

## CASE REPORT

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# Acute Retinopathy Following Pancreatic Head Resection for Chronic Pancreatitis: A Rare, Severe Complication

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### ABSTRACT

**Context** Chronic pancreatitis is a pancreatic disorder affecting endocrine and exocrine pancreatic functions with a variety of mainly abdominal symptoms.

**Case report** We report the rare complication of acute retinopathy with visual loss following pancreatic head resection due to chronic pancreatitis.

**Conclusions** Acute retinal dysfunction is a rare severe complication of acute and chronic pancreatitis. Early recognition and therapy are of utmost importance in restoring normal visual acuity and avoiding irreversible damage.

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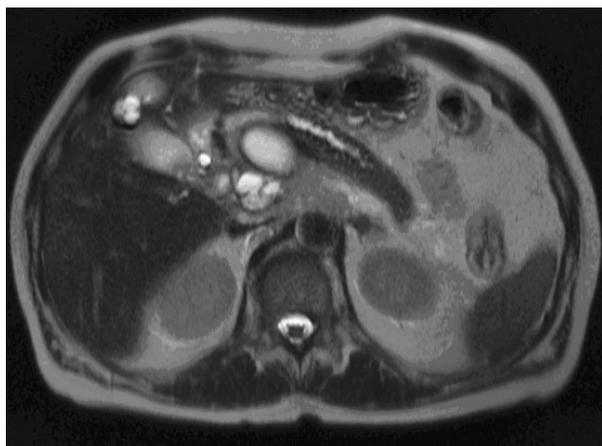
### INTRODUCTION

Chronic pancreatitis is a frequent pancreatic disorder with an incidence of 8/100,000 people per year in the western world [1]. During its course, endocrine and exocrine pancreatic functions are affected resulting in a variety of abdominal and metabolic symptoms. Thirty to 50% of all patients require interventional or surgical therapy due to complications of the disease [1]. Retinal affections in patients with chronic pancreatitis are almost exclusively associated with diabetes mellitus [2]. In contrast, non-diabetes-associated retinal affections similar

to post-traumatic retinopathy (Purtscher's disease [3]) have been observed in severe acute pancreatitis [4, 5, 6, 7]. This retinal affection has been described in infectious and autoimmune diseases as well as for chronic renal failure [8, 9, 10]. To our knowledge, no case with this form of retinopathy has been reported in chronic pancreatitis or as a complication following pancreatic resection.

### CASE REPORT

A 57-year-old female patient was admitted to our department with a history of chronic pancreatitis. The patient had suffered from recurrent episodes of upper abdominal pain accompanied by elevated pancreatic enzyme levels and chronic diarrhea for 7 years. She had no endocrine dysfunction and normal blood glucose and HbA1c levels (5.1% of total hemoglobin; reference range: 0-6.1%). NMR imaging showed typical signs of chronic pancreatitis with fibrosis and stasis of the Wirsung duct as well as calcifications and a cystic mass in the pancreatic head (Figure 1). The etiology of the pancreatitis remained unclear, alcoholic or autoimmune genesis were excluded anamnestically and by IgG4 measurement, respectively. Surgical therapy was indicated due to recurrent pain and the possibility of malignancy of the mass in the pancreatic head. The patient underwent a duodenum-preserving resection of the pancreatic head with a side-to-side pancreaticojejunostomy and Y-roux



**Figure 1.** Preoperative MRI scan showing dilatation of the pancreatic duct and a multicystic pancreatic head mass.

reconstruction [2]. The postoperative surgical course was uneventful; oral food intake was able to be started on the first day. The endocrine pancreatic function remained normal.

On the 7<sup>th</sup> postoperative day, the patient noticed visual affections with bilateral lower hemianopsia. Ophthalmologic examination revealed retinal microangiopathy, optic disc swelling and multiple retinal micro-infarction on both sides. Therapy was immediately started with heparin, hydrocortisone and somatostatin analogues. Thereafter, visual acuity improved rapidly. The patient was released after visual acuity was completely restored on day 15.

## DISCUSSION

Chronic pancreatitis is a common disease leading to disorders mostly associated with exocrine and endocrine insufficiency of the organ, including malabsorption, weight loss and diabetes mellitus. Most of the patients show a history of chronic alcohol consumption, suggesting that ethanol and its metabolites exert a toxic effect on the pancreas [11]. Chronic pain is the most frequent complication of the disease [12]. Furthermore, pancreas-related complications include obstruction of the duodenum, bile duct or large vessels (portal vein, superior mesenteric vein) by the mass in the head of the organ. Complications affecting other

organs are mainly related to long term diabetes mellitus and include polyneuropathy, nephropathy, atherosclerosis and slowly developing retinopathy [3].

Acute retinopathy is caused either by a direct retinal pathology, such as bleeding, ischemia or eye trauma, or it is a complication of other diseases. It has been described in association with severe trauma (Purtscher's disease), autoimmune diseases such as lupus erythematosus or chronic renal failure and HIV infections [4].

Purtscher-like retinal failure is morphologically characterized by cotton-wool spots, retinal hemorrhage, optic nerve edema and depression of receptive waves in the electroretinogram. It leads to reduction of visual acuity with consecutive anoptic areas and is usually, at least in part, reversible [5]. The pathophysiology of these forms of acute retinopathy remains unclear. Microembolism, edema, hypotensive perfusion failure and thrombotic vessel occlusion by leukocyte and platelet aggregations are discussed.

Several reports have described patients as having acute pancreatitis complicated by acute retinal affections [3, 7, 8]. Alcohol-related toxicity and intravasal coagulation have been considered as possible pathophysiological mechanisms [9, 10]. In chronic pancreatitis, chronic visual loss has been observed. A correlation with metabolic disorders such as zinc or vitamin A and diabetes mellitus are under debate [13, 14].

However, acute retinopathy associated with visual loss has never been described in relation to chronic pancreatitis, or interventional or surgical manipulations. Up to 50% of all chronic pancreatitis patients undergo interventional or surgical treatment during their history [1]. So far it is not known what the influence of surgical trauma on the development of acute retinal failure is. Theoretically, the operative trauma itself can induce acute pancreatitis. Furthermore, intraoperative circulatory disorders, inapparent fat embolisms or postoperative systemic inflammatory responses leading to leukocyte and platelet aggregations are possible triggers for pancreatic inflammation.

In the case reported, both the operation and the postoperative course were completely normal. No postoperative pancreatitis was evident. There was no circulatory problem which could have been symptomatic of an embolism. Moreover, the patient did not show any abnormal inflammatory response and recovered quickly. Therefore, we did not observe any risk factor or event leading to the development of acute retinopathy.

Some authors recommend routine fundoscopy for patients with acute pancreatitis to detect retinal abnormalities since the development of retinal affections indicates a severe course of acute pancreatitis [3, 7, 8, 9]. There is no doubt that early recognition is essential to restore retinal function completely.

No standardized therapeutic procedures exist for acute retinopathy. Management is solely symptomatic. The therapy of the patient reported here included high dose heparin anticoagulation (body weight-adapted application of low-weight-heparin twice a day), crystalloid infusion therapy (2,500 mL Ringer's solution per 24 h), steroid application (hydrocortisone 100 mg per day) and secretory inhibition of the remaining pancreas by somatostatin analogues (200 µg/24 h). Under this regimen visual loss was almost completely reversible within one week. Therapeutic success by means of the inhibition of platelets and blood coagulation as well as by the lowering of blood viscosity emphasizes the possible role of microcirculatory disorders and embolic events during retinal failure. Steroids were used as anti-edematous and anti-inflammatory agents for their non-specific supportive effects. Somatostatin analogues as a pancreas-specific therapeutic approach also seem to be effective. Thus, in our case, acute retinopathy might have also been triggered or aggravated by pancreatic enzyme secretion. The success of the multifactorial therapeutic approach underlines the complex pathogenesis of acute retinopathy.

## CONCLUSION

Acute retinal dysfunction with loss of visual acuity and anopsia is a rare severe

complication of acute pancreatitis and has never been described in association with chronic pancreatitis. We observed this complication for the first time after pancreatic head resection for chronic pancreatitis. The pathogenesis of acute retinopathy is not completely clear. Microcirculatory disorders as well as optic nerve edema seem to be important pathogenetic factors. Diagnostic measures are visus control and fundoscopy. Empiric management includes anti-coagulation, inhibition of pancreatic secretion and steroid application. Early recognition and therapy are of utmost importance in restoring normal visual acuity and avoiding irreversible retinal damage.

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