LIFE-THREATENING DUODENAL GRAFT ULCERATIONS FOLLOWING SINGLE PANCREAS TRANSPLANTATION

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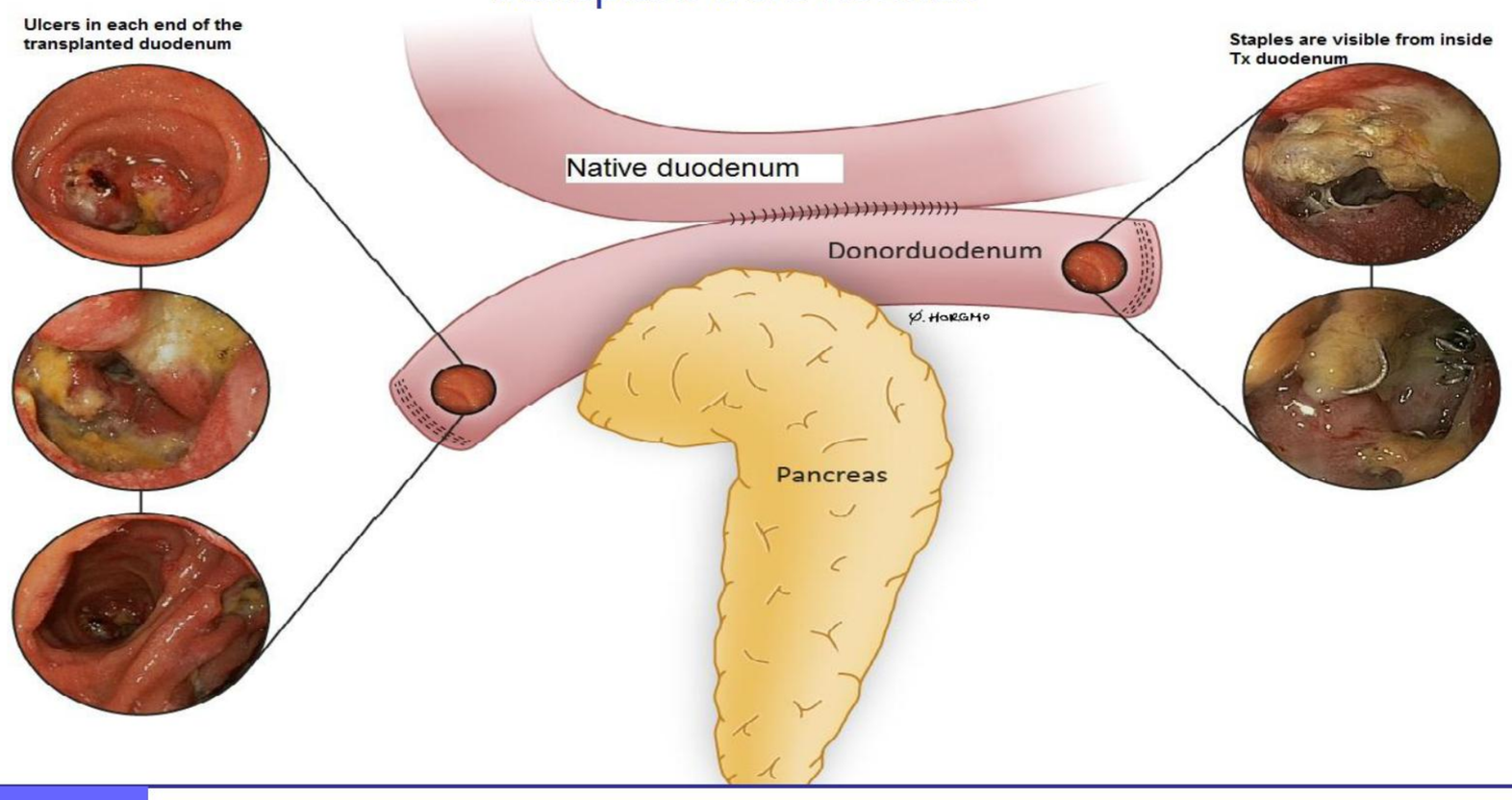
INTRODUCTION AND AIMS:

We hereby report a patient who experienced severe duodenal graft complication and eventual graft loss after Pancreas transplantation (PTx).

METHODS/CASE REPORT:

A 40-year old woman with brittle diabetes received a PTx.
Immunosuppression : ATG induction, prednisolone, tacrolimus (trough 8-12µg/L) and mycofenolate mofetil (MMF) 1g x 2.

Post operative course was excellent, no sign of rejections. Preemptively treated with oral Valganciclovir due to pos CMV PCR in blood.
Endoscopic ultrasound guided surveillance biopsies at week 3 and 6 post-transplant were normal.



RESULTS

12 weeks post-PTx a gastroscopy was performed due to abdominal pain and low Hgb. Gastroscopy showed multiple ulcers in donor duodenum and biopsies found duodenal graft rejection grade 4, no sign of pancreas rejection. This was successfully treated (biopsy proven) with i.v. methylprednisolone (total of 1,375g) and by an increased oral prednisolone dose. After 15 weeks abdominal pain led to a new gastroscopy which showed large ulcers in each end of the donor duodenum. There were no rejection or signs of viral, fungal and bacterial infection. CMV PCR was positive although negative CMV immunstaining. The patient received iv ganciclovir with some clinical effect.

The duodenal ulcers were extensive and considered at risk of perforation, but surgical intervention was not considered feasible without risk of graft loss. The further treatment were high-dose proton-pump inhibitor, prednisolone reduction and discontinuation of ASA, and reduction of MMF to 500mg x 2 since there were no signs of rejection. Pancreas function remained stable with C-peptides about 900mmol/L.

Repeated gatroscopies showed no sign of heeling and the abdominal pain persisted. She maintained treatment for CMV. At month 7 after PTx the patient experienced acute severe abdominal pain and a CT scan showed occluded arteries, and severe damage of the pancreatic head. Graftectomy was performed without further complications. Ultrastructural investigations of the resected graft showed chronic inflammation and no signs of rejection. Staining for CMV, Herpes 1, adeno- and polyomavirus were also negative.

CONCLUSIONS

The etiology of the severe transplant duodenal ulcerations remains unknown. Initially rejection of the duodenal graft was suspected and later biopsies also indicated CMV infection as a causal factor. However, several other factors such as duodenal ischemia, enteral enzymes and treatment with steroids, MMF and ASA may also have contributed.

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