**CASE REPORTS**

**Wernike’s Encephalopathy as a Part of Refeeding Syndrome**

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**Abstract**

Cases have been reported about refeeding syndrome after bariatric surgery for obesity, in head and neck cancer patients, in patients with anorexia nervosa, hyperemesis gravidarum, in persons on hunger strike, malnourished alcoholic persons and persons doing religious fasting. Refeeding after prolonged fasting can cause severe morbidity and even mortality, if not done properly. Depletion of intracellular electrolytes, depletion of nutrients and vitamins, decreased BMR, decreased renal functions, decreased insulin, decreased GI functions all contribute to it, once you start refeeding. It takes sometime to regain the original functions by the organs and mismatch between supply and increased demand after refeeding can cause havoc.

Here we report the case of a person, who did water only fasting for 51 days and developed refeeding syndrome and Wernike’s encephalopathy four days after starting liquid diet.

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**Case Report**

A 55 year old male embarked on a religious fast, consuming only water all day.

He did his daily chores independently till the last day of his fast. Up to 40 days of fasting, he walked down daily 3 floors of stairs to get the blessings of his priest and climbed up, with the support of his wife as his building had no lift.

After 51 days, the completion of the fast was celebrated by feeding him tiny spoon of saffron-sugar-cardamom water by friends and relatives. The total amount must have been less than 100 ml. He sat for 2 hours during the ceremony.

Next day, he took frequent, small amount of Ker-water (fruits of desert plant-caparis decidua soaked overnight in the boiled water), and water of soaked black paper. On 3rd day diluted milk was added and quantity was increased. On 4th day moong-dal water was given instead of moong-water with a drop of ghee. He was independent for he felt imbalance while sitting. He was taken to the toilet by his wife as he swayed while walking. He was drowsy. His wife increased his feeds to combat weakness, but his drowsiness increased. He was arousable after repeated stimulation but preferred to keep his eyes closed.

On day-5 of refeeding, after giving a bath, his condition worsened.

They avoided medical treatment as they felt that medicines might do more harm than good for a person like him fasting for such a long time. They knew stories of people in such situation, deteriorating fast after hospitalization.

He was brought to an emergency department of the hospital in a pulseless state at 6 pm.

His BP was not recordable, extremities were cold and abdomen was distended. He was put on normal saline along with noradrenaline drip. The target was to maintain his BP at about 90-100 mm of Hg. Ryle’s tube aspiration was 1500 ml. His S. creatinine-3.2 mg%, S. Cal-6.4 mg%, S. Phos-2.8 mg% S.K. -3, S. Mg-1.6, Hb-13 gm% (dehydration), WBC count- 6000/cmm with 81% neutrophils and there was no acetone in the urine. His cardiac work up (EKG, 2D Echo) was normal. His Hb dropped down to 11.8 gm% after fluid replacement.

He came out of prerenal azotemia over a period of 2 days with intravenous normal saline and noradrenaline. He was put on RT feeding on 2nd day, 10 ml/hr, which was slowly increased to 40 ml/hr over a period of 4 days. Feeding was started cautiously as his problem had been precipitated by refeeding injury.

There was no focal neurological deficit. His deep tendon reflexes were sluggish. He moved all his extremities, but kept his eyes closed. Doll’s eye movements were normal. His T3-0.03 ng/ml, T4-3.31 ng/ml, and TSH-0.096 µIU/ml were low, with increased S. cortisol- 66 ug/dl. His B12 was 110 pg/ml. The thyroid replacement started at a low dose (25 microgram/day) on the third day to avoid sudden increase in his BMR.

S. insulin was on the lower side of normal hence daily blood sugar was done. His vit. D was less than 3 ng/ml. He was given inj. Vit. D along with oral calcium. He was on oral potassium supplements, along with diet rich in phosphates. There was a strict watch on S. electrolytes, and blood sugar. IV saline was given just to maintain his BP between 90 to 100 mm of Hg along with injectable B-complex. Intravenous fluids were given cautiously to avoid fluid overload. No IV or oral glucose or direct sugars were given after admission. As he was improving, relatives wanted to avoid brain imaging.

Even after 4 days of admission he was drowsy and kept his eyes closed. He was given bed side sitting on a recliner chair, on the 4th day of admission. He sat with eyes closed and responded monosyllabically after repeated tactile and verbal stimulation.

On the 5th day, during bed side sitting, he had generalized convulsions with tongue bite. His pulse and BP were normal as seen on the monitor. His S.

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Calcium was 6.8 mg% and Mg-2 mg%. EEG was suggestive of drowsy state.

MRI Brain report showed Symmetrical altered signal with restricted diffusion in bilateral medial thalami, hypothalamus, mammillary bodies and periaqueductal region of midbrain, tectum, dorsal medulla and precentral gyrus : Wernike’s encephalopathy.

Convulsions were attributed to hypocalcaemia and treated by increasing the dose of calcium and vit. D. He was not put on long term anticonvulsants except a loading dose of levetiracetam.

He was put on injectable Thiamine 100 mg 8 hrly. His alertness improved from 3rd day.

He was shifted to a general ward after one week. Within few hours of shifting to the ward he became febrile and dyspnoeic and his saturation dropped. His X-ray showed ?pneumonitis. He was shifted back to ICU. He received treatment for pneumonitis. He was afebrile during ICU stay. After 3 days, when he was reshifted to a general ward, he again got fever in the afternoon for a day and then became afebrile. His attention was better with proper eye contact. His gait was ataxic which started improving fast. He had sleep disturbances, disorientation to time and place which improved before discharge. Patient had amnesia of levetiracetam.

Symmetrical hyperintense signal with restricted diffusion on DW images is seen in bilateral medial thalami, hypothalamus, mammillary bodies and periaqueductal region including midbrain tectum, dorsal Medulla and in precentral gyrus (Figure 1).

He did not turn up for the check up. He came on 10th March 2017, when called to give consent for the article, he was not on any medicines. He stopped thyroid supplements after 3-4 months. His thyroid tests were normal after 2 months of stopping medicines. He had put on from 41 kg at the time of discharge to 59 kg at present.

**Discussion**

Complications of refeeding were described in severely malnourished American prisoners at Japanese war camps in world war II.1 Cases of cardiac arrest and delirium had been reported while refeeding adolescent girls with anorexia nervosa.2

Prolonged fasting is associated with decreased levels of all enzymes, hormones, micronutrients along with decreased functional capacity of gastrointestinal system and kidneys. The second important issue is that of ‘Wernike’s encephalopathy,’ which may get precipitated after refeeding. General condition of a patient can deteriorate after refeeding as in our case.

Our patient was admitted with prerenal azotemia, due to acute dialation of stomach after refeeding. On the 2nd day of admission, drowsiness was attributed to central hypothyroidism and exhaustion. After convulsions, MRI done, showed Wernike’s encephalopathy which explained all his symptoms.

His inattention and imbalance could be explained on the basis of bilateral thalamic involvement due to Wernike’s encephalopathy. The lack of febrile response to infection might be related to autonomic involvement as a part of Wernike’s encephalopathy. He had sleep disturbances with sometimes disorientation to time and place, related again to thiamine and B group vitamin deficiencies. His diarrhoea can be due decreased intestinal functioning during fasting, hence it occurred after after refeeding. Diarrhoea and vomiting can be a part of autonomic involvement due to thiamine deficiency. Prerenal azotemia was more due to hypotension than decreased renal functions during fasting.

Retrospectively, patient developed symptoms of thiamine deficiency in the form of imbalance, inattention, autonomic disturbances after getting ‘calories’ from the diet as he was totally independent before refeeding.

**How does thiamine deficiency get precipitated after refeeding?**

Let us see what happens during fasting: In early starvation, for 1st 24-48 hours, body uses sugar as a source of energy. Then, instead of using carbohydrate, body switches to use protein and fat as a main source of energy. BMR decreases by 20-25% during fasting. The central suppression of thyroid gland again contributes to lowering of BMR and preservation of energy.

During prolonged fasting, by hormonal and metabolic changes body tries to preserve proteins and muscles, and uses fatty acids. Resulting increase in ketone bodies in the blood stimulates brain to use ketones instead of glucose.
as its main source of energy. The liver decreases gluconeogenesis preserving muscle proteins.

During fasting, as in our patient, vitamin B1 deficiency should not occur with ‘zero calorie intake’ because thiamine is required as an enzyme for decarboxylation of alpha-ketoacids derived from glucose and amino acid metabolism. In a starving state, body uses fatty acids and brain uses ketone bodies. Thiamine requirement also decreases with lowering of caloric intake and decreased physical activity. Half life of thiamine is 9-18 days. It is excreted by kidneys. During prolonged fasting, renal functions are decreased and BMR is low, hence thiamine stored in the body is enough for person’s requirement during that period, but after refeeding, even with low calories its deficiency occurs.

The thiamine functions as, part time enzyme thiamine pyrophosphate –TPP, required for energy production, carbohydrate metabolism and nerve cell function. After refeeding, with increased BMR and shift of metabolism from fat to carbohydrate, its requirement suddenly increases and its absence precipitates, Wernike’s encephalopathy, Korsakoff’s psychosis, polyneuropathy, high output heart failure.3

Thiamine is absent in refined carbohydrates. Hence refeeding with sugary liquids can precipitate thiamine deficiency and even injections of 5% glucose can precipitate Wernike’s encephalopathy in such patients.

As early as 1974, Akerman WJ, reported a case of a 73/M, alcoholic person brought up to hospital in stuporose condition with bradycardia, hypotension and hypothermia who responded dramatically to rapid thiamine infusion.4

To heighten the awareness of Wernike’s encephalopathy in a non-alcoholic patient, there was a case report of a woman with 40 days water-only fasting5. The patient required thiamine supplementation for 6-8 months.

Dose of thiamine in deficient patients is 50 mg-100 mg and in patients with Wernike’s Encephalopathy it has to be injectable -100 mg three times a day for 5-7 days and continue oral replacement for 6 months.

Refeeding syndrome3 can be defined as the potentially fatal shifts in fluid and electrolytes that may occur in malnourished person after refeeding. These shifts may cause hormonal, metabolic changes and dangerous hypophosphatemia. But the syndrome is complex, and may cause sodium and fluid imbalance, change in glucose, protein and fat metabolism, thiamine deficiency, hypomagnesimia and hypokalaemia.

Anybody with negligible food intake for more than 5 days can develop refeeding syndrome

After prolonged starvation, one has to start refeeding with as low as 10kcal/kg/day with carbohydrate-50-60% Fat-30-40% and Protein-15-20%. IV or oral fluids restricted to zero balance with proper phosphate, potassium, magnesium supplementation and sodium restriction, along with IV thiamine and B complex vitamins 30 minutes before refeeding to prevent Wernike’s encephalopathy and dreaded refeeding syndrome. Slowly increase feeds daily by 5 kcal/kg/day over a period of 9-10 days as per patient’s tolerance. If there is intolerance, decrease or stop feeding and restart slowly with 5 kcal/kg/day.6

In short, refeeding after prolonged fast should be done slowly over a period of 9-10 days or even more before going for a normal diet and don’t forget to replace thiamine and other vitamins to avoid wernike’s encephalopathy even at the start of refeeding3 from day one.

Conclusion

Prolonged religious fasting shows strong will power but refeeding after fast requires patience. During refeeding after prolonged fasting, avoid overhydration, avoid glucose drips and give low sodium, low calories, high phosphate, potassium and magnesium diet along with intravenous replacement of thiamine and other B group vitamins from day one to avoid refeeding syndrome and Wernike’s encephalopathy, a reversible condition.

References