However, we think most would agree that FMD in only one kidney from an older donor could be used with excision of the diseased area and reconstruction of the renal artery.

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Victims of cyanide poisoning make suitable organ donors

Received: 2 June 1995
Accepted: 6 June 1995

Sir: Renal transplantation is the elective treatment for a majority of patients suffering from end-stage renal disease. Despite the progressive growth in the number of renal transplantations presently being performed in most developed countries, the gap between the number of patients receiving a renal graft and the number of those on the waiting list is increasing. This shortage of organs makes it necessary for us to consider any and all potential donors and then to carefully evaluate each one. We report here on the results at 24 months of two renal transplantations performed using the kidneys from a donor who died of cyanide poisoning.

The donor was a 56-year-old male with no other significant past medical history than a long-standing depressive syndrome and two prior attempts at suicide. The patient was brought to the emergency room 20 min after a respiratory arrest occurred at his home as a consequence of the ingestion of a substance later identified as potassium cyanide.

Cardiorespiratory resuscitation techniques resulted in the rapid recovery of satisfactory cardiac function, although secondary, irreversible, hypoxic brain lesions ensued. We did not find the patient to be a suitable candidate for dicobalt edetate treatment and so we decided to implement the potential donor maintenance protocol. The donor rapidly reached hemodynamic stability with blood pressure of 140/80 mmHg and diuresis between 1 and 2 ml/min. Glycemia, electrolytes, GOT, GPT, hemogram, and coagulation parameters were normal and remained stable. Serum creatinine and urea were 1.3 mg/dl and 50 mg/dl, respectively. A qualitative determination of blood cyanide was negative 24 h later. Both kidneys were removed and preserved with Euro-collins solution.

The recipients of the kidneys were two men, aged 27 and 28 years, with end-stage renal disease secondary to IgA nephropathy and nephroangiosclerosis, respectively. Cold ischemia times were 16 h and 27 h, respectively. Both of the recipients were negative for lymphocytotoxic antibodies. They had three and two HLA antigen mismatches, respectively, with the donor. Immunosuppression consisted of prophylactic OKT3 and cyclosporin with low doses of prednisone. Both had immediate good renal function and were uneventfully discharged 9 days post-transplantation. Twenty-four months later, the first recipient has a serum creatinine of 1.6 mg/dl. The second recipient went back to dialysis 9 months after transplantation because of an irreversible acute rejection episode related to the patient's own decision to stop the immunosuppressive treatment. His serum creatinine was 2.2 mg/dl when last monitored.

Cyanide intoxication may occur in three different ways [8]: by hydrocyanic acid inhalation, which may prove fatal in less than 1 min [4]; by potassium, sodium, or calcium cyanide ingestion, which shows

a more delayed toxicity, between 10 min and 2 h, depending on the gastric repletion (faster absorption with an empty stomach); or percutaneously which is the case with some nitriles (Nitroprusside) [5, 7] that may release cyanide ions for several days and lead to a very delayed intoxication. After absorption, tissue distribution is unequal and dependent upon the affinity of cyanide to the metal ions (ferric iron, cobalt, copper). Its toxicity seems to be related to the inhibition (by fixation of the ferric iron) of cytochrome oxidase, which is the mitochondrial enzyme responsible for the oxidative phosphorylation [2] that represents 85 % of cellular oxygen utilization. Respiratory arrest is due to the extreme vulnerability of the nerve cells of the respiratory center to hypoxia. Cyanide excretion is usually achieved in less than 2 h if the physiological ways of detoxification are not saturated. Apart from the respiratory and the renal ways [1], the essential mechanism of detoxification in mammals is achieved by the hepatic enzyme rhodenase, reported by Lang [6], which converts the ionic form of cyanide into thiocyanide, which is nontoxic.

We feel that although victims of fatal cyanide poisoning may suffer irreversible, hypoxic brain injury, some of their organs may be suitable for transplantation [3]. Therefore, such patients should not be systematically discarded as potential donors until a careful evaluation of each one has been carried out.

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