DISCUSS THE BIOCHEMICAL CHANGES THAT OCCURS IN GASTRIC OUTLET OBSTRUCTION

The biochemical changes varies with time, however in a patient with gastric outlet obstruction accompanied by protracted non-bilious vomiting (persistently loosing gastric acid) or prolonged gastric suctioning. The final picture of the biochemical changes is as listed below:

- Hypochloremia
- Hyponatremia
- Hypokalemia

Metabolic alkalosis and formation/excretion of acidic urine (urine with low PH, PH less than 5.5), due to loss of hydrogen ions in the urine

In a patient that has metabolic alkalosis, the loss of acid in urine is not consistent with the expected response which is the excretion of alkaline urine while conserving hydrogen ions in order to reverse the metabolic alkalosis. Therefore, the renal excretion of acidic urine is paradoxical; hence phenomenon is termed **PARADOXICAL ACIDURIA**.

The pathophysiology of the biochemical changes

Following persistent vomiting of gastric juice which is rich in water, HCL (about 150mmol/L). The initial derangement is dehydration, hypochloremia and metabolic alkalosis. Hyponatremia soon follows because gastric secretion contains about 60-100mmol/L, while the development of hypokalemia is delayed because of the low concentration of potassium in the gastric juice(5-10mmol/L) and because there is repletion of lost serum potassium by egress of potassium from the intracellular reservoir (intracellular potassium is about 140mmol/L)

The normal renal in the presence of dehydration and hyponatremia is to conserve water and sodium and produce scanty urine (via the activation of the renin –angiotensin-aldosterone system): In the renal tubules sodium is reabsorbed and a positively charged ion (preferably potassium) is excreted. If the vomiting is protracted the loss of potassium from two fronts simultaneously (in gastric juice and in urine) leads to depletion of extracellular potassium. The extracellular potassium is replenished initially by egress of potassium from the intracellular space into the extracellular fluid until the intracellular potassium is also depleted. Upon depletion of total body potassium, another positively charge ion will have to be exchange (excreted in the urine) in the renal tubules for the reabsorption of sodium and water. The next available cation that will replace potassium in this system happens to be hydrogen ion, this forced excretion of hydrogen ion in urine in exchange for sodium and water reabsorption leads to production of acidic urine in the presence of metabolic alkalosis – “paradoxical aciduria”

Note that in addition to the loss of the serum acidity through secretion of hydrochloric acid in the gastric juice, for every millimole of chloride secreted into the gastric juice an equivalent amount of bicarbonate is reabsorbed into circulation in the stomach. Also, in the renal tubules, sodium is reabsorbed as sodium bicarbonate. These two sources of increasing serum bicarbonate lead to worsening of the metabolic alkalosis. The metabolic alkalosis leads to functional hypocalcaemia because of reduced concentration of ionized calcium which is the functional/active form of serum calcium.