8.3: Acidosis and Renal Failure

8.3.1: Mechanisms

Metabolic acidosis occurs with both acute and chronic renal failure and with other types of renal damage. The anion gap may be normal or may be elevated.

A generalization that can be made is:

• If the renal damage affects both glomeruli and tubules, the acidosis is a high-anion gap acidosis. It is due to failure of adequate excretion of various acid anions due to the greatly reduced number of functioning nephrons.

• If the renal damage predominantly affects the tubules with minimal glomerular damage, a different type of acidosis may occur. This is called Renal Tubular Acidosis (RTA) and this is a normal anion gap or hyperchloraemic type of acidosis. The GFR may be normal or only minimally affected.

8.3.2: Uraemic Acidosis

The acidosis occurring in uraemic patients is due to failure of excretion of acid anions (particularly phosphate and sulphate) because of the decreased number of nephrons. There is a major decrease in the number of tubule cells which can produce ammonia and this contributes to uraemic acidosis.

Serious acidosis does not occur until the GFR has decreased to about 20 mls/min. This corresponds to a creatinine level of about 0.30-0.35 mmols/l.
The plasma bicarbonate in renal failure with acidosis is typically between 12 & 20 mmols/l. Intracellular buffering and bone buffering are important in limiting the fall in bicarbonate. This bone buffering will cause loss of bone mineral (osteomalacia).

Most other forms of metabolic acidosis are of relatively short duration as the patient is either treated with resolution of the disorder or the patient dies. Uraemic acidosis is a major exception as these patients survive with significant acidosis for many years. This long duration is the reason why loss of bone mineral (and bone buffering) is significant in uraemic acidosis but is not a feature of other causes of metabolic acidosis.

8.3.3: Acidosis due to Acute Renal Failure

Retention of metabolic acids occurs with acute renal failure.

The clinical details in these patients are often complex and the actual severity of acidosis is variable. Some other complicating factors are catabolism (increased metabolic acid production), vomiting, diarrhoea, lactic acidosis due to poor perfusion, bicarbonate therapy and dialysis.

Hyperkalaemia is often present and is often the factor determining the need for acute dialysis.

References