# Gastric outlet abstraction

**Causes:**

***Two*** common causes of gastric outlet obstruction

1) Gastric CA
2) Pyloric stenosis secondary to peptic ulcer disease. Here the stenosis in peptic ulcer disease is seldom in the pylorus, but it is in the first part of the duodenum, so it is a misnomer term.

Now a days, gastric CA is the commonest & gastric outlet obstruction should be considered malignant until prove otherwise.

**Clinical features:**

In benign gastric outlet obstruction, there is usually long history of peptic ulcer disease.

The patient usually complains from repeated vomiting.

The vomiting is characteristically un pleasant in nature & is totally lacking in bile & it is often possible to recognize food stuff taken several days ago.

The patient may have pain which may be unremitting or largely disappear.

The patient commonly complains from weight loss, feeling unwell & dehydration.

**Examination**

It reveals distended stomach & succussion splash.

**Metabolic effects:**

Vomiting of hydrochloric acid lead to hypochloraemic alkalosis. Initially the sodium & potassium levels may be relatively normal. But as dehydration progress, more profound metabolic abnormality arises, partly related to renal dysfunction.

Initially, the urine has low chloride & high bicarbonate content reflecting the primary metabolic abnormality. The sodium is excreted along with bicarbonate ions causing hyponatremia & more profound dehydration.

Then, because of dehydration a phase of sodium retention follow & potassium & hydrogen ions excreted in preference to sodium & the urine become paradoxically acidic & hypokalemia ensues.

Alkalosis decrease ionized calcium causing tetanus.

**Management**

This involve correcting the metabolic abnormality & dealing with the mechanical problem. The patient should be rehydrated with intravenous isotonic saline with potassium supplement.

Replacing the sodium chloride & water allows the kidneys to correct the acid base abnormality.

The metabolic abnormality is less if the obstruction is due to malignancy as the acid base disturbance is less pronounced because of the hypochloremia associated with gastric CA.

The stomach should be emptied by wide bore nasogastric tube & some times by orogastric tube. This allows investigating the patient with endoscopy or contrast radiology.

Biopsy of the area around the pylorus is essential to exclude malignancy.

The patient should also be given gastric antisecretary agents e.g. ranitidine given initially intravenously.

Early cases of obstruction, may settle with conservative treatment as edema around the ulcer diminish as the ulcer heal.

Severe cases treated surgically usually with gastroentrostomy rather than pyloroplasty. The addition of vagotomy may be appropriate.

Endoscopic treatment with balloon dilatation may be most useful in early cases, but this has the following problems:

1) Dilating the duodenal stenosis may lead to perforation.
2) Dilatation may have to be performed several times & some times may not be successful in the long term.

*mugdad fuad*