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IV.9.2 Leukopenia

Guideline

A. Because leukopenia is relatively common after kidney transplantation, regular screening and careful evaluation of its causes are recommended. Azathioprine and mycophenolate mofetil may lead to leukopenia. The combination of allopurinol and azathioprine should be avoided. Leukopenia is often associated with viral infections. (Evidence level B)

Commentary on Guideline IV.9.2: Leukopenia

Guideline A. Leukopenia in long-term renal transplant patients is usually caused by one of two factors, namely toxic effects of drugs and infection.

Azathioprine and MMF may cause leukopenia. The risk of leukopenia is related to the dose. Some patients (slow methylators) with a genetic defect of thiopurine methyltransferase may develop granulocytopenia even with low doses of azathioprine [1]. The use of allopurinol in patients given azathioprine should be avoided or the dose of azathioprine should be reduced to one-third or less, as allopurinol inhibits the enzyme xanthine-oxidase, which inactivates metabolites of azathioprine. Other drugs that may induce leukopenia in transplant patients are ganciclovir, TMP-SMX and α -methyl dopa.

Moreover, a number of infections, particularly viral infections (e.g. CMV) and overwhelming bacterial infections, may be associated with granulocytopenia.

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IV.9.3 Erythrocytosis

Guideline

A. In the case of erythrocytosis, the first-line treatment should be administration of ACE inhibitors or angiotensin II receptor antagonists. (Evidence level B)

Commentary on Guideline IV.9.3: Erythrocytosis

Guideline A. Erythrocytosis, or polycythaemia, is defined as a haematocrit > 52% in men and > 49% in women. The incidence has been reported to vary between 8 [1] and 22% [2]. In ~30–40% of cases, polycythaemia may resolve spontaneously. Long duration of dialysis, acquired cystic disease, polycystic kidney disease, graft artery stenosis, graft hydronephrosis, diabetes mellitus, smoking and arterial hypertension may contribute to the development of post-transplant erythrocytosis [3] by overproduction of either erythropoietin [4] or IGF-1, which is an important regulator of erythropoiesis [5].

Patients with polycythaemia vera may suffer from arterial venous occlusive events associated with thrombocytosis, platelet dysfunction and hypervolaemia. Although one study in 53 polycythaemic transplant patients failed to find an increased incidence of thromboembolic events [6], it is likely that erythrocytosis may increase the risk of cardiovascular