SECTION: PANCREAS

066

PLEURAL EFFUSION AS A COMPLICATION OF PANCREATITIS

Aiglová K.

2nd Clinic of Internal Medicine, Olomouc

Introduction

Pleural effusion complicates 25 % of cases in the course of acute pancreatitis. It also appears in patients with chronic pancreatitis along with pancreatic-pleural fistulas.

Patients and Methods

We present case reports of two patients with chronic pancreatitis and pancreatic fistulas.
These were patients examined for breathlessness, at whom the fluidothorax with hyperamylasemia in pleural exudat was diagnosed. Both were examined with ERCP and CT, during which a communication with pleural cavity was found.

Results

In case of the patient with right-side hydrothorax, stenosis of pancreatic duct, pancreatic-pleural fistula, with which a cystic formation in the hind mediastinum was also filled, the conservative therapy with octreotide and endoscopic intervention led to the regression of the diagnosed status.
Naso-pancreatic drainage was introduced. During the next examination a dilatation of stenosis was done as well as pancreatic-duodenal drainage through stents. In the case of the other patient a surgical solution was chosen, because the conservative therapy of chronic pancreatitis and repeated pleural punctures were not successful.

References

Johnson C. P., Imrie C. W., Pancreatic Disease, 167–188

067

FAECAL ELASTASE 1 – THE MARKER OF THE PROGRESSION OF CHRONIC PANCREATITIS

Dastych M. jr., Dítě P., Přecechtělová M., Žáková A., Starý K., Šenkyřík M.

Department of Gastroenterology, University Hospital Brno, Czech Republic

Introduction

In the course of chronic pancreatitis (CP) the exocrine pancreatic function (EPF) decreases. A simple test for determination of EPF could serve as a marker of progression of the disease. We aimed to evaluate a new polyclonal Elisa test developed for the determination of faecal elastase 1 as a marker of EPF.

Methods

110 patients were enrolled into this study. They were divided into two groups according to the Cambridge criteria. Fifty patients had mild or moderate CP (CPI) and thirty patients had severe CP (CPII). Thirty patients, without gastrointestinal disease, served as a control. Faecal Elastase 1 was examined in all patients. Elisa test (Bioserv) based on sandwich technique with four polyclonal antibodies was used for the determination of faecal Elastase 1.

Results

Faecal Elastase 1 concentrations were significantly lower in patients with CPII compared with controls (116.18 ± 121.21 vs 464.70 ± 97.54, $p < 0.0001$) and patients with CPI (116.18 ± 121.21 vs 389.82 ± 149.12, $p < 0.0001$) respectively. There was no significant difference between faecal Elastase 1 concentrations in patients with CPI and controls (389.82 ± 149.12 vs 464.70 ± 97.54, $p < 0.017$). Sensitivity for CPII was 96 %, for CPI below 40 %, specificity for CPII as well as for CPI was 90 %.

Conclusion

The new Elisa test is sensitive for determination of the EPF decrease in CPII but it failed in mild and moderate CP. On the other hand, the drop of EPF in mild and moderate CP is not pronounced. From this point of view the regular examination of the Faecal Elastase 1 can serve as a marker of progression of CP but not for the diagnosis of mild and moderate CP. Moreover, using the test in question, severe CP with or without exocrine pancreatic insufficiency can be distinguished.

References

Chr Loser *et al.* Faecal elastase 1: a novel, highly sensitive, and specific tubeless pancreatic function test. Gut 1996; 39: 580–586.
Lucio Gullo *et al.* Faecal Elastase 1 Determination in Chronic Pancreatitis. Dig Dis Sci 1999; 44: 210–213.
Lankisch *et al.* Faecal elastase 1: not helpful in diagnosing chronic pancreatitis associated with mild to moderate exocrine pancreatic insufficiency. Gut 1998; 42: 551–554.
Gunkel U. *et al.* Value of combinations of pancreatic function tests to predict mild or moderate chronic pankreatitis. Z Gastroenterol. 2001; 39: 207–211.

068

RESULTS OF TOTAL PANCREATECTOMY FOR CHRONIC PANCREATITIS AND CANCER

Froněk J., Ryska M., Bělina F., Strnad R., Langer D.

Transplant Surgery Department, Institute for Clinical and Experimental Medicine (IKEM), Prague, Czech Republic

Introduction

Total pancreatectomy (TP) seems to be the most extensive surgical treatment modality for patients with pancreatic cancer (PC) and chronic pancreatitis (ChP).
The aim of this retrospective study is to define the morbidity and mortality of this surgical procedure, survival rate in patients with pancreatic cancer, quality of life and pain score in patients with chronic pancreatitis.

Patients and Methods

Pancreatic cancer (PC): The advantages and disadvantages of TP have crystallized over the past 10 years. Current indications are as

follows: potentially multicentre ductal pancreatic cancer and risk of tumor growth in pancreatic-resection margin. In the period from 4/1998–11/2002 169 patients underwent surgery for PC in IKEM: eight of them (4.73 %, five men, three women, mean age 50 years, range: 44–67 years) were treated by total pancreatoduodenectomy with lymphadenectomy and splenectomy. R0 resection was performed in seven patients. Morbidity was 50 % (5 complications in four patients: pneumonia 2x, wound infection 2x, intraabdominal abscess 1x), mortality was 0 % in this group. Alive and tumor-free were two patients (25 %; 38 and 248 weeks), six died (75 %), mean survival was 28 (5–78) weeks. Hospital stay was 16 days (11–31 days).
Chronic pancreatitis (ChP): Total pancreatectomy was the last resort for patients with chronic pancreatitis and for patients with no response to previous therapy including surgery. The salvage procedure is beneficial only for half of the patients. During the same period 184 patients underwent surgery for ChP: seven were indicated for total pancreatoduodenectomy (3.80 %, 5 men, 2 women, mean age 46 years, range: 20–50 years). The indications were as follows: 4x severe ChP with complications and symptoms resistant to conservative care and previous surgery, 3x tumor suspicion. Etiology of ChP was 3x (42.87 %) alcohol abuse, 2x (28.57 %) idiopatic, 1x (14.29 %) juvenile pancreatitis, 1x (14.29 %) pancreas divisum. Morbidity in this group was 42.85 % (3 complications in 3 patients: intraabdominal abscess 1x, wound infection 2x), mortality was 0 %. Hospital stay was 14 days (10 – 16 days). Four of seven patients accepted our follow-up system. They filled in the Quality of life questionnaire. Apart from the cognitive part of the questionnaire we found better quality of life in all functioning scale scores (ns – because of the small cohort). The pain score decreased 48 % (ns).

Conclusion

The postoperative morbidity rate was higher after TP than the other resection procedures; the mortality rate in our group of patients was zero. Within TP there is a better ground for adequate lymphadenectomy. There is no risk for postoperative pancreatitis and leakage, and there is a good chance to control postoperative diabetes mellitus and exocrine insufficiency in majority of patients. In patients with ChP TP represents an operation of the last resort, where previous medical management and operative approaches failed.
In our group of patients the morbidity is acceptable for this kind of surgical procedures and the early results were successful. Mean survival rate in patients with PC is unfortunately similar to the palliative treatment modalities and not to the other resection procedures. Results are non-significant; two patients with low-grade tumor are still alive and tumor free. In the group of ChP it is difficult to collect data of the follow-up because of the well-known compliance of patients with ChP, according to the most common etiology. Patients, who accepted the follow-up, are doing well with better quality of life (ns) and lower pain score (ns).

References

Ihse I, Andrén-Sandberg A (1988) Surgical treatment: Total pancreatectomy. In: Beger et al.: Pancreas
Russell Ch (1998) Surgical Failues – Total pancreatectomy. In: Beger et al.: Pancreas
Gall FP, Műhe E, Gebhardt C (1981) Results of Partial and Total Pandreaticoduodenectomy in 117 Patients with Chronic pancreatitis. World J. Surg. 5, 269–275
Frey FC, Suzuki M, Isaji S, Zhu Y 1988 Pancreatic Resection for Chronic Pancreatitis. Surg Clin North Am, 69, 499–528
Miyake H, Harada H, Kunichika K, Ochi K, Kimura I (1987) Clinical Course and Prognosis of Chronic Pancreatitis. Pancreas, 2, 378–385.

069

THE TREATMENT OF EXTERNAL PANCREATIC FISTULAS WITH THE LONG-ACTING DEPOT FORM OF OCTREOTIDE – CASE REPORTS

Hoskovec D., Kašpar M., Varga J.

Department of Surgery of the 1st Faculty of Medicine and Institute for Postgraduate Medical Education
Department of Radiodiagnostics of the 1st Faculty of Medicine and Institute for Postgraduate Medical Education
University Hospital Na Bulovce, Prague, Czech Republic

Introduction

External pancreatic fistulas are a complication of pancreatic and other surgical procedures performed in the upper quadrants of the abdomen. Approximately 80 % of these fistulas are treated non-operatively. Surgery is reserved for fistulas that do not respond to conservative therapy.
Conservative therapy includes:
– Artificial nutrition (parenteral or enteral)
– Inhibition of exocrine secretion by Somatostatine and its analogues
– Endoscopic therapy.
The choice of surgical procedure depends on the fistula's origin. Internal drainage with Roux-en-Y pancreaticojejunostomy is usually used.

Patients and Results

We present the cases of three patients with postoperative external pancreatic fistulas. Two fistulas occurred after pancreatic surgery (Whipple procedure and necrosectomy for severe necrotizing pancreatitis). The third fistula occurred after reoperation for colorectal cancer. Two of them were treated by artificial parenteral nutrition without success. We used Sandostatin LAR® 20 mg in a single dose. All patients were managed as outpatients without nutritional support. The fistulas closed within 4 weeks after.

Conclusion

According to our experience low output pancreatic fistulas can be treated with a long-acting depot form of octreotide without any artificial nutritional support. Management of patients in outpatient departments is more economical, despite the cost of Sandostatin LAR (1 dose = 51 500 CZK). Cooperation with the health insurance agency is necessary.

070

A FAMILY WITH HEREDITARY PANCREATITIS CAUSED BY THE PRSS1 MUTATION R122H

Kolek A.[1], Koudová M.[4], Procházka V.[2], Horák D.[3], Grym J.[5], Václavec L.[6], Zoubek V.[7]

[1] *Department of Pediatrics,*
[2] *2nd Department of Internal Medicine and*
[3] *Department of Radiology, Palacký University, Olomouc*
[4] *Institute of Biology and Medical Genetics, Centre for Cystic Fibrosis, Charles University Prague*
[5] *Department of Pediatrics,*
[6] *Department of Surgery, Hospital Krnov*
[7] *Internal and Gastroenterological Department, Krnov*

Background

Hereditary pancreatitis (HP) is a rare autosomal dominant disorder inherited with variable penetrance. The point mutations R122H, N29I, A16V in the cationic trypsinogen gene (*PRSS1*) in the long arm of chromosome 7 (region 7q35) are most frequently found in patients with HP.

Patients and Methods

A family in which four members spanning three generations suffered from pancreatitis was studied. The most frequent *PRSS1* mutations, R122H and N29I were tested in eight of nine family members.

Results

Chronic pancreatitis was documented by a clinical course, biochemical results and by imaging methods. HRCT of pancreas was performed in the father (proband) and MR in both of his children. Endocrine and exocrine pancreatic insufficiency was documented in the father but not in affected children. The R122H PRSS1 mutation in the heterozygotic state was found in the father and in affected children. It is suggested that the father inherited the R122H mutation from his father (grandfather of the affected children) who had died of acute pancreatitis and his DNA could not be tested. The R122H mutation was excluded in other five asymptomatic family members.

Conclusion

The identification of cationic trypsinogen gene mutations in patients with pancreatitis confirms the diagnosis of HP. It may reveal affected family members with asymptomatic course of the disease.

References

Whitcomb DC, Gorry MC, Preston RA, *et al.* Hereditary pancreatitis is caused by a mutation in the cationic trypsinogen gene. Nat Genet 1996; 14:141–45.
Teich N, Ockenga J, Hoffmeister A, *et al.* Chronic pancreatitis associated with an activation peptide mutation that facilitates trypsin activation. Gastroenterology 2000;119:461–65.
Dítě P. et al.: Chronická pankreatitida, Galén, Praha, 2002.

071

CHRONIC PANCREATITIS WITH DIABETIC SYNDROME; OUR EXPERIENCES WITH TREATMENT

Loyková K., Loyková V., Ehrmann J.

2nd Department of Internal Medicine, University Hospital, Olomouc

Introduction

The exocrine and endocrine pancreases are linked very closely in terms of anatomy and physiology. It has been recognised in the past that pathological conditions in the exocrine tissue can cause impairment of endocrine function and vice versa.
Pancreatic exocrine dysfunction has been described frequently in IDDM and NIDDM patients. Most authors have tried to explain this finding as a diabetic complication. On the other hand, diabetes secondary to chronic pancreatitis might be more common than is believed to date.
Aetiology: 15 %.....idiopathic
80 %....alcoholic
Diabetes mellitus is a syndrome of impaired glucose metabolism. Pancreatic disease can induce diabetes mellitus. In acute pancreatitis temporary hyperglycaemia can be observed in about 50 % of patients, persisting diabetes may affect 1–15 %. In chronic pancreatitis about 60 % of patients are reported to have diabetes, of which 30 % is insulin-dependent.
Alcoholic pancreatitis is more often complicated with impaired glucose tolerance and diabetes mellitus regarded as another cause of pancreatitis.

Methods

In a retrospective study we aim to evaluate treatment of secondary diabetes mellitus in 47 patients with chronic pancreatitis, under review 5,6 years.

Results

We observed – occasion of insulinotherapy, type of insulin – occasion of nutritional subvention – diet, education, self-monitoring – organ complications

Conclusion

We evaluated 47 patients with chronic pancreatitis with secondary diabetes mellitus. All patients are treated with insulin – IIT. Very good education about diet, application, and self-monitoring is necessary. The selection of insulin type is also consequential. Humalog is effective.
There is also often a problem with malnutrition – most frequently in patients with alcoholic chronic pancreatitis. Application of nutritional subvention is then necessary.

072

USE OF NEOPTERIN AS A MARKER OF CELLULAR IMMUNE SYSTEM IN SMOKERS WITH CHRONIC PANCREATITIS

Pazourková M.[1], Tomandl J.[2], Tomandlová M.[2], Dítě P.[1]

[1] *Department of Internal Gastroenterology, Faculty Hospital Bohunice, Brno, Czech Republic*
[2] *Department of Biochemistry, Faculty of Medicine, Masaryk University, Brno, Czech Republic*

Introduction

Neopterin is a marker of the activated monocyte/macrophage system stimulated by interferon-gamma. The incidence of chronic pancreatitis is higher in smokers than in the rest of the population. The aim of the study was to determine whether cigarette smoking correlates with a marker of inflammation, neopterin.

Patients and Methods

Neopterin was analysed by ELISA in the serum of 21 subjects with chronic pancreatitis and 23 subjects with pancreatic carcinoma without focal or systemic infection. Both groups were divided into smokers and non-smokers. We also evaluated other factors related to disease, e.g. number of cigarettes smoked, duration of chronic pancreatitis, and age.

Results

The neopterin level in serum, corresponding to an activated cellular immune system, correlated significantly with cigarette smoking in patients with chronic pancreatitis (Spearman's correlation coefficient *Rs = 0.72, p = 0.0004*). In contrast, no significant correlation was found in patients with pancreatic cancer who smoke.

Conclusion

Neopterin as a marker of cellular immune response correlated positively with duration of smoking and number of cigarettes smoked. Given that chronic pancreatitis and smoking are the risk factors of pancreatic cancer, this study proposes the use of neopterin as a potential parameter in screening programs.

References

Murr C, Widner B, Wirleitner B, Fuchs D. (2002) Neopterin as a marker for immune system activation. Curr Drug Metab 3, 175–187.
Tomandl J. (1999) Pterins, methodological and diagnostic study. Thesis. Olomouc, Palacky University.

073

PANCREATIC CARCINOMA AND ICTERUS

Pešková M., Gürlich R.

1st Surgical Clinic Charles University, Prague, Czech Republic

Introduction

During recent years a number of authors presented results pointing out the higher occurrence of infectious complications in pancreatic resection following a pre-operative biliary drainage. At the same time it also takes away the surgical procedure.

Patients and Methods

In the period of 1990–2002 we performed 304 pancreatic resections for pancreatic carcinoma. We reviewed clinical data of patients included in this group who suffered from pre-operative icterus and underwent (group A) or did not (group B) endoscopic drainage of the biliary tree prior to surgery. We compared the results of surgical resection and presence of postoperative complications in both groups.

Results and Conclusion

We aim to open a discussion about this controversial theme by summarizing literature data and results of our study.

References

Aly E. A. H., Johnson C. D. (2001) Preoperative biliary drainage efore resection in obstructive jaudice. Dig.Surg.18, 84–89.
Bottger T. C., Junginger T. (1999) Factors influencing morbidity and mortality after pancreaticoduodenctomy: Critical analysis of 221 resections. World J. Surg. 23, 158–163.
Cameron J. L. (2001) Pancreatic cancor. BC Decker, Hamilton-London, 274.
Heslin M. J. *et al.* (1998) A preoperative biliary stenos is associated with increased complications after pancreaticoduodenectomy. 133, 149–154.
Leffler J.(2002) Návrh standardu léčby karcinomu pankreatu. Bulletin HPB, 10, 20–23.
Nakeeb H., Pitt H. A. (1995) The role of preoperative biliary decompression in obstructive jaudice. Hepatogastroenterology. 42, 332-337.

074

INVOLVEMENT OF ACTIVATED MACROPHAGES AND OXIDATIVE STRESS IN CHRONIC PANCREATITIS AND PANCREATIC CARCINOMA

Tomandl J.[1], Pazourková M.[2], Dítě P.[2], Tomandlová M.[1]

[1] *Department of Biochemistry, Faculty of Medicine, Masaryk University, Brno*
[2] *Department of Internal Gastroenterology, Teaching Hospital Bohunice, Brno, Czech Republic*

Introduction

Chronic pancreatic inflammation and oxidative stress may represent an early step in development of pancreatic cancer. The aim of the study was to assess the role of activated monocyte/macrophage system in patients with chronic pancreatitis and pancreatic carcinoma.

Patients and Methods

Twenty-one patients with chronic pancreatitis (CP) (age 50 ± 10 years) and twenty-three patients with pancreatic cancer (PCa) (age 45 ± 8 years) were studied. Neopterin was used for the evaluation of monocyte/macrophage system stimulated by γ-interferon and vitamins A and E. Malondialdehyde and the total antioxidant capacity of plasma served for the appraisal of the extent of oxidative stress. Neopterin was measured in serum by the enzyme-linked immunosorbent assay, malondialdehyde and vitamins A and E by high-pressure liquid chromatography. No patient showed any signs of infection.

Results

Patients with both CP and PCa had increased serum levels of neopterin compared to the physiological cut-off value in 96 % and 67 % cases, respectively. However, a more activated cellular immune system was observed in PCa as compared to CP ($P = 0.06$). Neopterin in PCa patients was correlated neither with the duration of

the inflammation nor with Ca 19–9. All tested parameters of oxidative stress were within the reference ranges. Nevertheless, we observed significantly increased levels of vitamin E in PCa as compared to CP ($p < 0.05$). Nevertheless, more significant correlations between serum neopterin and parameters of oxidative stress were observed in CP patients than in PCa patients.

Conclusion

Both CP and PCa patients had an activated monocyte/macrophage system. This activation had between the two patient groups a different susceptibility to markers of oxidative stress.

References

Birk D., Gansauge F, Gansauge S, Schwarz A, Beger HG. (1999) Levels of serum neopterin are increased in pancreatic cancer patients and correlate with the prognosis. Eur J Med Res 4, 156–160.

Murr C, Widner B, Wirleitner B, Fuchs D. (2002) Neopterin as a marker for immune system activation. Curr Drug Metab 3(2), 175–187.