduction

Ketosis denotes the creation of ketone bodies, from fats and amino acids used as an alternative to glucose during fasting or extreme carbohydrate restriction. limiting carbohydrate, by fasting or by decreased dietary intake, leads to decreased insulin levels, thus reduces fat buildup and lipogenesis. insufficient glycogen reserves leads to decreased supply of glucose whuch is essential for normal β-oxidation. Instead Acetyl-CoA is used in the production of ketone bodies via acetoacetyl-CoA and β -hydroxy- β-methylglutaryl-CoA that acts as fuel to the Central Nervous System (CNS), which regularly depends on glucose. This method of ketogenesis allows coenzymes to be unfettered for continued fatty-acid β-oxidation [1-8].

The normal levels of ketone bodies is very low (<0.3 mmol/L) related to glucose (approx. 4 mmol) As ketone bodies and glucose have a similar Km, the CNS begin to utilise the ketone bodies as an energy source when they reach a concentration of about 4 mmol/L which is close to the Km for the monocarboxylate transporter [9,10].

'keto-flu'

While shifting from a standard, complex carbohydrate diet to 'keto-induction' may lead to unlikable side effects. Due to increased urinary sodium, potassium and water loss in response to lowered insulin levels, keto-induction leads to headache, constipation, halitosis, diarrhoea muscle cramps, fatigue during 1–4 days of ketogenic diet as decreased glucose supply to brain occurs during 1–3 days. blood glucose normalises after fourth day. Constipation is due to decreased food volume or decrease intake of fibre These symptoms are called as 'keto-flu. These effects diminishes the efficacy, amenability and acceptability the of Keto diets [11].

Adverse effects are classified as mild, moderate, and severe or short term and long term. KD may also lead to GI discomfort mainly abdominal cramps, diarrhoea, and vomiting [12]. The moderate adverse effects are metabolic acidosis, dyslipidemia, mineral deficiencies, risk of renal stones. It may even raise triglycerides in 6 months [13.14]. reduced protein intake may lead to hypoproteinemia [15]. The severe effects are due to raised levels of ketones such as redox imbalance which leads to increased risk of mortality and morbidity in diabetic patients [16].

Long-term effects of KD

In mice, Due to insufficient insulin, insulin resistance, and reduced beta and alpha cell mass lead to glucose intolerance due to effects on pancreatic endocrine cells [17]. Other risks are raise in bone marrow and visceral fat,reduced insulin-like growth-factor 1, increased leptin, reduced transcription factors promoting osteoblastogenesis, reduced bone mineral density and decreased bone formation [18]. Plasma markers associated with inflammation and dyslipidemia triglycerides, leptin, cholesterol, monocyte chemotactic protein-1, Interleukin [IL]-1, and IL-6 were increased, and KD-fed mice showed signs of hepatic steatosis after 22 weeks of KD [17].

Conclusion and recommendations

Keto Diet is gaining attention but it has to be performed under strict medical supervision of dieticians and physicians to be operative and require hospital settings for its initiation. The diet protocols has to be modified to facilitate the, patient tolerability, acceptability and palatability. The knowledge of clinical impacts, safety, tolerability, efficacy, duration of treatment, and prognosis after discontinuation of the diet is provocative and necessitates further

research to recognize the disease-specific mechanisms. Exercise, smaller meals, increased fiber intake, increased sodium and fluid intake can often prevent or alleviate the complaints. To minimize complications, regular follow-ups has to be done. Future studies are mandatory to assess the long-term effects on health and reversing of diabetic complications in humans.

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