

Prospective analysis of pancreatic grafts with duplex-Doppler ultrasound: value of resistive index in the diagnosis of rejection

R. Gilabert¹, L. Fernandez-Cruz², C. Bru¹, M.J. Ricart³, A. Saenz², and E. Astudillo²

Departments of ¹ Radiology and ² Surgery, and ³ Transplant Unit, University of Barcelona, Barcelona, Spain

The diagnosis of rejection and its differentiation from other causes of pancreatic graft dysfunction remain the basic problem in pancreas transplantation. A previous study with pulsed Doppler (PD) done at our institution demonstrated an increase of the resistive index (RI) during pancreatic graft rejection episodes [1]. The aim of the study was to determine prospectively the utility of duplex-Doppler (DD) ultrasound (US) in identifying the cause of pancreatic graft dysfunction. The major clinical categories were graft rejection and graft pancreatitis.

Key words: Duplex-Doppler ultrasound, pancreatic rejection – Pancreatic rejection diagnosis

Patients and methods

The study group included 23 whole pancreas grafts transplanted in to 22 patients (16 male, 6 female; mean age 36 years). Eighteen patients had combined kidney/pancreatic grafts from the same donor, 3 patients had sequential (not simultaneous) kidney and pancreatic grafts, and 2 patients had pancreas-alone grafts. The surgical technique consisted of whole pancreas transplantation with anastomosis of the graft's portal vein and celiac trunk to the recipient vena cava and iliac artery, respectively. The exocrine graft secretions were drained into the urinary bladder. DD examinations were performed with a 3.75-MHz transducer. Pancreatic size, echostructure, perigraft and intra-abdominal fluid were evaluated by US. The PD study was done to assess the permeability of the vascular pedicle at the hilar level. A venous and arterial Doppler spectrum (DS) were also obtained at the graft parenchyma (head, body, tail). The arterial DS was quantified by the RI [RI = (peak systolic velocity – end diastolic velocity)/peak systolic velocity], and the final RI was derived from the average of the 3 parenchymal levels sampled in the study. All patients underwent a baseline study 48–72 h after grafting, during graft dysfunction episodes and in the follow-up investigation.

The diagnosis of graft pancreatitis was based on clinical data (abdominal pain), increase of serum amylase and lipase levels (>800 U/l or >400 U/l, respectively), improvement after urinary drainage, rise of cytomegalovirus immunoglobulin (IgG)/IgM antibody or pancreatic biopsy (+) revealing inclusion bodies. The rejection status of the graft was based on clinical data (pain, temperature >38°C), reduction of urinary amylase levels (>50% of normal pre-rejection value), fluctuations of serum amylase level and improvement after rejection treatment.

Results

Normal grafts have a homogenous structure, and the graft size did not exceed the expected dimensions of a normal pancreas. Peripancreatic fluid was found in 22.7% of these grafts after surgery, but it did not persist more than 6 weeks in uncomplicated cases. On PD examination, the venous spectrum consisted in a continuous flow. The arterial waveform was characterized by a systolic peak followed by a continuous flow throughout diastole. No significant differences were observed between RI on baseline exploration (0.63 ± 0.01) and those obtained in stable grafts (0.62 ± 0.01). Nineteen episodes of graft pancreatitis were diagnosed, being documented at a mean time after transplantation of 246 ± 58 days ($r = 11-850$). The aetiology of these episodes was bladder dysfunction or urethral stenosis in 13 and infection in 5 (cytomegalovirus infection in 4, urinary tract infection due to *Pseudomonas aeruginosa* in 1). The cause of the remaining episode could not be determined. US results were pathological in 10 of 19 episodes of graft pancreatitis, disclosing perigraft fluid ($n = 10$), hypoechogenicity ($n = 2$) and heterogenous echostructure ($n = 1$). No increase of RI was observed during the episodes of graft pancreatitis: mean RI was 0.61 ± 0.001 , which is not significant when compared with RI prior to the episode of graft pancreatitis (0.63 ± 0.006) and with RI in stable grafts.

Some 24 episodes of pancreatic graft rejection were diagnosed; simultaneous kidney/pancreas rejection in 13 and pancreas-alone rejection in 11. Rejection episodes occurred at a mean time after transplantation of

54 + 11 days ($r = 7-235$). Kidney graft rejection preceded the pancreas rejection in 6 episodes. US results were pathological in 14 episodes of graft rejection, disclosing perigraft fluid ($n = 9$), increase of graft size ($n = 8$), heterogeneous echostructure ($n = 8$), hypoechogenicity ($n = 3$) and duodenal wall oedema ($n = 1$). PD showed an increase of RI in 18 episodes of pancreatic graft rejection: mean RI was $0.75 + 0.03$ in simultaneous kidney/pancreas rejection. These values were statistically significantly higher, $P < 0.001$, when compared with basal pre-rejection values; the mean increase of RI was also significantly higher in isolated pancreas rejection (38%) than in cases of simultaneous kidney/pancreas rejection (22%). Reversal of the rejection was seen in 18 episodes (75%). Loss of pancreatic function occurred in 6 episodes, with progressive worsening on PD study in spite of rejection therapy.

In conclusion, real-time US abnormalities were more prominent in pancreatic graft rejection than in graft pancreatitis. The RI in graft pancreatitis was not significantly different than that in stable grafts. Rejection was associated with a significant drop of the urinary amylase activity and a marked increase of RI. The mean increase of RI over basal values was 22% in pancreas grafts with simultaneous kidney rejection and 38% in isolated pancreas graft rejection.

Reference

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