

Iatrogenic Dumping Syndrome

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Abstract

Dumping syndrome is a common post surgical complication after gastric surgery and is caused by large amount of food passing quickly into the small intestine. It is accompanied by a number of gastrointestinal and vasomotor symptoms due to changes in the anatomy and physiology of the stomach caused by gastric surgery. In this paper we report an interesting case who had not undergone any surgery of the stomach and yet displayed symptoms of 'late dumping syndrome' after food, whenever he was on proton pump inhibitors. The symptoms of late dumping were not observed when the PPI was discontinued.

Introduction

Dumping syndrome is a relatively rare disorder in which the stomach contents are delivered too quickly to the small intestine. It occurs as a physiological reaction to the consumption of too much simple or refined sugar in some persons, when simple sugar exits the stomach too rapidly it attracts fluid into the upper intestine, and the blood volume decreases as it attempts to absorb the sugar.¹ Dumping syndrome is one of the unwanted post operative sequelae to partial gastrectomy. This symptom complex is believed to be due to rapid emptying of the gastric contents into the upper small intestine and also an increased transit rate through the small bowel itself right through the colon. As a consequence of this dumping a large volume of hyperosmotic fluid enters the small intestine. In an attempt to bring the osmotic tension to the physiological range large amount of fluid cross the intestinal mucosa. As a result the plasma volume is reduced which in turn exerts a vasomotor

effect.² The vasomotor symptoms comprise general weakness, pallor, sweating, palpitation and light headedness. Another set of symptoms of gastrointestinal disturbance such as epigastric discomfort, nausea, vomiting and possibly an episode of diarrhoea is observed. The biochemical changes that occur in dumping syndrome are hyperinsulinaemia followed by hypoglycaemia due to rapid transport and absorption of food from small intestine after a meal. Since the conversion of glucose to glycogen and its storage consumes potassium, its level in blood decreases in dumping syndrome and hence hypokalaemia may be seen too. These symptoms classically occur some 5-30 minutes after eating and are more marked if the meal is large particularly if it contains a substantial amount of carbohydrate and liquid. The choices for managing dumping syndrome include dietary changes, medications and surgery.³⁻⁶ Although dumping syndrome classically develops after gall bladder surgery, it may also occur after other abdominal operations, such as duodenal ulcer surgery or surgery for severe reflux.⁷⁻⁸ Sometimes it may be seen in people born with unusually small stomach and very rarely in those with stomach abnormalities. Here we report a

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unique and interesting case who had not undergone any surgery of the GI tract and yet manifested the symptoms of dumping whenever he was treated with proton pump inhibitors (PPI) and was relieved of the same when the treatment was discontinued.

Case Report

A 60-year-old male presented with complaints of uneasiness of chest and flushing 2 hours after taking proton pump inhibitors (PPI). The patient passed large bulky stools following which he developed lethargy, myalgia and extreme prostration. He was unable to even stand, however no fainting episodes occurred. The patient was on Pantoprazole a PPI for management of his acid peptic disease. The patient discontinued PPI for about five months during which he was normal without the above symptoms. But when he was restarted with the PPI he developed the above symptoms again. He was investigated for the same and since the symptoms were more in alignment with dumping syndrome, he was subjected to test for dumping.

Test for Dumping

Initially blood test was done to determine the baseline values of all parameters included in the study. Next the patient was given a carbohydrate feed and was kept under observation. This consisted of 75 g of dextrose and a breakfast of 2 dosas with tea. Two hours after this intake, the patient had a feeling of bowel mass evacuation reaction and passed a very large bulky stool. Thereafter the patient felt extreme weakness, tiredness and felt like lying down to take rest. After being in a supine position for 10 minutes he felt relieved. Simultaneously his vitals were recorded and blood samples were withdrawn for estimating sugar, insulin and electrolytes at regular intervals after taking the meal. Corresponding urine samples were also collected for investigations and his ECG was also recorded.

Discussion

When the signs and symptoms of dumping occur during a meal or within 15 to 30 minutes following a meal it is called early dumping and if it occurs after 2 hours, it is called late dumping. Some people experience both early and late dumping signs and symptoms. The subject in this study

experienced late signs like extreme weakness, fatigue after 2 hours and lay down to take rest for about 10 minutes after which he felt better.

In this case of dumping syndrome, no matter when problems develop, the symptoms were worse in the aftermath of a high carbohydrate meal, especially one that is rich in sugars such as sucrose (table sugar) or fructose (fruit sugar). In concordance in this patient also these symptoms were observed two hours after he consumed a meal of dosas and 75 gms dextrose in water. Some people also experience hypoglycaemia related to excessive levels of insulin delivered to the blood stream. In this patient too the blood sugar levels increased to 146 mg/dl after 1 hour and subsequently decreased to 74 mg/dl after 2 hours. Insulin levels increased to 249.10 μ IU/ml at the end of 30 minutes and it was 163.10 μ IU/ml after 1 hour. This can be explained as follows. The hyperinsulinaemia is due to rapid passage of food to the small intestine. The high concentration of carbohydrates in this region results in rapid absorption of glucose and hence hyperinsulinaemia. This in turn was responsible for the decrease in glucose levels seen in this subject after 2 hours. In this subject hypokalaemia was observed because potassium level was 2.9 mmol/l at 30 minutes after the meal, and the corresponding insulin and glucose levels were 249.1 IU/ml and 130 mg/dl respectively. Table 1 gives the values of the various parameters tested. Since their introduction in the late 1980's proton pump inhibitors have demonstrated gastric acid suppression superior to that of histamine H₂-receptor blockers and have enabled improved treatment of various acid peptic disorders. PPIs are absorbed in the proximal small bowel and cross the parietal cell membrane and enter the acidic parietal cell canaliculus. In the acidic environment PPI becomes

Table 1 : Shows the various parameters recorded including vitals and blood investigations

Sample No.	Temp. °F	Pulse	Vitals			Blood Samples				Insulin mIU/ml
			Resp. Rate	B.P. mm Hg	Na mmol/l	K mmol/l	Sugar			
							Blood	Urine		
I. Resting (To give 75 gms of Glucose orally)	98.4	74/mt	22/mt	110/70	130	3	75	Nil	18.63	
II. At the end of 15 mts	98.2	72/mt	24/mt	110/60	135	3.3	117	Nil	59.82	
III. At the end of 30 mts	98.4	74/mt	20/mt	120/80	129	2.9	130	Nil	249.10	
IV. At the end of 1 hr	98.2	72/mt	24/mt	110/70	127	3.0	146	Green	163.10	
V. At the end of 2 hrs	98.4	74/mt	22/mt	110/70	132	3.2	74	NA	40.34	

protonated which is the active form that binds to the H⁺/K⁺ AT Pase enzyme and causes irreversible inhibition of acid secretion by the proton pump. The most striking feature of this case was however the fact that the dumping syndrome manifested when the patient was put on proton pump inhibitors. The symptoms of dumping syndrome disappeared when he was off the same.

Conclusion

Gastric mucosal function is altered by surgery and acid and enzymatic secretions are decreased. Also hormonal secretions that regulate the gastric phase of digestion are adversely affected after gastric surgery and as a consequence dumping syndrome manifests in the patient. Whether prolonged use of PPI lead to alterations in acid secretion and inturn resulted in a condition similar to dumping syndrome as evidenced in the case of the patient described in this paper is debatable.

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