Comments on CMV Infections in
Renal Transplant Patients

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Studies on 34 renal transplant's (in patients who received kidneys from living re-
lated donors) and 19 renal transplants (in patients who received kidneys from
cadaver donors) have now been completed. Sixty-two percent of the living related
kidney recipients became infected with cytomegalovirus (CMV) and 79% of the
cadaver kidney recipients became infected. Of the group receiving kidneys from liv-
ingar related donors, 11 had an HLA identical match, and seven of these became
infected, a fraction not significantly different from the overall percentage of infection
in the living related recipients.

The CMV antibody status of the kidney donor appears to be extremely important
in determining whether the recipient manifests CMV infection or not. Table 1 relates
the CMV complement fixation (CF) antibody status of the kidney donor to CMV in-
fecction in living related recipients. It can be seen that if the kidney comes from a
CMV antibody-positive donor there is a significantly increased chance that the
recipient will become infected ($P < 0.02$). Table 2 relates the antibody status of
kidney donors to CMV infection in seronegative recipients. The same increased risk
of infection if the kidney is from an antibody positive donor is noted ($P < 0.005$). Ta-
ble 3 relates the antibody status of the kidney donor to CMV infection in all recipi-
ents. Once again the pattern of increased risk of infection if the kidney is
received from an antibody-positive donor is evident ($P < 0.005$). This finding is strik-
ing and consistent.

In an attempt to demonstrate virus in the donor kidneys, 19 kidney biopsies were
cultured by growing the cells on glass or cocultivating with human embryonic lung
fibroblasts in an attempt to recover virus. Eight of the kidney biopsies were from
seronegative donors, six were from seropositive donors, and from five there was no
blood available. No virus was cultured from the kidney biopsies.

In five of the study patients and in three additional patients not in the study, we
were able to clearly identify a particular type of response to CMV infection. Six
weeks or longer after receiving the kidney, this group of patients developed fever,
chills, leukopenia, and lymphocytosis usually accompanied by an increase in creatinine and a transient elevation of SGOT. All five of the study patients had no
CMV CF antibody initially present, received kidneys from antibody positive donors,
showed a fourfold or greater rise in antibody, and excreted virus in their urine. Four
of the five had positive buffy-coat cultures, and four of the five were from the living
related group.

This clinical presentation was initially felt to be possible evidence of rejection, but

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no corroboration of rejection was demonstrated from kidney biopsies done on the first two patients. Nevertheless, these two patients were treated as though they were experiencing rejection. Subsequent patients were not treated by an increase of steroids and/or immunosuppression, and they underwent spontaneous recovery. Interestingly, of the two patients who were treated, one developed pancreatitis and the other developed a severe right lower lobe pneumonia. This syndrome is predictable if there is a good tissue match and it is known that the recipient is antibody-negative at the time of kidney transplant and has received a kidney from an antibody-positive donor.

If the recipients who had excellent kidney function are compared to those with less-than-excellent kidney function, there is no significant difference in the percentage of each group who are infected with CMV. This finding was valid for both the living related and cadaver transplant groups. In gross terms this raises a serious question as to the overall impact of CMV infection in kidney transplant patients.

Addendum. Since submission of this manuscript a study of CMV infectious renal transplant recipients has appeared with very similar findings (Ho, M., Suwansirikul, S., Dowling, J., Youngblood, L., and Armstrong, J., The transplanted kidney as a source of cytomegalovirus infection, *New Engl. J. Med.*, 293, 1109–1112, 1975).