ETIOLOGY OF ACUTE PANCREATITIS

Popovici Raluca*

* University of Oradea, Faculty of Environmental Protection, Gen.Magheru st., no.26, 410048, Oradea, e-mail: rugeraluca@yahoo.com

Abstract

The physical pathological and molecular research marked the understanding of the primary events that take place in the activation of the acute pancreatitis, though the early diagnosis in the pancreas diseases, generally, continue to be a source of frustration in the modern medicine.

Key words: easy acute pancreatitis, severe acute pancreatitis, trauma, ischemia, infections.

INTRODUCTION

The theory of pancreatic "autodigestion" was for a long time shaded by other two older theories: the theory of "the common biliary channel" (Opie), which sustains that the existence of a common biliary and pancreatic channel permits the biliary reflux in the pancreatic channel with activation of the pancreatic enzymes and the second theory, of the obstruction and pancreatic hyper secretion. Recently, the experimental researches suggested that the fragilization of the acinar cells' membranes or an incomplete way of storing would determine the discharge of lysosome hydrolase and their colocation with the pancreatic pro enzymes stored cellular (the theory of colocation) with the discharge of some small quantities of trypsin and which releases the waterfall of activations and enzyme auto activations.

OBJECTIVES

The Evaluation of the etiologic factors which lead to the appearance of the venous thrombosis, respectively to the pulmonary embolism.

MATERIAL AND METHODS

In order to accomplish the proposed objectives it was used the retrospective study.

The reaction of the study was extended for 5 years (01.01.2005-31.12.2009).

The material basis of the study included the observation papers of the patients, submitted at the archive of the hospitals, respectively the computerized data of the two units.

The data obtained were interpreted statistically on the basis of the determination and calculation of a series of indices: the report of the OR quota (having an interval of confidence of 95%), the chi square test, the Fisher test (for the identification of the significance degree), the absolute and relative frequency.

The processing of the data was accomplished with the help of the program Microsoft Office Excel 2003.

The representing of the results was made with the help of the graphics and tables.

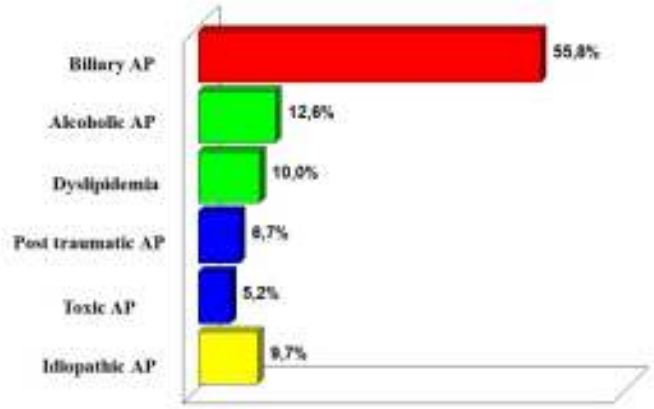
THE RESULTS

Most of the cases of Acute pancreatitis (AP) had a biliary etiology (55,8%), followed by the alcoholic one (12,6%), no matter the degree of severity of AP (53,7%, respectively 12,1% in PAU (Easy Acute Pancreatitis) and 58,3%, respectively 13,3% in PAS(Severe Acute Pancreatitis)) (p=0,324).

Table no. 1

The distribution of the cases depending on the etiology.

Etiology	PAU		PAS		Total	
	No.	**%**	**No.**	**%**	**No.**	**%**
Biliary AP	80	53,7	70	58,3	150	55,8
Alcoholic AP	18	12,1	16	13,3	34	12,6
Dyslipidemia AP	15	10,1	12	10,0	27	10,0
Post traumatic AP	12	8,1	6	5,0	18	6,7
Toxic AP	10	6,7	4	3,3	14	5,2
Idiopathic AP	14	9,4	12	10,0	26	9,7
Total	**149**	**100,0**	**120**	**100,0**	**269**	**100,0**



Graphic no. 1. The distribution of the cases of AP depending on the etiology.

DISCUSSIONS

The acute pancreatitis is a disorder that has numerous causes and an obscure pathogenesis. The calculi of biliary channel and the alcohol abuse represent together approximately 80% of the acute pancreatitis.

Most of the episodes of biliary pancreatitis are associated with transitory impacted of calculus in vial (which provokes an obstruction of the pancreatic channel, with arterial hypertension of the channel) or a transit from calculus, in duodenum. Other causes of acute pancreatitis are: different toxins, drugs, obstruction causes (as would be the malign affections or fibrotic of the Oddi sphincter), metabolic disorders, trauma, ischemia, infections, auto immune diseases.

10% of the case with acute pancreatitis, are not identified, because we are talking about idiopathic pancreatitis. The occult biliary micro lithiasis can be the cause of two thirds of the cases of idiopathic pancreatitis.

CONCLUSIONS

The majority of the cases of AP had a biliary etiology (55,8%), followed by the alcoholic (12,6%), no matter the degree of severity of AP.

REFERENCES

1. Acalovschi I., 1998, Terenul cu afectare pancreatică, în Tratat de patologie chirurgicală, vol. II, coord. G. Litarczek, Ed. Med. Bucuresti, pp. 234-246.
2. Atkinson S., Seiffert E., Bihari D., 1998, A prospective, randomized, double-blind, controlled clinical trial of enteral immunonutrition in the critically ill, Crit. Care Med., vol.26, no.7, pp. 1164-1171.
3. Appelros S., Borgstrom A., 1999, Incidence, aetiology and mortality rate of acute pancreatitis over 10 years in a defined urban population in Sweden, Br. J. Surg., pp. 8, 465-470.
4. Anderson R., Eckerwall G., Haraldsen P., 2000, Novel Strategies for the Management of Severe Acute Pancreatitis, Yearbook of Intensive Care and Emergency Medicine 2000, edited by J.L. Vincent, Springer Verlag, pp. 379-389.
5. Boucher B.A., 2000, Procalcitonin: clinical tool or laboratory curiosity?, Crit. Care Med., vol.28, no.4, 1224-1225.
6. Bryce Taylor, 1998, Acute pancreatitis in the critically ill, Principles of Critical Care, edited by J. Hall, G. Schmidt, L. Wood pp. 1269-1277.
7. Meier R., Sobotka L. 2000, Nutritional support in acute and chronic pancreatitis, in Basics in Clinical Nutrition, edited for ESPEN Courses, pp. 189-197.
8. Malledant Y., Tanguy M., Seguin P., 2000, Pancréatites aiguës graves, Actualités en réanimation et urgences pp. 155-168.
9. Rattner D.W., Warshaw A.L. 1992, Acute Pancreatitis, in Care of the Critically Ill Patient, edited by J. Tinker, M. Zapol, Springer Verlag, pp. 633-648.
10. Steer M.L., 1995, Acute Pancreatitis, in Textbook of Critical Care, edited by Shoemaker, Ayres, Grenvik, Holbrook, W.B. Saunders Comp., pp. 984-990.
11. Venneman I., Deby-Dupont G., Lamy M., 1993, Pancreatic Cellular Injury after Cardiopulmonary Bypass, in Yearbook of Intensive Care and Emergency Medicine edited by J.L. Vincent, Springer Verlag, pp. 297-309.
12. Vincent J.L., 2000, Procalcitonin: THE marker of sepsis?, Crit. Care Med., vol.28, no.4, pp. 1226-1227.