Tab. 3. *Chronic pancreatitis after 1 attack of acute pancreatitis – without metabolic syndrome (no. 28)*

		Alcohol	Biliary	Total
Obesity (BMI)	> 30.0	3 (10.7%)	5 (17.8%)	8 (28.6%)
HDL cholesterol	< 1.3 mmol/ L	2 (7.1%)	4 (14.3%)	6 (21.4%)
Glycaemia	> 5.6 mmol/ L	9 (32.1%)	4 (14.3%)	13 (46.4%)
Art. hypertension	> 130 mm Hg	3 (10.7%)	3 (10.7%)	6 (21.4%)
	> 90 mm Hg	3 (10.7%)	3 (10.7%)	6 (21.4%)
Triacylglyceridemia	> 1.7 mmol/ L	2 (7.1%)	3 (10.7%)	5 (17.8%)

Tab. 4. *Comparison of patients with and without metabolic syndrome*

	Metabolic syndrome – (no. 16)	**Metabolic syndrome – (no. 28)**	**P-value***
	Positive	*Negative*	
Obesity (BMI > 30)	82.3%	28.5%	P < 0.01
HDL cholesterol	35.2%	21.4%	NS.
Hyperglycaemia	76.5%	46.4%	NS.
Art. hypertension	70.5%	21.4%	P < 0.01
Hypertirglyceridemia	82.3%	17.8%	P < 0.01

**(significant P-value < 0.05), NS – not significant*

and conclusion in this context is that acute pancreatitis is not a diffuse, but rather a focal process, with the presence of changes that are subsequently known from the histomorphological picture of chronic pancreatitis.

The original view was that only very rarely is acute recurrent biliary pancreatitis a factor in the development of chronic pancreatitis, in contrast to the alcoholic form of acute pancreatitis (15). The explanation was as follows: while alcoholic acute pancreatitis histomorphologically affects the whole glandular parenchyma, in the case of the biliary form the changes are localised mainly in the peripancreatic region, without any response in the pancreatic tissue. Changes in pancreatic tissue are the main factor in glandular fibrotisation.

In our study we proved, as in the studies of Yadava (16) and Takayema (13) that the development of chronic pancreatitis from acute pancreatitis is not uncommon in the case of the biliary etiology of the disease, not just alcoholic. A number of studies have shown that smoking, in addition to alcohol, is a risk factor for the development of chronic pancreatitis from acute pancreatitis (12, 13). Why some people develop chronic pancreatitis after acute pancreatitis and others do not has been the subject of a number of studies (17, 18). Sandzen (19) published the so-called „hypothesis of the two strikes in the induction of chronic pancreatitis". The first „strike" is the presence of metabolic, genetic and external factors combined with the influence of other possible risk factors, e.g. smoking, to together create the changes which turn into chronic pancreatitis.

In 2015, Berlinsson et al. published a retrospective study evaluating a total of 1457 individuals after their first attack of acute pancreatitis (20). 48% of cases of acute pancreatitis were of biliary etiology, 17% of alcoholic etiology. Less than 10% of cases of acute pancreatitis were evaluated as the severe form of the disease. The risk factors for the progression from the acute form to chronic form were determined to be smoking, alcohol and the seriousness of the acute pancreatitis.

The meta-analytical studies performed by Sakarana et al. (21) after evaluating 19 previous works showed that 10% of people became chronic after a first attack of acute pancreatitis, 36% of people with acute recurrent pancreatitis ended up with chronic pancreatitis. Alcohol, smoking and male gender are listed as risk factors in the study.

A Dutch multicenter study evaluated data obtained from 669 patients after the individuals had acute pancreatitis in 15 different Dutch hospitals (22). After the first episode of acute pancreatitis the chance of pancreatitis relapsing was found to be 17%, and within 5-years 7.6% were diagnosed with chronic pancreatitis.

In the set of 59 persons suffering from chronic pancreatitis after their first episode of acute pancreatitis, we diagnosed a subgroup of 16 patients who were diagnosed with metabolic syndrome. Metabolic syndrome is a condition that is involved in gastroenterology in the development of gastroesophageal reflux (23) and Barrett's esophagus (24) but is mainly associated with the finding of non-alcoholic hepatic steatosis (NAFLD - non-alcoholic fatty liver disease) and non-alcoholic steatohepatitis (NASH - non-alcoholic steatohepatitis) (25, 26). Adipocytokines play an important role in the pathogenetic process similar to the inductive of a spectrum of pancreatic changes – non-alcoholic steatopancreatitisacute or chronic pancreatitis (27–30).

In our study, we showed that in the persons suffering from chronic pancreatitis after a first attack of acute pancreatitis the presence of metabolic syndrome plays an important role. Obesity, hypertriglyceridemia, arterial hypertension and diabetes mellitus (though the results concerning diabetes mellitus were on the border of statistical significance) are among the particularly important components of metabolic syndrome in the induction of pancreatic changes. In 2018, work performed by Melitas et al. (31) was published which presents 3 patients with the metabolic syndrome, steatopancreatitis and hypertriglyceridemia who developed chronic pancreatitis. The possible role of alcohol in the induction of pancreatic changes was excluded in all subjects. The authors, in accordance with our findings, refer to the important role of hypertriglyceridemia. In therapeutic practice, prevention of metabolic syndrome is important in preventing the induction of chronic pancreatitis (32) which arises in connection with those who have an attack of acute pancreatitis. The presence of metabolic