1. HypoMg can cause HypoK as Mg is needed to suppress K section from the ROMK channel. When Mg is low, ROMK channel is not suppressed, more K section follows.
2. HypoMg is also related with HypoCalcemia. The major factors resulting in hypocalcemia in hypomagnesemic patients are hypoparathyroidism, parathyroid hormone (PTH) resistance, and vitamin D deficiency. The effect of acute and chronic HypoMg on PTH is different (See Uptodates).
3. To differentiate Mg loss from renal or extrarenal is sometimes difficult. Calculation of fractional excretion of magnesium is useful. Less than 2: extra renal; above 3-4: renal wasting (see Uptodates)
4. RTA is diagnosed in normal renal function patients, but not in advanced CKD patients.
5. Transplant with a positive cross match can happen with SLKT but not with kidney alone transplant. Liver transplant always precedes the kidney transplant in these individuals. The donor liver usually scavenges all DSA such that the DSA generally decreases rapidly after the liver transplant. If a repeat cross match is done after the liver transplant, it is usually negative.
6. Most centers do not use Cellcept for primary immunosuppression after liver transplant. Liver transplant patients are usually very immunosuppressed. Thymoglobulin use carries a very high morbidity and even mortality risk. Hence, many centers prefer not to use Thymo for induction or acute rejection (antibody or cell-mediated) in these patients. Hence, our patient was treated with PLEX (5 times) with IVIG (3 times) for AMR.
7. Rising DSA is unusual after SLKT. Our patient had it, associated with primary non-function of the renal allograft. Hence the suspicion for AMR. Per our previous experience, most positive cross matches were associated with Class I DSA. In this patient, there were high DSA titers for class II antibodies. It is unclear whether this the reason for AMR in this patient, but no AMR in previous SLKT patients with a positive cross match at our center. It is known that the outcomes of AMR with class II antibodies are worse than that of class I antibodies.
8. Etiology of AKI after transplant - 1. Volume depletion; 2. Foley obstruction; 3. ATN because of blood loss, especially in liver patients with coagulopathy, and 4. DGF. Reasons for DGF: 1. Donor's condition, multiple and high-dose pressors need; 2. Long cold ischemic time; 3. Vascular disease in donor, 4. Long warm ischemia time because of surgical issues with vascular anastomosis - multiple vessel anastomosis. Bedside doppler can assess vascular anastomosis.
9. DSA turnaround time may be 2-3 days in some hospitals.
10. Multiple blood transfusion exacerbates antibody formation. Hemodilutional effect by transfusion needs to be taken into account when interpreting post-transplant DSA titers particularly early on in the transplant, because of the dilution effect.