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Kidney Transplantation in a Patient with CDC Positive Crossmatch, Negative Flow Cytometry Crossmatch and Donor Specific Antibody

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Case Study : The aims of this presentation are to discuss the role of different crossmatch strategies in view of an offer of deceased donor kidney to a 50-year-old female with CKD5 secondary to glomerulonephritis post pregnancy had been on haemodialysis, with h/o blood cell transfusion but no previous transplantation. The pre-transplant lab results mismatch on tissue matching with positive Complement dependent cytotoxicity crossmatch (CDCXM) for both B and T. The flow cytometry crossmatch (FCXM) was negative for both B and T and no Donor Specific Antibodies (DSA) on Luminex-SAB.
History of Present Complaints: A 50 year, female with ESRD secondary to glomerulonephritis was on maintenance hemodialysis twice a week for six months and has received PRBC twice for anaemia. She was evaluated for kidney transplantation with her prospective donor.
Interventions and Clinical Findings:
Flow cytometry crossmatching (FCXM) and Complement dependent cytotoxicity crossmatch (CDCXM) were done as part of allograft recipient assessment.
FCXM: FCXMs to detect T and B lymphocytes antibodies against CD3 and CD19 and were negative.
CDCXM: Showed presence of 25% the lymphocyte crossmatching (LCXM) using AHG-CDCXM technique (anti-human globulin). Later recipient serum was checked with dithioerythritol to exclude IgM antibodies.
Luminex-SAB technique: Luminex- single antigen based technique was negative for DSA (donor-specific antibody).
Management: In view of positive CDCXM, the patient was informed about an option of paired kidney donation transplantation, but she denied then we shifted to desensitisation protocol.
Desensitisation: Desensitisation protocol was done by plasmapheresis, followed by hemodialysis and two doses of IVIg, bortezomib and methyl prednisolone. Along with, that received mycophenolate sodium and tacrolimus.
Post desensitisation tests: Showed CDCpositive crossmatch though FCXM/DSA were negative. The presence of auto antibodies was excluded by auto crossmatch. Thus positive CDCXM could be due to Ig M antibodies which were

confirmed with IgM fix complement test.
Induction therapy was given with rabbit-anti-thymocyte globulin, and methylprednisolone intravenously for three days. Maintenance immunosuppression was given in the form of prednisolone, tacrolimus and mycophenolate sodium.
On the post operative day, two her serum creatinine was 1.5mg/dL and urine output, 8.9liters. Renal allograft biopsy performed on the fifth post-transplantation day given increasing creatinine to 1.91mg/dL and fall in urine output to 3.1 litre, which did not show any immune injury and C4d was negative by immunohistochemistry and immunofluorescence.
CDCXM and DSA were negative after two weeks of transplantation.
She was discharged in stable condition with SCr. 1.1 mg/dL and tacrolimus level of 8 ng/mL and maintained for two-month post-transplantation.
A data analysis shared by United Network of Sharing (UNOS) registry showed 55% had CDCXM positive transplants who were FCXM negative. Considering the present case scenario of positive CDCXM with negative FCXM this could be explained by a false positive CDCXM, a false negative FCXM, or by IgM as the responsible antibody.
In situation of positive CDCXM with negative FCXM as in present case scenario that could be explained by IgM antibodies which are usually not detected on standard FCXM under anti-IgG tool as IgM is not of any pathological significance in transplant science.

Keywords : renal tranplant, Donor Specific Antibody