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KIDNEY TRANSPLANT RECIPIENTS WHO UNDERWENT PARATHYROIDECTOMY FOR TERTIARY HYPERPARATHYROIDISM

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After kidney transplant, tertiary hyperparathyroidism (ThPT) can cause hypercalcemia and worsen bone disease. While calcimimetic drugs can be potentially useful, their use after transplant is not approved or covered. We present our prevalence data, indications for parathyroidectomy (PTx), and accuracy of sestamibi scan in predicting parathyroid pathology.

In this retrospective study, we identified transplant recipients who underwent kidney transplantation between 2008 and 2013 and who underwent PTx for ThPT at Houston Methodist Hospital. In addition to demographic and clinical information, data collected included serum calcium, phosphorus, Vitamin D and intact PTH (iPTH) levels obtained pre transplant, and at months 1, 3, 6 and 12 post-transplant and also bone density measurement and parathyroid scan.

We identified 26 (2.5%) patients who underwent PTx, among the 1001 patients who underwent kidney transplant at our center, during the 5-year study period. Mean age was 52±12 yrs and 58% were women. The majority (88%) underwent deceased donor transplantation and all were on Tacrolimus, Mycophenolate and Prednisone. Prior to surgery, mean calcium and iPTH levels were 10.4±1.3 mg/dl and 508±404 pg/ml, respectively. Median time duration between transplantation and PTx was 482 days (Q1, Q3: 225,909). Indications for PTx included persistent hypercalcemia (42%), osteoporosis/worsening osteopenia (35%) or both (15%). Few patients (8%) underwent surgery after failing medical therapy, including calcimimetics. Sestamibi scan, obtained in all patients, showed glandular hyperplasia in 50% and adenoma in 34%. However, surgical pathology showed hypercellular parathyroid tissue in 25 specimens (96%). Only one biopsy showed parathyroid adenoma with thick, fibrous trabecula and calcifications.

In kidney transplant recipients, ThPT is common and in small proportion of cases, it warrants PTx for hypercalcemia and worsening bone disease. Parathyroid hyperplasia is the commonest pathology. Sestamibi scan is inaccurate in making the distinction between hyperplasia and adenoma.

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MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS SECONDARY TO LIGHT CHAIN DEPOSITION DISEASE: A RARE CASE OF MONOCLONAL GAMMOPATHY OF RENAL SIGNIFICANCE.

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Light chain deposition disease (LCDD) is a rare entity characterized by tissue deposition of kappa or lambda light chains. It is a part of the new recognized kidney-related disease spectrum, monoclonal gammopathy of renal significance (MGRS). We report a case with nephrotic syndrome and hypertension from LCDD without overt lymphoproliferative disorders.

A 92 year-old female with a history of low grade B cell non-Hodgkin lymphoma in complete remission was admitted due to worsening bilateral lower extremity edema and hypertensive urgency. Urine analysis showed nephrito-nephrotic profile with proteinuria of 20 gm/day. She was aggressively treated with diuretics. Extensive workup including hepatitis B, hepatitis C, HIV, ANA and cryoglobulin were all negative with mildly low C3 and normal C4. SPEP with immunofixation detected IgM lambda monoclonality. UPEP with immunofixation and serum free light chain assay were negative. Kidney biopsy showed MPGN under light microscopy. Immunofluorescence (IF) study showed abundant lambda light chain deposits. Electron microscopy (EM) showed subendothelial electron-dense deposits. Bone marrow biopsy was negative for active malignancy. CT chest abdomen and pelvis showed slightly increased intra-abdominal lymphadenopathy. The patient was treated with rituximab for a 4 –week course with improvement of proteinuria. The patient was followed up by oncology and nephrology for 6 months to date with no evidence of recurrent lymphoproliferative malignancy.

It is important to recognize LCDD as a part of elaborating MGRS. Evaluation must include SPEP and UPEP with immunofixation, serum free light chain assay, bone marrow and imaging studies. A kidney biopsy with detailed IF and EM studies to identify deposition composition and pattern of organization is necessary. Currently, there is no standard established treatment protocol. Early diagnosis with preserved renal function can facilitate treatment management with better long-term renal outcome.

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COMPARISON OF THE SAFETY OF FOUR DIFFERENT INTRAVENOUS IRON FORMULATIONS IN CKD PATIENTS WITH IRON DEFICIENCY ANEMIA (IDA).

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There are many intravenous (IV) iron compounds available today, but few studies have compared them directly. It has been claimed that the use of larger doses leads to a higher rate of adverse events (AEs).

This is a retrospective chart review of patients receiving various IV iron agents at our institution. Patients with CKD and IDA were included. K-DOQI criteria and prevailing clinical practice guided the need and usage of IV iron in these patients. We collected data on patient demographics, comorbidities, baseline renal and hematological parameters, and adverse events noted with drug administration. Iron sucrose (IS), ferric gluconate (FG) and ferumoxytol (Fm) were given in doses used routinely in clinical practice; whereas low molecular weight iron dextran (ID) at our center was given as a total dose infusion (TDI) of 1000 mg. AE rates were compared by Chi-Square test, using SPSS.

892 doses of IV iron were administered to patients, 28% of whom were white, 69% AA, 70% male; age 61±13 years. Baseline values: serum Creat 3.9±3.0 mg/dL, eGFR 26±16 mL/min, Hgb 9.9±1.5 g/dL, iron 43±22 mcg/dL, T-sat 15±6%. Only minor AEs were noted in 2 out of 215 administered doses with ID (0.93%), and 1 of 329 doses with Fm (0.3%). No AE was noted with either IS or FG. A sub-analysis comparing Fm to ID revealed no difference in their AE rates ($P > 0.33$).

	IS	FG	Fm	ID
Doses (n)	78	270	329	215
Adverse reactions	0	0	1	2
Anaphylactic reactions	0	0	0	0

We did not find a statistically significant difference in the AE rates of the above four IV iron formulations ($P > 0.6$). All of the AE's noted were minor, and none were life-threatening. Of note, the agents given in larger doses (Fm and ID) were as safe as those given in smaller doses (IS and FG). Moreover, there was no difference in the AE rates of Fm and ID.

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TUNNELED DIALYSIS CATHETER REMOVAL IS A SKILL THAT CAN BE TAUGHT TO ALL NEPHROLOGY FELLOWS IN TRAINING.

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Removal of tunneled dialysis catheters (TDC) is usually done in the Intervention suite or at the bedside by interventional nephrologists or radiologists. Scarce evidence exists in the literature to assess the safety, complication rate and success of this procedure when performed by trainees under supervision at academic centers, prompting this study.

This is a retrospective chart review of all TDC removals performed on an outpatient basis by nephrology fellows under faculty supervision during the most recent 5-year period at our academic training center. Data were collected regarding patient demographics, basic laboratory studies, pertinent clinical information and procedure-related variables. We evaluated the success, safety and complications of each procedure.

We identified 72 TDC removals that met the above criteria. Mean age 63±10 years. All patients were male and hypertensive; 68% were diabetic; 67% African-American. 88% of procedures were performed in ESRD patients; the rest had needed dialysis for acute kidney injury. Notably, 68% of the patients were on one or more of Aspirin, Plavix or Coumadin at the time of TDC removal. Only 2 possible complications were identified.

TDC removal on an outpatient basis by fellows in training, done under supervision, is successful (99%) and safe in the vast majority of cases. Complications are rare (rate <2%). There was no increase in risk of bleeding; even in patients on Aspirin, Plavix or Coumadin, only 1/49 patients (2%) had a minor bleeding complication. **We propose, based on the evidence in this study, that TDC removal is a skill that can and should be taught to all nephrology fellows**. This would expand the scope of practice for "regular" nephrologists, and facilitate patient care.