

**Visual Improvement After Long-term Success of Pancreatic Transplantation**

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Several investigators have reported that pancreatic transplantation does not appear to have a favorable effect on diabetic retinopathy.<sup>1,2</sup> In a series of 22 patients who were followed up an average of 24 months, Ramsey and associates<sup>2</sup> reported that the functional success of the transplantation did not prevent progression of diabetic retinopathy and that visual loss secondary to diabetic maculopathy occurred in some patients.

Fourteen patients received cadaveric pancreatic transplants at our institution between March 1983 and November 1985 after or in conjunction with renal transplantation. Patients were immunosuppressed with oral cyclosporine and prednisone to which antilymphocytic globulin, the monoclonal antibody OKT3, and azathioprine were added as needed. Patients were examined at least every six

months with visual acuity measurements, ophthalmoscopy, and fundus photography. Photographs were evaluated and graded by using conventional criteria.<sup>3</sup>

Eight patients had functional pancreatic grafts and maintained euglycemia (mean total hemoglobin A<sub>1c</sub>, 5.0 to 7.4 g/dl) for more than six months without diabetic medications. Of this group of 16 eyes, no eyes showed improvement in diabetic retinopathy scores during the period of graft survival, five worsened, and 11 remained stable.

Four patients maintained functioning pancreatic grafts for more than 48 months. These patients were 31, 33, 38, and 45 years of age, respectively (Cases 1 through 4). All patients had had combined kidney and pancreatic transplants except the patient in Case 2, who had a kidney transplant performed before pancreatic transplantation. All four patients had been treated previously with panretinal laser photocoagulation in each eye. In Case 1, the patient also had had a vitrectomy in the right eye and macular grid treatment in the left eye.

At study entry, one patient (Case 1) had visual acuity of R.E.: light perception and L.E.: 20/50. Macular edema was noted in the left eye as determined by clinical examination and as seen on stereoscopic fundus photographs. Four years later, the macular edema had resolved and visual acuity in the left eye improved to 20/25.

The patient in Case 2 had visual acuity of 20/60 in both eyes with bilateral macular ede-

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ma at entry. Over the four-year period, a vitrectomy was required in each eye and additional panretinal photocoagulation was needed in the right eye. The macular edema resolved and visual acuity improved to 20/40 in each eye at 48 months.

In Case 3, the patient had visual acuity of R.E.: no light perception and L.E.: 20/20 before pancreatic and kidney transplantation. Visual acuity remained unchanged throughout the follow-up period and the previously treated proliferative diabetic retinopathy remained stable.

The patient in Case 4 had visual acuity of R.E.: 20/40 and L.E. 20/100 with diabetic macular edema in both eyes before transplantation. Additional panretinal photocoagulation was added during the course of follow-up. Visual acuity improved to R.E.: 20/30 and L.E.: 20/60 at 48 months, and the macular edema in each eye resolved.

Although no patient had a significant lens opacity before pancreatic transplantation, the left eye of one patient (Case 1) and both eyes of another patient (Case 2) developed posterior subcapsular cataracts associated with long-term prednisone treatment. Uneventful cataract removal and intraocular lens implantation were performed during the course of follow-up.

The findings in these patients confirm the previous finding that pancreatic transplantation in patients with preexisting proliferative diabetic retinopathy does not prevent progression of the retinopathy despite euglycemia. Our cases, however, suggest that long-term excellent visual function can be attained if the proliferative disease is treated appropriately. Four of six seeing eyes ultimately improved two lines of visual acuity or better, whereas the remaining seeing eyes maintained stable vision. Improvement of vision was not inconsequential, as it allowed two patients to drive their automobiles and return to work.

It is difficult to ascertain precisely how the functioning pancreatic grafts in these patients affected visual outcome. We find it noteworthy, however, that the macular edema noted initially in these patients eventually subsided without subsequent macular grid therapy. Although this improvement could be partially attributed to kidney transplantation and restoration of renal function, neither vision nor the maculopathy improved during the first 18 months after transplantation. These patients also had additional panretinal photocoagulation, but this type of laser treatment generally exacerbates diabetic maculopathy rather than improves it.

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## References

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## Duane's Retraction Syndrome in the Fetal Alcohol Syndrome

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The fetal alcohol syndrome is characterized by prenatal and postnatal growth retardation, mild to severe mental retardation, central nervous system, cardiovascular, and skeletal anomalies, and characteristic facial features including a narrow forehead, short and upturned nose, a long upper lip with indistinct or absent philtrum, and low nasal bridge.<sup>1</sup> A number of ocular malformations have been reported in the fetal alcohol syndrome including bilateral mild to moderate blepharoptosis (which usually becomes more noticeable in adolescence), horizontally shortened palpebral fissures, epicanthus, strabismus, retinal vessel tortuosity, optic nerve hypoplasia, myopia, cataract, Peters' anomaly, and microphthalmia.<sup>2</sup> We treated a patient with bilateral Duane's retraction syndrome type III (limited abduction and adduction of involved eye<sup>3</sup>) and the fetal alcohol syndrome.

An 11-year-old girl with fetal alcohol syndrome had characteristic facial features, growth retardation, and central nervous system dys-