diagnosed with chronic pancreatitis until their attack of acute pancreatitis. According to the new „mechanistic definition" (10) of chronic pancreatitis, the initial stages of chronic pancreatitis cannot be determined, because at that time they have no clinical, only histological correlates at the cellular level and markers of tissue immunity. The etiology of genetically induced pancreatitis and autoimmune pancreatitis was excluded. Consequently, a diagnosis of metabolic syndrome was established on the basis of fulfilling the criteria of the so-called „harmonized" definition issued in 2009 (1). The data was collected retrospectively during the period from 2015 until 2017. Statistical calculation was performed by means of the khí quadrate test (the level of statistical significance that was chosen was p = 0.05).

Results

In a group of 59 patients in whom chronic pancreatitis was proven to be etiologically dependent on acute pancreatitis, the average age was 54 years (43-64 years). By gender, there were 32 men (54.2%) and 27 women (45.8%). Alcohol predominated in the etiology of the pancreatitis (52.5%). As for alcoholic etiology of the pancreatitis, the men were dominant (37.3%), while in the case of the patients suffering from the biliary form of pancreatitis the women were dominant (27.1%) (Tab. No. 1). In the group of 59 persons suffering from chronic pancreatitis after a first attack of acute pancreatitis, we diagnosed a subgroup of 16 patients (27.1%), mostly obese individuals, who had a diagnosis of metabolic syndrome in addition to that of chronic pancreatitis. The other subgroup consisted of 28 patients (47.6%) with chronic pancreatitis diagnosed after a first attack of acute pancreatitis but without the presence of metabolic syndrome. In 15 patients (25.3%) the diagnosis of metabolic syndrome and a follow-up could not be performed for several reasons (some patients had moved away or they were not willing to be included in the study).

The diagnosis of metabolic syndrome was determined on the basis of meeting the criteria of the so-called harmonized definition from 2009 (1).

In the group of persons with an alcoholic etiology of their pancreatitis, of the components of the metabolic syndrome obesity was present in 29.4%, hypertriglyceridemia and arterial hypertension in one third, and type 2 diabetes mellitus was found in 47.0% of the subjects. (Tab. No. 2).

In the group of patients with a biliary etiology of their pancreatitis, compared to the alcoholic etiology, the proportion of obesity was significantly higher, namely 52.9%. We found arterial hypertension and hyperglycemia in 29.4% of the subjects. Hypertriglyceridemia was diagnosed in 52.9% of patients. (Tab. No. 2).

In all, regardless of the etiology of the pancreatitis, in the 16-member group of people suffering from chronic pancreatitis after a first attack of acute pancreatitis with metabolic syndrome, obesity was present in 82.3% of the subjects. At the same time, 82.3% had hypertriglyceridemia as well. Arterial hypertension was found in 70.5%. Type 2 diabetes mellitus was found in 76.5% and a decrease in HDL cholesterol was found in one third of the subjects (Tab. No. 2).

We compared this group with the group of 28 patients suffering from chronic pancreatitis after a first attack of acute pancreatitis but without the presence of metabolic syndrome. In this group of people, regardless of the etiology of their pancreatitis, obesity was found in only 28.5% of the patients, type 2 diabetes mellitus was diagnosed more frequently, i.e. in 46.4%. Hypertriglyceridemia was also present in a significantly smaller number of patients, compared to persons with metabolic syndrome, which turned out to be in 17.8% (Tab. No. 3).

Comparing the two groups, a statistically significant difference was found in obesity, hypertriglyceridemia and arterial hypertension. We therefore believe that these components play a crucial role in the development of the process from acute inflammation of the gland to the stage of chronic pancreatitis, without metabolic syndrome (Tab. No. 4).

Finally, when evaluating the interval during which chronic pancreatitis occurred after acute pancreatitis, the mean time in subjects with metabolic syndrome was 12 months (10–14 months), while in the group without metabolic syndrome the time interval was longer, 20 months (16–29 months).

Discussion

In 1992, Kloeppel and Maillet (9) published their necrosis-fibrosis sequence hypothesis. It was a pathologist's view of the development of chronic pancreatitis from acute pancreatitis. The basis was a process of fibrotisation of initially necrotic tissue, leading to histomorphological and functional changes in pancreatic tissue. An important observation

Tab. 1. *Chronic pancreatitis in patients after 1 attack of acute panreatitis (no. 59)*

Aetiological factors	Alcohol	Biliary	Idiopathic	Total
	31 (52.5%)	24 (40.7%)	4 (6.8%)	59 (100%)
Gender (male)	22 (37.3%)	8 (13.6%)	2 (3.4%)	32 (54.2%)
Gender (female)	9 (15.2%)	16 (27.1%)	2 (3.4%)	27 (45.8%)

Tab. 2. *Risk factors – chronic pancreatitis – subgroup with metabolic syndrome - after 1 attack of acute pancreatitis (no. 16)*

		Alcohol	Biliary	Idiopathic	Total
Obesity (BMI)	> 30.0	5 (29.4%)	9 (52.9%)	0	14 (82.3%)
HDL cholesterol	< 1.3mmol/L	4 (23.5%)	2 (11.7%)	0	6 (35.2%)
Glycaemia	> 5.6mmol/L	8 (47.0%)	5 (29.4%)	0	13 (76.5%)
Art. hypertension	> 130 mm Hg	6 (35.3%)	5 (29.4%)	1 (5.9%)	12 (70.5%)
	> 90 mm Hg	5 (29.4%)	4 (23.5%)	0	9 (58.8%)
Triacylglyceridemia	> 1.7 mmol/L	5 (29.4%)	9 (52.9%)	0	14 (82.3%)