Human renal isografts: A clinical and pathological analysis
This report documents Peter Bent Brigham Hospital experience over 13 years with 22 patients receiving renal transplants from their presumed identical twin. Seventeen of the recipients had clinical and morphological evidence of glomerulo-nephritis. At the time of this writing, eleven had developed a recurrence of a similar disease in the isotransplanted kidney. Clinical and morphologic studies of the original disease identified the characteristics of patients carrying a high risk of recurrence. Recurrent glomerulonephritis was clinically characterized by protein-uria and hematuria and often by the subsequent development of the nephrotic syndrome and progressive renal failure. Glomerular hypercellularity was the most common histologic lesion associated with recurrence. Mesangial thickening was seen with and without clinical evidence of recurrence. Glomerular deposition of immunoglobulin G., Beta lc globulin and fibrinogen was associated with recurrent disease. By electronmicroscopy both subendothelial and subepithelial deposits were seen. It is concluded by the authors that recurrent glomerulonephritis in the recipient of renal isografts is most likely a manifestation of the same underlying process responsible for the original disease which led to transplantation.
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Deposits of immunoglobulin and fibrin in human allografted kidneys
Eighty-two kidney sections from 28 human recipients of renal allografts were examined by immunofluorescence for deposits of immunoglobulin, complement and fibrin. Deposits of IgM and IgG, but not IgA were detected in vessels of all sizes; lining the endothelium, extending towards the media, and in larger vessels in the thickened intima. Linear and granular deposits were also present in glomeruli. Complement and fibrin were present but not albumin or alpb½-macroglobulin. Of 49 sections examined during clinical rejection, immunoglobulin, complement and fibrin were demonstrated in a significant number when compared with the incidence in 14 non-rejecting kidneys. The demonstration of these deposits suggests that humoral antibodies may play an important role in the rejection of human allografted kidneys.
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Plasma renin activity in aldosterone excretion, after renal homotransplantation
This study was performed to evaluate the effects of renal denervation on the renin response to sodium restriction and to the upright posture. Three patients who received renal homotransplantation 1 year or more previously, and their kidney donors were studied. Also, one recipient was investigated two months after renal transplantation. All patients were placed on a
100 mEq/day Na diet for a minimum of 3 days and then on a 10 mEq/day Na diet for 5 days. Plasma renin activity, aldosterone, and Na excretion were measured. Two recipients and one donor had plasma renin activity measured after being recumbent for 8 h and after 4 h of upright posture. All subjects responded to sodium restriction with a decrease in sodium excretion, an increase in plasma renin activity, and an increase in aldosterone excretion. The renin response to the upright posture, was similar in both recipient and the donor subjects. The data demonstrate the patients with kidneys denervated by virtue of renal transplantation can manifest increases in plasma renin activity in response to sodium deprivation and the upright posture.

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Serial studies of glomerular filtration rate and renal plasma flow in kidney transplant donors, identical twins and allo graft recipients


In normal subjects undergoing unilateral nephrectomy as transplant donors there were rapid hemodynamic changes. The glomerular filtration rate and effective renal plasma flow in the remaining kidney increased to approximately 65% of the combined function of both kidneys. The changes found appeared to be complete at the end of the first week and probably occurred within the first few days of nephrectomy.

The comparable clearances in donors and identical twins indicate that these adaptive increases in function are independent of renal innervation and that they are not limited by ischemia (within the time limits usually required for transplantation) or heterotopic transplantation of a kidney into a uremic patient. In contrast to function in the donors and those with isografts, the function of the single allo-grafted kidney during the first few months after transplantation was generally less than 50% of the donors original clearances. Despite of a hostile immunologic environment, therapy with immunosuppressive agents frequently was successful in reversing the rejection process, thus permitting many of the kidneys to achieve levels of renal function comparable to the single kidney of the donors and to the isografted kidney of identical twins.

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Summaries – Resumes

Hepatic dysfunction associated with renal transplantation


Five patients of 49 who had received renal transplantation for end-stage renal failure between November, 1966, and August, 1967, developed abnormal liver function at varying times following the transplant. All patients died, and abnormal morphological features were found in their livers. Three individuals were prepared with hemodialysis prior to transplantation and two had only peritoneal dialysis. All five patients were hemodialyzed after renal transplantation and were given multiple blood transfusions. After transplantation, they were treated with corticosteroids and azathioprine as basic immunosuppressive agents. Preoperatively, abnormal liver function tests were observed in only one patient but another had hepatosplenomegaly. Four of the individuals had clinical signs of hepatic insufficiency after renal transplantation. Jaundice was profound in two and mild in two others. One patient had no clinical signs of liver dysfunction although the transaminase levels were raised. Microscopically all the livers were abnormal with a variety of changes which included liver-cell necrosis, liver-cell pleomorphism, bileduct proliferation and an increase in portal-tract connective tissue. It was noted by the
authors that the morphological appearance of the livers in these cases was not entirely typical of
those seen in infectious hepatitis or with cytomegalovirus infection. The exact cause of the
hepatic dysfunction remains unclear.

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Erythrocytes in human transplantation: Effects of pretreatment with ABO groups-specific
antigens


Erythrocyte group antigens A and B can act as potent and group-specific transplantation antigens
in man. ABO group-incompatible recipients pretreated with such antigens were shown to reject
skin allografts obtained from donors incompatible for the same antigens in an accelerated (4-5
days) or white graft manner. Skin grafts applied to the same recipients from ABO-compatible
donors were accorded first set survival times. Intact erythrocyte suspensions and antigens
isolated from hog (A substance) and horse (B substance) stomachs, were equally capable of
inducing this type of allograft sensitivity. The latter observation broadens the spectrum of
heterologous antigens capable of inducing allograft sensitivity in the mammalian host and
provides a readily available, heat stable and water soluble source of antigens for further studies
of allograft rejection mechanisms in man.

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