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Multiple infections after commercial renal transplantation in India

Sir,

The increased demand for transplantable kidneys has not met with a corresponding increase in the supply of these organs. Many patients travel to other, mostly developing countries, in search of commercial transplantation. In order to perform the procedure rapidly, standards of transplantation are compromised [1]. Besides the clinical issues, ethical problems are also of equal concern.

We report the case of a 56-year-old Slovenian male who underwent renal transplantation for undiagnosed chronic renal failure. He refused a suggested haemodialysis and await for transplantation. With no consultation with a nephrologist, he privately arranged the transplantation in India. Live-donor renal transplantation was performed in September 2004, in a New Delhi private clinic. The donor was a 28-year-old male from Bangladesh. The post-operative course was uneventful, and the patient was discharged from the hospital on the day 10. Tacrolimus and methylprednisolone were used for immunosuppression. The patient immediately returned to Slovenia and consulted his nephrologist. His initial renal function and laboratory parameters were within normal ranges.

Three weeks after the transplantation he became febrile; ESBL-producing *Escherichia coli* was isolated from blood and urine cultures. Despite treatment with imipenem he remained febrile. *Aspergillus terreus* was isolated from a partially dehiscent post-operative wound, followed by positive serum galactomannan assay. Treatment with voriconasol was initiated. On the day 40, deep venous thrombosis of the right iliofemoral vein developed (the allograft vein was anastomosed to the right external iliac vein). A few days later, *Plasmodium falciparum* and *Plasmodium vivax* were found in the peripheral blood smear (Figure 1A). He was treated with intravenous quinine; parasitaemia (initially 4.8%) cleared in 6 days and his condition temporarily improved.

On the day 53, symptoms and signs of infection reappeared and renal function began to deteriorate. On the basis of a computed tomography scan and sequential renal scintigraphy, a urine leak from the lower renal pole was suspected; the allograft was removed and immunosuppression stopped, the patient was placed in the intensive care unit. The clinical suspicion was confirmed, as the lower pole of the kidney was found to be necrotic (Figure 1B). From the necrotic kidney tissue, ESBL-producing *E. coli*, *Mucor* spp. (Figure 1C) and *Mycobacterium fortuitum* were isolated. Furthermore, strongly birefringent crystalline vascular deposits typical of tule in the wall of small interlobular artery accompanied by segmental fibroproliferative granulomatous vasculitis with elastica destruction (van Gieson-Weigert staining, polarization microscopy).

![Fig. 1.](image-url)
deposits, most probably talc, accompanied by focal proliferative endarteritis were found in the kidney tissue, suggesting intravenous drug addiction in the kidney donor (Figure 1D). Follow-up quantitative Polymerase chain reaction (PCR) for cytomegalovirus (CMV) revealed 65,000 copies/ml. Liposomal amphotericin B, therapy for non-tuberculous mycobacteriosis and ganciclovir were added to the therapeutic regime. The patient’s condition did not improve and despite all efforts, he died 102 days after the transplantation.

This case demonstrates an unfortunate combination of bacterial, fungal and parasitic infections (CMV was reactivation) following kidney transplantation from a living unrelated donor in India. To the authors’ knowledge, 43 cases of post-transplant malaria have been reported [2–4]. During his stay in India, our patient remained in New Delhi and was therefore not exposed to malaria-bearing mosquitoes, so the most likely source of malaria was the allograft. It is highly likely that deep mycosis (A. tereus, Mucor spp.) and M. fortuitum were transmitted by the renal allograft as well. Renal mucormycosis, sharing similarities with our case, has previously been reported in a patient actively using intravenous drugs [5].

Over half of all the renal transplant recipients in tropical countries develop a serious infection at some point in the post-transplant period and 20–40% of them succumb to these infections [1,6,7]. A multitude of factors (unhygienic conditions, hot and humid climate, scanty diagnostic techniques, etc.) contribute to this dismal outcome. In commercial transplantations, the primary objective of the medical team is often profit, and not necessarily the well-being of either donors or recipients.

Conflict of interest statement. None declared.

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Vitamin E and selenium co-supplementation attenuates oxidative stress in haemodialysis patients receiving intra-dialysis iron infusion

Sir,

Parenteral iron administration is a common practice in the era of advanced haemodialysis (HD). A major concern is that the oversaturation of transferrin and subsequent propagation of redox-active iron occurs with the recommended doses of parenteral iron (1–4 mg/kg) [1]. Redox-active iron is a potent pro-oxidant that triggers free-radical chain reaction by the formation of hydroxyl radicals (Fenton reaction) [1–3]. Intravenous iron also increases neutrophil respiratory burst and thus the generation of oxygen radicals [4]. Lipid peroxidation, protein oxidation and DNA damage are the main consequences of this oxidative stress (Oxs) [2]. It has been suggested that high cumulative doses of iron may contribute to increased morbidity and mortality among end-stage renal disease (ESRD) patients, through increasing OxS which favours atherosclerosis and is an independent risk factor for cardiovascular mortality [5,6]. Vitamin E and selenium are effective body antioxidants that prevent free-radical formation and halt the damaging free-radical chain reaction once it begins [7–10]. The aim of this study was to assess the efficacy of vitamin E and selenium supplementation on reducing the OxS in HD patients receiving intradialysis iron infusion.

Nineteen ESRD patients (mean age, 43 ± 12 years; 11 male and 8 female) on chronic HD were enrolled to this prospective and interventional study. All patients had been undergoing HD twice weekly, while receiving an iron infusion (100 mg iron/5 ml as ferric hydroxide sucrose complex in 10 min via the venous line of the dialysis circuit) 10 min after the beginning of a HD session. Supplements were prepared as capsules, each containing 400 IU vitamin E and 600 μg sodium selenide. All patients received one dose of the aforementioned supplement, 6 h in advance of a scheduled HD session. The same patients were used as the control if they had not consumed the supplement before the HD session. The study was approved by the local research council and ethics committee and informed consent was obtained from patients ahead of the study.

The venous blood samples were drawn immediately before (~10 min after the beginning of HD) and 45–50 min after iron infusion; they were then separated to serum and stored in a refrigerator until biochemical analysis. The serum concentration of malondialdehyde (MDA), an intermediate product of lipid peroxidation [11] was used as a marker of OxS. Briefly, MDA was reacted with thiobarbituric acid by incubating for 1 h at 95–100°C. Fluorescence intensity was then measured in the n-butanol phase using a fluorescence spectrophotometry with excitation and emission at 525 and 547 nm, respectively. The statistical analyses

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