

# DIABETES AND GASTROINTESTINAL TRACT

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The impairment of gastrointestinal function in chronic diabetes (Katz and Spiro, 1966; Goyal and Spiro, 1971) and acute Ketoacidosis (Bearwood, 1935) may be due to autonomic neuropathy (Rundles, 1945), microangiopathy (Angervall and Save-Soderbergh, 1966), changes in insulin and glucagon release and acute metabolic disturbances (Scarpello and Sladen, 1978).

Acute manifestations like anorexia, nausea and vomiting in diabetic Ketoacidosis are attributed to gastric stasis. Gastric atony may be due to glucagon and insulin release (Aylett, 1962; Herrera et al., 1967), reversible autonomic disturbances (Scott and Lloyd-Mostyn, 1976), intracellular hypokalemia due to electrolyte changes in Ketoacidosis and sudden stress in recent onset diabetes (Howland and Drinkard, 1963).

Haematemesis in diabetics is secondary to erosive gastritis which results from increased concentration of urea in the retained gastric fluid (Devonport, 1968).

Acute abdominal pain with leucocytosis and raised amylase (Beardwood, 1935; Alberti and Hockaday, 1977; Knight et al., 1973) also occurs in these patients. Persistent abdominal pain may also occur as a result of diabetic radiculopathy (Longstreth and Neweomer, 1977).

Autonomic neuropathy affects the oesophagus and manifests as dysphagia, diffuse oesophageal ulceration and diminished or absent oesophageal peristalsis (Vinson and Wilder, 1933; Mandelstam and Leiber, 1967).

Atrophic gastritis is more frequent in diabetics than in the age matched controls (Katz and Spiro, 1966; Angervall et al., 1962). It results in diminished gastric secretion (Angervall et al., 1961) and an increased incidence of pernicious anaemia (Arapakis et al., 1963). Gastric parietal cell antibodies have also been frequently encountered in Diabetics (Moore and Neilson, 1963). Incidence of duodenal ulcer appears to be low in patients with diabetes (Wood, 1947; Dotevall, 1959).

Visceral neuropathy affecting the small bowel results in diarrhoea of diabetes and Steatorrhoea (Bergen et al., 1936; Wruble and Kaiser, 1964) without any histological changes in small intestinal mucosa. It appears that autonomic neuropathy diminishes the small bowel transit resulting in the development of bacterial overgrowth and thus deconjugation of bileacids and diarrhoea. Diarrhoea in these patients responds to antibiotics (Gren et al., 1968). Diabetics in whom bile acid deconjugation does not occur are treated with cholinergic drug (Vinnik et al., 1962) and Sympathomimetic agents (Malins and French, 1957) with variable results.

Severe constipation (Rundles 1945; Mayne, 1965) and colonic dilatation with faecal impaction (Paley, 1961) may be due to colonic neuropathy.

Gastrointestinal manifestations of diabetes may be due to visceral neuropathy or transient hormonal and metabolic disturbances (Scarpello and Sladen, 1978).

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