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Clinical and laboratory determinants of a person on religious fasting at rural medical college, North Gujarat, India

Dr. Urvesh A Mistry[1*], Dr. Ketan R Jansari[2], Dr. Bhogilal V Patel[3], Dr. Vinubhai C Singel[4], Dr. P C Patel[5]

[1]Professor, Department of Medicine, NMCRC, Visnagar, Gujarat, India

[2]Assistant Professor, Department of Medicine, NMCRC, Visnagar, Gujarat, India

[3]Associate professor, Department of Medicine, NMCRC, Visnagar, Gujarat, India

[4]Professor & Head, Department of Medicine, NMCRC, Visnagar, Gujarat, India

[5]Medical Superintendent, Department of Anesthesia, NGH & NMCRC, Visnagar, Gujarat, India

*Corresponding Author: **Dr. Urvesh A Mistry**

Email: drurveshmistry@gmail.com

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ABSTRACT

Background: There are difference in practicing fasting in different religions. Difference may be in form of patterns (continuous versus intermittent) or duration. Although fasting simply defined as intentional abstinence of food, pathophysiology that run behind fasting is similar to starvation ketosis.

Case presentation: We observe clinical and laboratory determinants of healthy person who voluntary choose to go on religious fasting of Chaitri for 9 days at Nootan medical college & research center, a rural medical college at Visnagar, Mehsana, Gujarat. He has not taken any food or even water and no any parenteral treatment was given. Routine Laboratory blood investigation was done on day of admission and on daily basis for 9 days. He was closely monitored for vital examinations and any symptoms development every 4 hourly.

Results and discussion: On day of admission, he was conscious and no signs of any disease present. On admission, his weight was 76 kg, height was 176 cm, BMI was 24.53 kg/m2 and physical examination was within normal limit. Baseline vital data shows pulse was 68/min, blood pressure was 124/88 mmHg, respiratory rate was 16 per minute and oxygen saturation was 99% on admission. His weight remains stable during whole course of fasting. Increase in pulse rate was noted after 5th day onwards and no significant change in blood pressure noted. There was increase in urine output noted on 5th day of fasting followed by subsequent decrease because of ketosis and natriuretic property of ketone bodies. Routine laboratory blood investigation like complete blood count, renal function test and liver function test was within normal limit on date of admission and there was no significant change during fasting. His fasting blood sugar was 86 mg/dl on admission and decreased to level of 64mg/dl on last day of fasting, however no any symptoms of hypoglycemia was noted. Serum acetone was absent on day of admission but very next day it become positive and remain positive for next eight day of fasting. Urine acetone was trace on 2nd day of fasting and there was increased in grading as fast progress. Hormonal assay of insulin and growth hormone showed decremental response in insulin level and incremental response in growth hormone level. It shows that transition from the food replenished state to fasting and into prolonged starvation is mediated by a series of complex metabolic, hormonal and glucoregulatory mechanisms.

Conclusion: The body adapt to long-term fasting by increase in utilization of alternate source of fuel like ketone bodies and sparing need of glucose. The remarkable ability of the body to adapt to long-term starvation has been critical for survival in adverse situation.

Keywords: Fasting, Clinical examination, Laboratory parameters

BACKGROUND

Fasting is routinely practiced in different religions like Hinduism, Jainism, Buddhism, Muslims. There may be difference in patterns of fasting (continuous versus intermittent) or duration of fasting. Although fasting simply defined as intentional abstinence of food, pathophysiologic mechanism that come into play for sustaining life during this period is similar to starvation ketosis. [1][2]

Food supplies carbohydrates, protein and fats as an important energy source. In normal feeding state carbohydrates is mainly utilized for energy production and under the effect of insulin, excess carbohydrate will be store as glycogen in liver and protein & fats are stored in muscle and adipose tissue, respectively.

In cases of fasting, where carbohydrates have been depleted, insulin level are reduced and glucagon & epinephrine level are elevated in blood, which leads to proteolysis in muscle and lipolysis in adipose tissue. Triglyceride derived from lipolysis, are converted to free fatty acid and glycerol. Ketone bodies are generated from oxidation of these free fatty acids to fulfill the metabolic needs of tissues, especially the brain.

Ketone bodies like acetoacetate, beta hydroxybutyrates and acetone, produced by liver are always present in body in minimal state when person is not fasting. However, during fasting ketone bodies are present in abundance & mainly utilized for energy need of body tissue. [3][4]

Once treated with adequate carbohydrates, the insulin level will increase and counter-regulatory hormone levels will be reduced, and ketosis will be resolved.

We observe clinical and laboratory determinants of healthy person who voluntary choose to go on religious fasting for 9 days and excluding other possibilities diagnosis of starvation ketoacidosis was made.

CASE PRESENTATION

A 52-year-old male Hindu person without any comorbidities admitted for observation with predetermined fasting of Chaitri Navratri from 9 April to 17 April 2024 at Nootan medical college & research center Visnagar.

During this 9 days duration of fasting, he has not taken any food or even water, no any parenteral treatment was given, and this was closely monitored by continuous CCTV surveillance.

Physical examination was done on day of admission. Every day he was closely monitored for vital examinations like temperature, pulse rate, blood pressure, respiratory rate, oxygen saturation and any symptoms development every 4 hourly.

Routine laboratory blood investigation like Complete blood count, Renal function test with electrolytes, Liver function test, fasting blood sugar, serum & urine acetone was done on day of admission and on daily basis for 9 days. Daily weight chart and urine output chart was made. Urine acetone was performed with rapid dipstick method and urine acetone chart was made.

RESULTS

On the day of admission, he was conscious, cooperative & well oriented to time, place & person. There was no any signs of disease present and no significant past history of any chronic medical illness present.

On physical examination before commencement of fast, his weight was 76 kg and height was 176 cm. On general examination, his skin was pink and the head-to-toe examination shows no

any abnormalities. Baseline vital data shows pulse was 68/min, blood pressure was 124/88 mmHg, respiratory rate was 16 per minute and oxygen saturation was 99% on admission.
Examination of chest showed no any abnormalities and lungs were clear on auscultation. On cardiac examination, normal first and second heart sound on auscultation at apex and no any murmur present. The abdomen was soft, nontender and without evidence of hepatosplenomegaly. On neurologic examination there were no any abnormalities found.
Baseline electrocardiogram and chest Xray was within normal limit.
His weight was 76 kg on day of admission and remain stable during whole course of fasting. His urine output was 800 ml on day of admission, increased to 1400 ml on day 5 of fasting, and followed by subsequent decrease to 1100 ml on last day.
As seen by Kerndt, P. R et al, weight loss in the first 1–5 days of fasting was average of 0.9 kg per day and gradually slows to an average of 0.3 kg per day over the subsequent 3 weeks. The rapid initial weight loss is primarily due to salt and water diuresis. [5]
At low plasma concentrations, filtered ketone bodies are completely reabsorbed by the saturable Na+-coupled transporters in the proximal tubule. Ketonuria develops as plasma levels rise and the filtered load of ketoacid salt increases. The loss of Na+ coupled acetoacetate and β-hydroxybutyrate in the first of several days of fasting results in salt and water diuresis and negative Na+ balance and weight loss.[5][6][7]
Therefore, as seen by Kerndt, P. R et al, it is also noticed in our case that urine output has increased on day 5 as compared to day 1 because of natriuretic property of ketone bodies. Hormonal analysis in our case showed incremental response in growth hormone level and being potent anabolic hormone, there was no significant weight loss observed in our case.

Table 1: Showing Pulse and Blood pressure change during fasting

	Day 1	Day 3	Day 5	Day 7	Day 9
Pulse/min	68	72	68	80	84
Systolic BP mmHg	124	120	122	116	110
Diastolic BP mmHg	88	80	78	80	78

Base line pulse rate was 68/min on day of admission and on last day of fasting his pulse rate was 84/min. There was no significant change in pulse rate during first five days of fasting but after that, shows rise in pulse rate. Fasting cause impact on heart rate by several factors like increase energy demand, hormonal change & stress response. Mazurak, N. et al also noticed fasting induce a significant decrease of mean interbit intervals (IBIs) in his study. [8]
In our case blood pressure on admission was 124/88 mmHg and on last day 110/78 mmHg. There was no significant change in blood pressure noted during this fasting period and no any symptoms of orthostatic hypotension noted.

Table 2: Showing laboratory parameters during fasting

		Normal Value	First day of fasting	Last day of fasting
CBC	Hb	13-17gm%	13.5	14.3
	TC	4000-11000/cumm	5000	4700

	PLT	1.5-4.5 l/cumm	237000	238000
RFT	Urea	15-45mg/dl	17	18
	Creatinine	0.6-1.5	1	1
Electrolyte	Na	135-150mmol/l	140.4	136.7
	K	3.5-5.2mmol/l	4.05	3.66
LFT	Bilirubin Total	0.2-1mg/dl	0.65	0.83
	Direct	0-0.3mg/dl	0.26	0.25
	Indirect	0.3-0.8mg/dl	0.39	0.58
	S Protein	6.3-8.2gm/dl	6.53	6.67
	Albumin	3.4-5 gm/dl	4.35	4.34
	Globulin	2.3-3.5 gm/dl	2.18	2.33
	SGPT	0-45 u/l	20	14
	SGOT	0-40 u/l	19	16
	ALKPO4	53-128 IU/l	77	84

Routine laboratory parameters like Complete blood count, renal function test with electrolytes, Liver function test showed no significant changes during this nine days of fasting periods.

As shown in table 2 serum sodium level has decreased to 136.7 mmol/L on last day as compared to 140.4 on first day of fasting and serum potassium level has decreased to 3.66 mmol/L as compared to 4.05 mmol/L on first day of fasting. Serum electrolyte level of sodium and potassium decline slightly but not fell below lower limit of normal.

Natriuresis and Kaliuresis was observed by Kerndt, P. R et al in his observation of fasting pathophysiology. Natriuresis, in large part, was due to the generation and high early excretion of ketone bodies. The magnitude of natriuresis begins to decrease as ammonia genesis increases, allowing NH4 + to replace Na+ as the major urinary cation.

Table 3: Showing laboratory parameters of Sugar and Acetone during fasting

	Normal Value	Day 1	Day 3	Day 5	Day 7	Day 9
HbA1C	4-6.5%	5.4				
Fasting Blood sugar	70-110mg/dl	86	72	72	63	64
Serum Acetone		Absent	Present	Present	Present	Present
Urine Acetone		Absent	Present (+)	Present (++)	Present (+++)	Present (+++)

His fasting blood sugar was 86 mg/dl on admission and glycosylated hemoglobin was 5.4. There was gradual decrease in blood sugar level and on last day blood sugar was 64 mg/dl, however there was no any sign and symptoms of hypoglycemia was observed.

Serum and urine acetone was absent on day of admission but very next day they become positive after 24 hour of fasting and remain positive for next eight day of fasting. Urine acetone was trace on 2nd day of fasting and increased to grade + (10mg/dl) on third day, grade ++ (50mg/dl) on 5th day and grade +++ (100mg/dl) on seven days onwards.

Table 4: Showing hormonal assay

	Normal Value	First day of fasting	Last day of fasting
S insulin level	6.0-27 microU/ml	1.8	1.7
Growth Hormone level	Male 3ng/ml Female 8ng/ml	1.2	1.4

Hormonal assay of insulin and growth hormone showed value below lower limit of normal on first day of fasting however, serum insulin level has decreased on last day of fasting as compared to first day of fasting and growth hormone has increased level on last day of fasting as compared to first day of fasting.

DISCUSSION

There are many causes of ketoacidosis but important are diabetic ketoacidosis, starvation ketoacidosis and alcoholic ketoacidosis. Other causes of ketoacidosis include salicylate intoxication, SGLT2 inhibitor therapy, and calorie sufficient but carbohydrate-restricted diets. Similarities and metabolic consequences of ketogenesis is important, because of clinician to encounter one of this situation frequently. This case study describe the metabolic changes occur in normal healthy individual undergo fasting for prolonged period.

In the first 24 h of fasting, completion of dietary glucose absorption leads to a fall in blood glucose levels, leading to a decrease in insulin and increase in glucagon levels. Glucagon stimulates the release of glucose from glycogen stores in the liver, while the fall in insulin decreases transport of glucose into the skeletal muscle and adipose tissues certaining an adequate amount of blood glucose is available for the brain. [9]

As glycogen, stores become exhausted after 24 h of fasting, patients enter a gluconeogenic phase where substantial amounts of gluconeogenic precursors derived from amino acids are channeled to liver for synthesizing glucose. A persistent decrease in insulin levels promotes proteolysis in the muscle, providing the needed supply of amino acid for increasing hepatic gluconeogenesis. [10][11]

These amino acids are preferentially catabolized in the skeletal muscle to their α-keto acids and this increased concentration of keto acid exert an inhibitory effect on gluconeogenesis.

Reduced insulin levels activate lipolysis making fatty acids available to serve as an alternative fuel for the skeletal muscle in the later stages of the postabsorptive phase. The flux of fatty acids to the liver continues to increase during the gluconeogenic phase and is primarily directed to generation of ketone bodies. [12]

The protein conservation phase is characterized by a change in fuel utilization preference to maintain blood glucose and spare protein from continual degradation. During this phase, there is increased production of ketone bodies, which are utilized by brain in substitution for glucose. Ketone bodies directly inhibit muscle proteolysis and contribute to adaptation to prolonged fasting. [13][14][15]

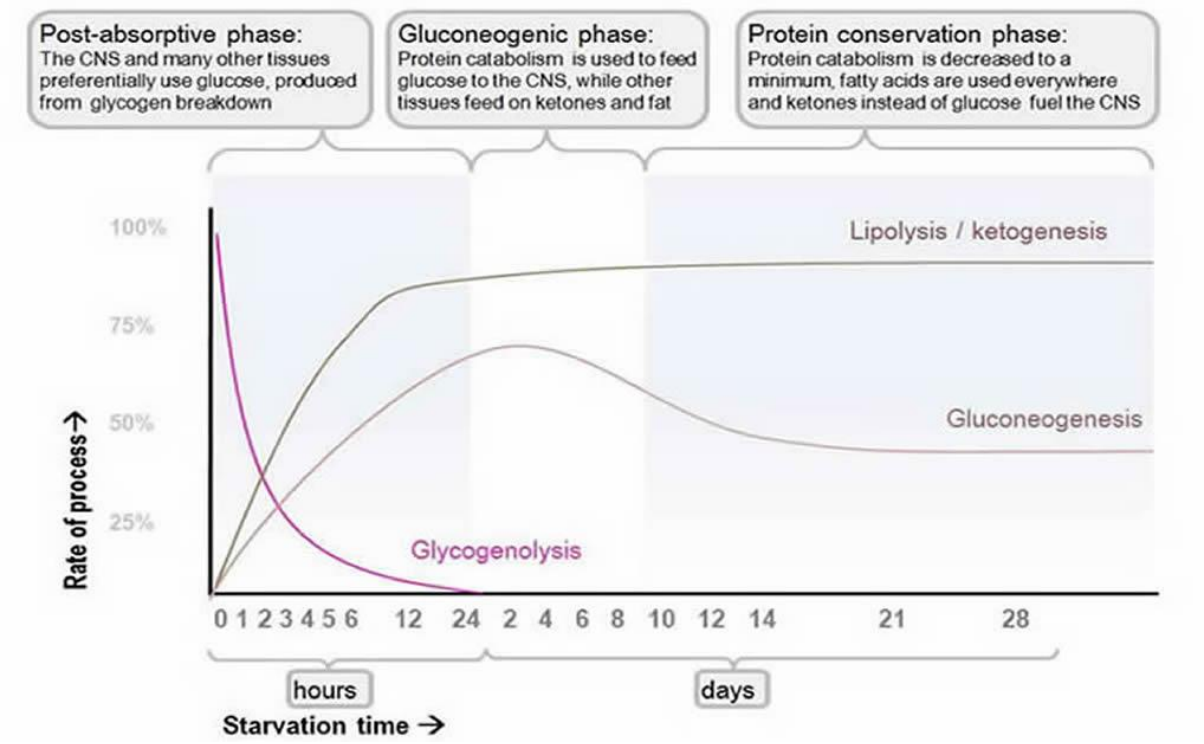

Figure 1: Showing fuel utilization in different phase of starvation and interrelationship between body fuel stores and the time sequence for their mobilization via glycogenolysis, gluconeogenesis, lipolysis and ketogenesis pathways. This shows that transition from the food replenished state to fasting and into prolonged starvation is mediated by a series of complex metabolic, hormonal and glucoregulatory mechanisms.

Management of starvation ketoacidosis in this person is replacement of glucose, as hypoglycemia is a trigger factor for ketone body formation. So as per predetermined course of fasting for nine days person, break his fasting by taking oral fluids and glucose on 10^{th} day and was discharged after assuring stabilized vitals and biochemical laboratory parameters.

CONCLUSION

The body adapt to long-term fasting by increase in utilization of alternate source of fuel like ketone bodies and sparing need of glucose. Shift in energy source utilization decrease mobilization of substrate amino acid from muscle, thus providing means to conserve protein. The remarkable ability of the body to adapt to long-term starvation has been critical for survival in adverse situation.

LIMITATION

Serum acetone was measured qualitatively and urine acetone was measured by dipstick method in this observation. Quantitative measurement helps in detailed evaluation of case of starvation ketosis.

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