As I started talking to patients, I realized how important each complaint was, however, trivial they may seem. I understood nausea, anorexia, fatigue – all these terms. I also realized my folly in ignoring these in many . Now, when I inspect the vascular access, the counter punctures, the minor hematomas, etc., I feel the pain. I realize that this is their life-line; it certainly deserves tender loving care. While interacting with the dialysis patients, I started realizing the subtle signs of depression. Regarding the non-compliance to the dialysis schedule and the medications/irregular follow-up, I realize that they are related to the emotional disturbances, which we very often overlook. I could see the enthusiasm with which my dialysis medical officer would correct the ultrafiltration rate, calculate the spKt/v, to achieve a “good” dialysis. Little did he analyze about the quality of life (QoL). The medication prescription written very often resembles a mini pharmacopeia. How often have we analyzed their sleep disturbances, social support, and QoL of the care givers? I feel many of us could do better.

As I look back on the day of my surgery, how well the nurses tried to pep me up, as I was wheeled into the theatre, how well they received me in the surgical intensive care unit (ICU) and took care of all the lines with utmost care and concern. Each member of the team excelled in bestowing that little extra tenderness. Our social worker walked in for a long gossip session. When I enquired how she felt working in Oncology, she came out with a very relevant observation and said “Doctor, the ‘cure rates’ in several malignancies are very good, thanks to the excellent chemo agents and innovations in radiation therapy, so targeted with minimal side effects, that I find it any day better than many other chronic ailments.” How true.

‘Cure rate’ is a phrase unheard of in several departments – including Nephrology. A very buoyant statement from our social worker!!

Friends and well-wishers continued to drop in to give pep talks. Many of them spoke at length about how Lance Armstrong could bounce back after advanced malignancy, to win the Tour de France – a race famed for its grueling intensity, how Steve Jobs could achieve the iPad/iPhone revolution despite his terminal malignancy. I recollected my days in the bed with the continuous chemotherapy pump that was set for 48 h. I would eagerly look for the oncologist to drop in and reassure me and my family members that all is well. It also made me ponder – how often have I sat down beside the patient and heard him or sat with the family members to tell them about immunosupression.

The practicing nephrologist needs to be an all-rounder – physician, well-wisher, psychotherapist, and a good soft-spoken gentleman. Let’s recollect our undergraduate teaching that health is defined as not only the absence of disease and infirmity but also the presence of physical, mental, and social well-being. I reckon that our post-graduate training empowers us with lot of skill in tackling disease, life teaches us the rest – how to deal with people.

Life is more accurately measured by the lives that we have touched than by the materials we have acquired.

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Post-renal transplant Kaposi’s sarcoma of skin

Sir,

A 35-year-old male underwent live-related renal transplantation in December 2005, his mother was a donor. His pre-transplant serology was negative for hepatitis B surface antigen and for anti-hepatitis C virus as well as anti-human immunodeficiency virus antibody. His plasma IgG for cytomegalovirus (CMV) was positive and IgM was negative indicating previous infections by CMV. Human Herpes virus-8 (HHV-8) testing was not performed in the donor or the recipient. Transplantation was without any
complication. He was on triple immunosuppressant and maintaining baseline serum creatinine of 1.5 mg/dL. In August 2010, he was admitted with graft dysfunction and on renal biopsy was found to have tacrolimus toxicity with chronic allograft nephropathy. His tacrolimus dose was reduced to 3 mg/day and he was discharged with serum creatinine of 2.5 mg/dL.

In March 2011, he presented with well-defined elevated violaceous nodular lesions on middle and ring finger of left hand for 2 months, there were two nodules on both fingers [Figure 1]. There were no other similar lesions anywhere on the body. There was no history of hemoptysis or melena. This time his serum creatinine was 4.5 mg/dl and was on tacrolimus 3.0 mg/day; mycophenolate (MMF) 1.5 g/day; and prednisolone 10 mg/day. Skin biopsy showed proliferation of small- to medium-sized vessels lined by endothelial cells suggestive of Kaposi’s sarcoma (KS) [Figure 2]. His chest X-ray and computed tomography scan of thorax and abdomen were normal. HHV-8 testing was not carried out. The immunosuppressive therapy was tapered to prednisolone 10 mg daily, tacrolimus and MMF were totally withdrawn. Radiation therapy was delivered by linear accelerators in dose of 3 Gy per fraction for a total of 10 fractions. He responded well to the treatment and all his lesions regressed. At present he is on maintenance hemodialysis and is enrolled in deceased donor transplant waiting list.

KS is a soft tissue cancer arising from endothelial cells of blood vessels involving; skin, lymph node, gastrointestinal tract, and lungs; characterized histologically by endothelial-lined vascular spaces and spindle-shaped cells.[1] Rare in general population, risk of its development is substantially increased in immuno-compromised persons like patients with acquired immune deficiency syndrome, solid organ transplant recipients, and in patients on chemotherapy for lymphoma.[2] Genetic predisposition and ethnic difference in incidence has been reported. It is endemic in African countries, has higher prevalence in people of Arabian countries and other Mediterranean descent, but it is rare in Indian ethnicity.[2] Infection with HHV-8 is very commonly associated with patients of KS, antibody against this virus are found in 70-90% of immune-compromised patients and in about 100% of non-immunocompromised patients having KS.[3] The incidence of KS in transplant recipients correlates with prevalence of HHV-8 in general population.[4] A small Indian study screened 108 samples of sera from blood bank for HHV-8 antibody and reported prevalence of 4%. In healthy population, prevalence of HHV-8 is 11% in USA, 12% in Thailand, and 15% in Malaysia.[4] First case of post-renal transplant KS in India was reported in 1998 from Kerala.[5] There is scarcity of data and rarity of occurrence of post-transplant KS in the Indian continent. Onset of KS usually occurs within first 2 years of transplantation. Here, we report a case of KS of skin in a renal transplant recipient occurring after 5 years of transplantation and was successfully treated with withdrawal of immunosuppressant and local radiotherapy.

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Sir,

A 21-year-old male was referred to our hospital for evaluation and management of renal failure. He presented with oliguria, dark color of urine, pedal edema, and muscular pain since 2 days. He had a history of ingestion of insecticidal liquid containing gamma benzene hexachloride (BHC) with a suicidal intention 3 days back. He had received gastric lavage and supportive treatment in the form of fluids at a primary care center.

At the time of admission his blood pressure was 120/80 mmHg, pulse rate 90/min, respiratory rate was 24/min, and urine showed dipstick-positive proteinuria (3+) with myoglobinuria. He had tenderness of muscles (more in lower limbs) with normal tendon reflexes and altered mentation. Hematological parameters (total leukocytes 10,500/mm$^3$, platelets 1.9 lac/mm$^3$, hemoglobin 11.7 g/dL) and liver function tests were normal except for a mild elevation in aspartate aminotransferase (AST 94 U/L) without any evidence of hemolysis. The presence of metabolic acidosis (pH 7.18, HCO$_3^-$ 9) and rise in creatinine from 0.9 to 3.1 mg/dL prompted us to initiate him on hemodialysis. Additional evaluation revealed total serum creatine phosphokinase (CPK) of 2890 U/L (range: 20-230 U/L), lactate dehydrogenase of 360 U/L, and a rise in potassium to 6.3 mEq/L. Immunological tests like complement levels (C3), anti-nuclear antibodies, and anti-neutrophilic antibodies were unremarkable. A clinical diagnosis of BHC-induced rhabdomyolysis leading to acute kidney injury (AKI) was made. Though myoglobinuria ceased over the next 3 days and total CPK decreased to 146 U/L by the end of 1 week, he required hemodialysis on alternate days for 1 week. After 2 weeks of hospitalization and supportive care, there was a gradual improvement in urine output, and a declining trend was noticed in creatinine. Similarly, his muscular tenderness gradually improved with the disappearance of uremic symptoms and creatinine decreased to 1.2 mg/dL on the 21st day.

Poisoning caused by industrial chemicals is an important cause of community-acquired AKI in Asia. Our case demonstrates AKI associated with ingestion of BHC which is very unusual. BHC is an organochlorine insecticide and acute poisoning usually leads to features of neurotoxicity like altered mentation and seizures. Other features like liver dysfunction, metabolic acidosis, and hematological and gastrointestinal toxicities have occasionally been described. BHC is known to be nephrotoxic as per reports of experimental animal models, but reports of renal involvement in humans with myoglobinuria have not been convincingly reported in the literature. Development of muscle necrosis and AKI have been reported only in one case in 1977.

Rhabdomyolysis is a life-threatening condition resulting from the breakdown of skeletal muscles and commonly occurs with trauma, burns, viral infections, polymyositis, convulsions, exertion, ischemia, alcohol abuse, and various toxins. Other causes of myoglobinuria were safely excluded in our patient as per clinical history and temporal ingestion of BHC leading to acute presentation suggesting its etiological role. The role of dehydration and sepsis leading to AKI was ruled out in this patient.

References

1. Tan HH, Goh CL. Viral infections affecting the skin in organ transplant recipients: Epidemiology and current management strategies. Am J Clin Dermatol 2006;7:13-29.
2. Jalilvand S, Shoja Z, Mokhtari-Azad T, Nategh R, Gharehbaghian A. Seroprevalence of Human herpesvirus 8 (HHV-8) and incidence of Kaposi's sarcoma in Iran. Infect Agent Cancer 2011;6:5.
3. Antman K, Chang Y. Kaposi's sarcoma. N Engl J Med 2000;342:1027‑38.
4. Ablashi DV, Chatlynne LG, Lapps W, Manassaram D, Cooper HR, Masood R, et al. Human herpesvirus-8 (Hhv-8) comparative sero-prevalence in U.S.A., India, Thailand, Malaysia, and its association with malignancies. J Acquir Immune Defic Syndr Hum Retrovirol. 1998;17:p A25.
5. Ajithkumar K, George S, Jacob M, Pulimood S, Chandi SM, Thomas PP, et al. Transplant associated Kaposi's sarcoma. Indian J Cancer 1998;35:171-2.