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# The Ketogenic Diet Revisited: Back to the Future

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## Introduction

Developed in 1920, when only bromides & phenobarbital were available for treatment of epilepsy, the introduction of Ketogenic Diet (KD) was greatly appreciated in clinical therapeutics. The diet gradually lost its popularity with advent of newer antiepileptic drugs & surgical techniques but there is resurgence of KD in management of intractable epilepsy of childhood in last few years.[1]

Ketogenic diet is comprising of high fat, low carbohydrate & adequate protein n ratio of 4 : 1 by weight with fat accounting for major source (90%) of energy. The high levels of ketone bodies produced by KD (ketonemia or ketosis) act as a source of energy for brain replacing the usual glucose & also contributes for it antiepileptic effect. The diet is primarily indicated as adjuvant therapy in medically refractory epilepsy of childhood and is effective against all seizure types (i.e. atonic, myoclonic, atypical absence) & epilepsy syndromes like Lennox-Gastaut syndrome, infantile spasm, tuberous sclerosis etc. KD is considered as life saving or treatment of choice in children with glucose transporter Glut-1 deficiency & pyruvate dehydrogenase deficiency and should be best avoided or even contraindicated in pyruvate carboxylase deficiency, fatty acid oxidation defects, mitochondrial disorder & acute intermittent porphyria etc.[2]

Being an unpleasant & bland diet with narrow food choice, the institution of KD under a strict dietary regimen pose a compliance challenge in young children. The efficacy of us is determined by its compliance. The common early adverse effect of KD includes diarrhoea, nausea, vomiting & hypoglycemia. The correction of hypoglycemia by intravenous glucose result in immediate loss of ketosis with resultant break thru seizure. The maintenance phase of KD is complicate by weight loss, hypoproteinemia, hyperlipidemia, growth retardation, pancreatitis & occurrence of renal stones.[3] A detailed metabolic screening of serum & urinary

aminoacids & lactate along with carnitine profile is mandatory before hand.

Ketogenic diet attribute to its antiseizure effect by producing major changes in basic physiology and biochemistry of central nervous system like adenosine upregulation, inhibition of excitatory glutamatergic transmission, activation of ATP dependent potassium pump & synaptic inhibition of motor pathways.[4] Associated caloric restriction & high levels of ketone bodies further add to its ability of seizure inhibition.

With use of KD, the antiepileptic drug in use should to be continued (with aim to gradual reduction to minimum or even withdrawal over a period of 2 years or so) until an optimal anticonvulsant effect of KD is attained. Since KD is not a supplementary diet so no additional food esp. rich in carbohydrate (biscuit, cold drinks, chocolates etc.) is permitted. At time of initiation of KD preferably an initial ketosis state is obtained by 24-72 hrs fasting & gradually KD with increasing ketogenic ratio 1 : 1 to 4 : 1 is initiated over a period of 4-5 days. Supplementation with essential vitamins & minerals is practised as KD is not a balanced diet.[5]

The clinical response of KD is visible in a week time but it takes around 3 months for its optimal anticonvulsant effect. A periodic check of urinary ketones (to keep 4+ i.e. 160 mg/dl) reflect adequate compliance. KD should be continued at least for 2 years or until the patient is seizure free off medications for a year.[6] The efficacy of KD is variable with 20% children show complete seizure control, 30% show reduction in seizure frequency up to 90% while the remaining show a substantial decrease in overall seizure frequency. Apart from its anticonvulsant effect the KD also produces improvement in cognition, behaviour, sleep pattern etc. Amongst the antiepileptics, sodium valproate is found to be more effective with KD.[7] Apart from it use in childhood epilepsy the KD because of its unique mechanism of action hold promises for its role in various diseases like amyotrophic lateral sclerosis (ALS), parkinson's disease, alzheimer dementia, diabetes mellitus as facture implication.

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