УДК 616.36-004-098

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COMPARATIVE ANALYSIS OF METABOLIC-ASSOCIATED ALCOHOLIC LIVER DISEASE VERSUS PURE ALCOHOLIC LIVER DISEASE

Introduction

Alcoholic liver disease (ALD) ranges from steatosis to cirrhosis and hepatocellular carcinoma, causing approximately 500,000 annual deaths globally – nearly 50% of all cirrhosis-related mortality [1]. Belarus has historically high alcohol consumption per capita, with strong links between alcohol intake and liver cirrhosis mortality [2]. In 2023, a multi-society consensus introduced new liver disease nomenclature, recognizing the frequent coexistence of metabolic dysfunction and alcohol consumption [3]. Three categories were established: metabolic dysfunction-associated steatotic liver disease (MASLD), ALD, and metabolic-associated alcoholic liver disease (MetALD) – an intermediate category for patients with at least one cardiometabolic risk factor consuming 20–50 g/day (women) or 30–60 g/day (men) of alcohol [3]. Both pure ALD and MetALD share pathogenic pathways including dysregulated lipid metabolism and immune activation [4]. However, MetALD involves synergistic injury where alcohol and metabolic dysfunction amplify each other through impaired fatty acid oxidation and enhanced inflammatory signaling [4]. MetALD may carry intermediate risks of fibrosis progression and mortality compared to pure ALD and MASLD, though Eastern European data remain limited [5]. No studies have characterized MetALD versus pure ALD distribution in Belarus. This distinction is essential for targeted screening and personalized treatment.

Goal

To compare clinical and demographic characteristics of MetALD versus pure ALD patients in a Belarusian hospital cohort and assess cardiometabolic risk factor prevalence and disease severity associations.

Material and methods of research

A cross-sectional study was conducted at Gomel State Clinical Hospital № 3, including 25 patients with hepatic anamnesis (history of liver disease) confirmed by clinical presentation, laboratory findings, and imaging. Data collection covered four domains: (1) demographics (age, sex, urban/rural residence); (2) alcohol consumption patterns (duration, daily intake); (3) cardiometabolic risk factors (BMI, diabetes, hypertension, dyslipidemia, abdominal obesity); and (4) liver disease severity (fibrosis scores, complications). Patients were classified according to the 2023 nomenclature [3]. Pure ALD was defined as alcohol intake >60 g/day with no cardiometabolic risk factors. MetALD was defined as alcohol intake with at least one cardiometabolic risk factor. Descriptive statistics were used.

The results of the research and their discussion

The study cohort comprised 25 patients receiving care for alcoholic liver disease at a Gomel city clinical hospital № 3. The demographic and clinical characteristics stratified by disease subtype are presented in table 1.

The characteristics of the laboratory data, disease progression and complications are presented in table 2.

Table 1 – Demographic and Clinical Characteristics of Patients with Pure ALD versus MetALD

Characteristic	Pure ALD (n=8)	MetALD (n=17)	Total (N=25)
Age, years(mean)	51.6	60.8	57.9
Sex, n (%)			
Male	3(37.5%)	12(70.6%)	15(60.0%)
Female	5(62.5%)	5(29.4%)	10(40.0%)
Residence, n (%)			
Urban (Gomel City	4(50.0%)	11(64.7%)	15(60.0%)
Rural (Gomel region)	4(50.0%)	6(35.3%)	10(40.0%)
Alcohol consumption			
Duration, years of alcohol consumption	19.2	17.4	18.0
Daily Intake, g(mean)	98.5	74.3	82.0
BMI category, n (%)			
Normal (25 kg/m^2)	8(100%)	0(%)	8(32.0%)
Overweight (25–30 kg/m^2)	0(0%)	9(52.9%)	9(36.0%)
Obese (>30 kg/m^2)	0(0%)	8(47.1%)	8(32.0%)
Cardiometabolic risk factors (%)			
Hypertension	0(0%)	14(82.4%)	14(56.0%)
Diabetes mellitus	0(0%)	7(41.2%)	7(28.0%)
Dyslipidemia	0(0%)	14(66.7%)	11(44.0%)
Liver disease severity, n (%)			
Cirrhosis present	7(87.5%)	12(70.6%)	19(76.0%)
Decompensated disease	5(62.5%)	8(47.1%)	13(52.0%)

Table 2 – Other Main Changes Between Pure ALD and MetALD

Domain	Pure ALD	MetALD	Main Interpretation
Ascites	More common (62.5%) and often large volume	Less common (47.1%)	More severe portal hypertension in pure ALD
Variceal bleeding	37.5%	17.6%	2× higher in pure ALD
Hepatic encephalopathy	50.0%	29.4%	More frequent in pure ALD
AST: ALT>1.5	75.0%	41.2%	Classic alcoholic pattern typical of pure ALD
GGT elevation	Marked (mean 387 U/L)	Moderate (mean 212U/L)	Greter cholestatic injury in pure ALD
Bilirubin, mean	Higher (76.4 μmol/l)	Lower (42.8μmol/l)	More severe hepatocellular dysfunction in pure ALD
Albumin, mean	Lower (26.4g/l)	Higher (31.2g/l)	Greater synthetic failure in pure ALD
Coagulopathy (INR>1.3)	75.0%	52.9%	More advanced synthetic dysfunction in pure ALD
Macrocytosis (MCV>100)	62.5%	35.3%	Direct alcohol effect more prominent in pure ALD
Thrombocytopenia	87.5% (mean 89×10^9/l)	64.7% (mean 127 ×10^9/l)	More severe hypersplenism in pure ALD
Anemia	75.0%(macrocytic)	47.1%(normocytic)	Different mechanisms nutritional vs inflammatory
Ferritin, mean	Markedly high (845 μg/L)	Moderately high(468μg/l)	Higher in pure ALD from hepatocellular release and inflammation

End of table 2

Domain	Pure ALD	MetALD	Main Interpretation
Renal Impairment	37.5% (hepatorenal syndrome)	23.5% (diabetic/ hypertensive)	Different mechanisms; more common and more severe in pure ALD
Potassium abnormality	Hyperkalemia (37.5%)	Hypokalemia (29.4%)	Opposite patterns: renal failure vs diuretic use
Chronic pancreatitis	25.0% (calcific)	17.6% (non-calcific)	Pancreas involvement more common and severe in pure ALD
Cardiomyopathy	25.0%	62.5%	Metabolic burden drives cardiac changes in MetALD
Gallbladder pathology	25.0%	47.1%	More common in MetALD, consistent with metabolic syndrome
Diabetes	–	Decompensation 70%, Compensated 30%	Diabetes dramatically accelerates disease progression

Conclusions

This study shows that there are two very different variants of alcoholic liver disease in Belarus.

On one side, it is pure ALD – typically younger women who drink heavily (around 100 g/day) but have no other health issues. They come to the doctor late, often already with advanced cirrhosis, ascites, bleeding varices, and severe liver failure. On the other side, MetALD affects mostly older men who drink moderately (around 75 g/day) but carry excess weight and have high blood pressure, diabetes, or unhealthy cholesterol levels. Their liver gets hit from two sides: alcohol and metabolic inflammation working together. They are easier to spot because their metabolic problems bring them to regular checkups, so we have a chance to intervene earlier. Patients with MetALD have less severe liver damage than patients with ALD. Yet even among them, those with diabetes have a much tougher course. Pure ALD needs us to actively look for heavy drinkers without metabolic red flags and get them help before complications set in. MetALD reminds us that when we see a patient with obesity, hypertension, or diabetes, we must also ask about alcohol because even moderate drinking can seriously damage the liver when combined with metabolic problems. Our findings also highlight that the Belarusian patients carry a particularly high burden of metabolic disease, so addressing both alcohol and lifestyle factors together is essential.

LITERATURE

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