Chronic renal failure and advances in the management of renal anaemia

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Common causes of chronic renal failure (CRF) include chronic glomerulonephritis, diabetic nephropathy, hypertension, renovascular disease, interstitial nephritis, hereditary renal disease, and longstanding urinary tract obstruction (Bradley 1997). A number of complications can occur in patients with CRF, causing a variety of symptoms and these include bone disease, hypertension and anaemia. The main aims of managing CRF are to treat symptoms, slow the decline in renal function and prevent further complications.

Bone Disease

Renal osteodystrophy is the bone disease that develops in almost all patients with severe chronic renal failure. This disorder often presents with hyperphosphataemia, hypocalcaemia, high levels of parathyroid hormone (PTH) and low levels of calcitriol (the major active metabolite of vitamin D). Treatment usually involves control of calcium, phosphate and PTH levels by diet, phosphate binders and vitamin D supplements. Studies have shown poor adherence with such regimes and therefore pharmacists have an important role to play in helping to counsel such patients.

Hypertension

Hypertension occurs in about 80% of patients with CRF and is treated with any combination of the following agents: beta-blockers, diuretics vaso-dilators, angiotensin converting enzyme inhibitors (ACEI), calcium antagonists and, more recently, angiotensin II blockers. The latter three groups of drugs have a potentially nephroprotective effect.

Anaemia

Anaemia is one of the major problems affecting patients with renal failure, particularly when they reach end-stage and require regular dialysis. Anaemia arises from a variety of reasons which include chronic blood loss, recurrent infections, aluminium toxicity, reduced red cell survival, iron deficiency and most importantly, lack of production of erythropoietin (EPO) (Macdougall 1995).

Treatment of anaemia using EPO is expensive, costing anywhere from $\pounds 2,000$ to above $\pounds 10,000$ per patient per year of treatment. The use of intravenous iron to help improve response to EPO is well recognised, but this too can incur a further cost pressure.

Pharmacists need to be involved in ensuring that expensive drugs such as EPO are used optimally by setting regular reviews of patients receiving the drug and monitoring the factors that affect the response to EPO.

More generally, pharmacists need to keep their patients well educated about their medication from the pre-dialysis stage to post transplantation. Simplification of treatments is vital to try and aid concordance with complex regimens. It is essential to ensure that the most suitable drugs are used and in the correct dosages in renal patients. The extension of the pharmacist's role into outpatient medication review clinics will help facilitate monitoring of medication in this group of patients.

Bradley, J. R. (1997). Renal disorders. The Hospital Pharmacist. 4: 137–139

Macdougall, I. C. (1995). How to get the best out of r-HuEPO. Nephrol. Dial. Transplant. 10 (Suppl 2): 85–91