Introduction
To date, diabetes mellitus is a metabolic disease of multiple etiologies characterized by the presence of hyperglycemia resulting from a defect in the secretion of insulin that is associated with disorders of carbohydrates, fats and protein metabolism [1].

World statistics of Diabetes Mellitus (DM) in 2013 showed that 382 million people in the world had the disease; type II diabetes mellitus accounted for 90% of cases.

Diabetic gastroparesis is a serious complication due to diabetes mellitus. Further studies suggest that diabetes mellitus is associated with up to 75% of gastrointestinal symptoms, in diabetic gastroparesis; the antral body contractions are ineffective, although the smooth muscle layers appear to be normal.

Diabetic gastroparesis is a syndrome characterized by a delay in gastric emptying [6]. Gastric emptying scintigraphy is considered the gold standard for diagnosis and quantification of delayed gastric emptying. The goal of treatment is to reduce symptoms, correct nutrition, and improve quality of life. Includes prokinetic medications like first line, nausea and vomiting can be controlled with some antiemetic medications, it should also include education and support diet, behavior changes.

Epidemiology
The prevalence is higher in women, 5.8% more than in men (3.5%) according to a study.
conducted in the United States in 2017 [12].

Diabetic gastroparesis is associated in a variable way with lower unemployment, lower household income and absenteeism [13].

These factors increase the incidence of hospitalizations due to gastroparesis; these include an increase in the prevalence of diabetes due to gastroparesis, changes in the diagnostic criteria, treatment and severity of gastroparesis, recognition, and diagnoses of these disorders or changes in hospital practice [14].

Pathophysiology

In diabetic gastroparesis, the contractions of the antral body are ineffective, although the smooth muscle layers appear to be normal [15,16].

Changes in cellular abnormalities in patients with diabetic gastroparesis include abnormalities in extrinsic innervation in the stomach, loss of neurotransmitters at the level of the enteric nervous system, mild muscle abnormalities, loss of interstitial cells of Cajal, and changes in the population of macrophages present in the muscular wall [17].

Chronic gastric motility abnormality is characterized by symptoms suggestive of obstruction mechanisms and delayed gastric emptying in the absence of obstruction mechanisms [6-18].

The associated symptoms that may occur in some patients are early satiety, prolonged postprandial fullness, swelling, nausea, and vomiting, abdominal pain [17], and weight loss [19].

Only 20% undergo these symptoms [20-22]. Physical examination is normal in most cases, in type II diabetes obesity is a risk factor for diabetic gastroparesis [23].

Diagnosis

Usually, it has been done using the breath test [24]. Scintillography for gastric emptying is considered the gold standard for diagnosis and quantification of gastric emptying delay [25]. Another supportive study that is useful for diabetic gastroparesis is through ultrasonographic methods [3].

Differential Diagnosis

The symptoms that occur in diabetic gastroparesis are like those presented with benign or malignant gastric obstruction and other organic causes such as cholecystitis [26,27]. DM has been related to pancreatic cancer [28], chronic intestinal obstruction resulting from alterations in the small intestine [29,30].

Vomiting from gastroparesis occurs later in the postprandial period and may occur for two reasons. First diabetic gastroparesis may predispose to regurgitation, however vomiting and regurgitation may coexist, second, initial regurgitation without effort is followed by subsequent vomiting [5].

Treatment

The goal of treatment is to reduce symptoms, a correct nutrition and to improve the quality of life [5]. It includes prokinetic drugs such as first line, nausea and vomiting can be controlled with some antiemetic drug, it must also include education and support diet, behavioral changes [18,24,25,31].

Other authors differ saying that antiemetics should be first line, followed by a diet modification, such as fiber, but is infrequently sustained [5,30-32]. In an accomplished study 30% of patients with diabetic gastroparesis implementing nutritional support have a calorie deficient diet [33]. The uncontrolled glycemia increases the symptoms of gastroparesis [34]. There are few evidences that rapid emptying is associated with hypoglycemia in DM [35]. The clinical consequences of diabetic gastroparesis include the induction of gastrointestinal symptoms, changes in medication absorption and loss of drug stabilization [36].

In endoscopic therapy for gastroparesis, intrapyloric botulinum toxin injection has been used [37]. Electronic stimulation and surgical intervention can be used in some patients and represents a therapeutic option [19], such as percutaneous endoscopic gastrostomy [38], and/ or jejunostomy [39]. Idiopathic gastroparesis treatment based on botulinum toxin has been shown [37]. Two studies showed that there is no benefit with the administration of tricyclic antidepressants for the improvement of rapid emptying in idiopathic gastroparesis and functional dyspepsia [40]. In the treatment it is important to consider the emotional variables [41].

References