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Research Article

**GASTRIC AND ESOPHAGEAL EMPTYING IN PATIENTS
WITH TYPE 2 DIABETES MELLITUS****¹Dr Rao Farhan Saleem, ²Dr Own Abbas, ³Dr Nimra Anwar.**
^{1,2,3}MBBS, Akhter Saeed Medical and Dental College, Lahore.**Article Received:** September 2020 **Accepted:** October 2020 **Published:** November 2020**Abstract:**

A very common complication in diabetes mellitus is abnormal gastrointestinal motility. In diabetic patients gastric and oesophageal symptoms are very remarkable due to gastrointestinal symptoms and it leads to delayed gastric emptying which consequently increases the level of blood glucose. A study conducted on type 1 diabetic patients has demonstrated that there was 42% delay in solid bolus in oesophageal emptying whereas there was 56% delayed in gastric emptying solid or liquid component. Studies have reported that there is more significant gastrointestinal motility disorder in type 2 diabetes mellitus as compare to type 1.

A test which is known as 9 emptying was performed. First of all 5ml of water was taken as a lubricant after that the participants was allowed to take the solid meal and was command to swallow the after every 15s. Meanwhile the cricoid movement was analyzed in the monitor to verify that bolus had entered esophagus. Until the bolus reached stomach this test was continued in monitoring. Analysis was performed by computer-drawn regions of interest corresponding to the oesophagus and the stomach. The time for 95% of the radioactivity to enter the stomach was calculated.

Although plasma glucose and glycosylated haemoglobin concentrations correlated closely in current, there was no significant relationship between gastric emptying and glycosylated haemoglobin.

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INTRODUCTION:

A very common complication in diabetes mellitus is abnormal gastrointestinal mobility. In diabetic patients gastric and oesophageal symptoms are very remarkable due to gastrointestinal symptoms and it leads to delayed gastric emptying which consequently increases the level of blood glucose. A study conducted on type 1 diabetic patients has demonstrated that there was 42% delay in solid bolus in oesophageal emptying whereas there was 56% delayed in gastric emptying solid or liquid component. Studies have reported that there is more significant gastrointestinal motility disorder in type 2 diabetes mellitus as compare to type 1.

We have used sensitive scintigraphic techniques to determine the prevalence of abnormal gastric and esophageal emptying in 50 Type 2 diabetic subjects. The relationships between gastric emptying, esophageal emptying, gastrointestinal symptoms and autonomic nerve function and glycemic control have been examined.

SUBJECTS AND METHODS:

It was a cross-sectional study. 50 participants were recruited in the study equally male and female. The mean age was ranged between (41-62). The body mass index was 28.3. The participants were selected randomly. All the participants who were using oral hypoglycemic drugs were recruited into the study. Patients who were taking medicine other than oral hypoglycemia were excluded from the study. The mean duration of known diabetes was 8 years (range 1-20), and the majority of the patients had other complications of diabetes mellitus including nephropathy, retinopathy and peripheral neuropathy. Delayed gastric or esophageal emptying due to organic obstruction was excluded by upper gastrointestinal endoscopy. An informed consent was taken after explaining the purpose of study. On one day, each diabetic patient underwent: (a) a subjective assessment of gastrointestinal symptoms, (b) an objective assessment of diabetic complications of autonomic neuropathy, peripheral neuropathy and retinopathy, (c) an assessment of glycaemic control, (d) measurement of gastric emptying of a mixed solid and liquid meal, and (e) measurement of oesophageal emptying of a digestible solid bolus. On the study day, smoking was prohibited, and no patient took any medication until after the completion of the tests. Gastric and 9 emptying measurements were also performed. All the participants were non-smokers, having no other medication with no evidence of

gastrointestinal disease. The diabetic group was having significantly higher BMI.

Sympathetic function was assessed by the fall in systolic blood pressure in response to standing. In response to standing sympathetic function was assessed by fall in systolic blood pressure. The scoring was done according to Ewing and Clarke. Definite autonomic nerve damage was indicated by score more than 3. Retinopathy was graded as none, background or proliferative on the basis of a recent ophthalmological assessment, which often included fluorescein angiography. Peripheral neuropathy was diagnosed clinically when absent ankle reflexes were associated with either sensory or motor changes.

A standard questionnaire was used to assess the gastrointestinal symptoms before performing the gastric emptying tests. The maximum possible total score for the gastric symptoms was 18 and was 9 for the 9 symptoms. The bowel regulatory was noted each week which includes the consistency of bowel and presence and absence of nocturnal diarrhea and fecal incontinence. At least three spontaneous bowel actions were required each week to avoid constipation. Hemoglobin A1c was indicated in the form of percentage the normal range considered is 3.0-6.0%. Time zero was defined as the time of meal completion, and then each study was continued for at least 2 h. The 50% emptying time for the solid meal was not used because some patients did not reach 50% emptying in the study period. Similarly, in some patients with markedly delayed emptying the linear emptying rate could not be determined accurately. For the liquid component, the percentage remaining at 10 min after meal completion and the time for 50% emptying (T50) were obtained. The percentage of liquid remaining at 10 min was used as an index of the early phase of gastric emptying.

A test which is known as 9 emptying was performed. First of all 5ml of water was taken as a lubricant after that the participants was allowed to take the solid meal and was command to swallow the after every 15s. Meanwhile the cricoid movement was analyzed in the monitor to verify that bolus had entered esophagus. Until the bolus reached stomach this test was continued in monitoring. Analysis was performed by computer-drawn regions of interest corresponding to the oesophagus and the stomach. The time for 95% of the radioactivity to enter the stomach was calculated.

RESULTS:

The rate of gastric emptying in in both groups diabetic and control group was slower than liquid. In

the diabetic patients there was remarkable delay in solid food emptying. The percentage retention of solid food at 100 min was greater than the upper limit of the control range in 20 of the 50 diabetic subjects (30%). The rate of liquid meal was slower in diabetic patients than control group. But there was no significant difference in remaining the liquid at 10 min in both control and diabetic group. The percentage retention at 10 min was greater than the upper limit of the control range in fifteen of the diabetic patients (25%) and less than the lower limit in eight patients (10%). The autonomic nerve dysfunction was significantly correlated with the age but not with the length of duration of diabetes mellitus. Six patients clinically had peripheral neuropathy. Fifteen patients had no retinopathy, four patients had background retinopathy; and one patient had proliferative retinopathy. Glycaemic control Plasma glucose and HbA_{1c} concentrations varied considerably between patients

DISCUSSION:

The study has demonstrated that there is high prevalence of gastric motor dysfunction in type 2 diabetes mellitus as compared to type 1. Participants with longer duration of diabetes mellitus were having high prevalence of complications such as peripheral neuropathy, retinopathy and autonomic neuropathy. There is very slight effect of aging on gastric emptying but there is significant difference in body weight between control and diabetic group.

On the basis of previous results in patients with Type 1 diabetes mellitus, we would predict that the majority of Type 2 diabetic patients with slow gastric emptying of radioisotopically labelled meals would have a normal result with liquid barium sulphate. The normal stomach empties liquid, digestible solid and non-digestible solid meal components at different rates and with different patterns, but the motor mechanisms controlling this are poorly defined. The gastro-duodenal pressure gradient, which is influenced by proximal gastric tone and pyloric and duodenal resistance, is of major importance in the control of liquid emptying. In controlling gastric emptying of digestible solid food grinding action of the antrum has a major influence by contrast. The current study has observed that there is diminished activity of proximal stomach and antral motor activity in patients with type 2 diabetes as compared to type 1 diabetic patients. One of the common disorder known as disordered pyloric motility also causes delayed motility.

Gastric bezoar has been reported in these patients and abnormalities of gastric emptying have been

demonstrated in two studies, which have measured gastric emptying in small numbers of selected patients with Type 2 diabetes mellitus.

Leatherdale *et al.* studied gastric emptying of a porridge meal in 10 patients with longstanding Type 2 diabetes mellitus and in 10 control subjects. The results of this study may be questioned because of probable technical inaccuracies, but the time for 50% of the "peak content" of the meal to empty from the stomach was longer in the diabetic patients. Sasaki *et al.* reported that gastric emptying of a water load was slower in a small group of obese Pima Indians with Type 2 diabetes mellitus compared to obese control subjects. In two of our 20 diabetic patients, gastric emptying of the liquid meal was faster than any control subject. This more rapid initial emptying has been demonstrated in Type 1 diabetes mellitus patients and may reflect impaired proximal stomach accommodation to distension, or abnormal pyloric motility. The emptying of larger non-digestible solid particles, which is dependent on the integrity of phase 3 of the gastric interdigestive myoelectric complex, was not assessed in our study, but in symptomatic Type 1 diabetic patients gastric interdigestive myoelectric complexes are characteristically absent, and gastric emptying of non-digestible solid particles is markedly delayed. This study demonstrates that the poor correlation between gastrointestinal symptoms and gastric emptying in Type 1 diabetic patients also exists in Type 2 diabetes mellitus patients. Although some patients with gastrointestinal symptoms had delayed gastric emptying, it also occurred in asymptomatic patients. The relatively high prevalence of gastrointestinal symptoms is not unexpected, and it is probable that gastrointestinal complications represent a generally underestimated cause of morbidity in both forms of diabetes mellitus. A high prevalence of autonomic nerve dysfunction in Type 2 diabetes mellitus patients has been reported, and it is well documented that abnormal autonomic nerve function tests precede the development of symptomatic autonomic neuropathy. The absence in this study of a significant relationship between gastric emptying and the severity of autonomic nerve dysfunction differs from findings in Type 1 diabetes mellitus patients and has several possible explanations. Only 4 of the 20 patients had no evidence of autonomic neuropathy, and it is possible that minimal abnormalities in cardiac autonomic nerve function are associated with disordered gastric motility. In addition, the relative impairments in sympathetic vs parasympathetic gastric innervation are likely to influence gastric emptying. It is also possible that in the diabetic autonomic neuropathy syndrome gastric and cardiac systems may be affected differently, and

that vagal gastric damage may not necessarily be inferred from cardiac vagal dysfunction, but this hypothesis conflicts with the demonstration of a strong association between gastric acid output impairment and cardiac autonomic neuropathy. The observation that the liquid lag period and the solid lag period are related to plasma glucose concentrations in Type 2 diabetic patients is consistent with our observations in Type 1 diabetes mellitus patients, although not with those made by other workers. Induced hyperglycaemia in normal subjects slows gastric emptying of nutrient-containing liquid meals and decreases fasting antral contractility and gastric phase 3 activity. The poor reproducibility of gastric emptying that has been observed in Type 2 diabetic patients, and the variable observations on liquid gastric emptying in Type 1 diabetic patients may possibly be partly ascribed to variations in plasma glucose concentrations. The mechanism(s) responsible for the inhibitory action of hyperglycaemia on gastric motility is not known. Hyperglycaemia may suppress vagal nerve activity and be an important factor in the aetiology of autonomic nerve dysfunction. Alterations in gastrointestinal hormone secretion (such as motilin, pancreatic polypeptide, somatostatin, glucagon, gastrin and gastric inhibitory polypeptide) may be important. Although plasma glucose and glycosylated haemoglobin concentrations correlated closely in current, there was no significant relationship between gastric emptying and glycosylated haemoglobin.

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