CT PERFUSION PATTERNS IN AUTOIMMUNE HEPATITIS–PRIMARY BILIARY CHOLANGITIS OVERLAP SYNDROME

Battalova G.[1,2], Zholdybay Z.[1], Kalshabay Y.[2], Baiguissova D.[2], Mukhamejanova A.[2], Kabidenov A.[2], Baidauletova A., Abzhaparova B.[2], Nersesov A.[1], Grinberg V.[2], Baimakhanov B.[2]

1 - Kazakh National Medical University named after S.D. Asfendiyarov, Almaty, Kazakhstan
2 - Syzganov National Scientific Center of Surgery, Almaty, Kazakhstan

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Battalova G.,
https://orcid.org/0000-0003-4261-3537
Zholdybay Z.,
https://orcid.org/0000-0003-0553-9016
Kalshabay Y.,
https://orcid.org/0000-0003-0493-6685
Baiguissova D.,
https://orcid.org/0000-0001-5724-7707
Mukhamejanova A.,
https://orcid.org/0000-0002-4487-1604
Kabidenov A.,
https://orcid.org/0000-0001-5038-2033
Baidauletova A.,
https://orcid.org/0009-0000-5510-3590
Abzhaparova B.,
https://orcid.org/0000-0001-9790-8151
Nersesov A.,
https://orcid.org/0000-0002-8601-3966
Grinberg V.,
https://orcid.org/0000-0002-6827-3721
Baimakhanov B. ,
https://orcid.org/0000-0002-9839-6853

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Author for correspondence:
Kalshabay Y.,
PhD, National Scientific Center of Surgery named after A.N. Syzganov, orcid.org/0000-0003-0493-6685
email: tulkubas16@gmail.com,
tel: +7 (775) 1999522

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CT perfusion, autoimmune hepatitis, primary biliary cholangitis, overlap syndrome, hepatic fibrosis

Abstract

Background: Autoimmune cholestatic liver diseases, such as the primary biliary cholangitis–autoimmune hepatitis overlap syndrome, represent a rare and diagnostically challenging group of chronic liver disorders. Liver biopsy remains the diagnostic gold standard; however, its invasiveness limits widespread use. Computed tomography perfusion imaging has emerged as a non-invasive method capable of assessing hepatic hemodynamics and detecting early fibrotic changes. To evaluate and compare computed tomography perfusion parameters—arterial flow, portal flow, and perfusion index—in patients with fibrosis and cirrhosis secondary to primary biliary cholangitis–autoimmune hepatitis overlap syndrome and in healthy controls.

Materials and Methods: This single-center prospective study included 30 patients with primary biliary cholangitis–autoimmune hepatitis overlap syndrome (18 with fibrosis and 12 with cirrhosis) and 20 healthy controls (potential living liver donors). Perfusion parameters were quantified using deconvolution-based analysis.

Results: A statistically significant increase in arterial flow values was observed in patients with fibrosis ($p < 0.001$) and cirrhosis ($p < 0.001$) compared with the control group. There was no statistically significant difference in portal flow or perfusion index values among the fibrosis and cirrhosis groups. Inter observer reproducibility for arterial and portal flow measurements was excellent, interclass correlation coefficient = 0.873 and 0.837, respectively.

Conclusion: Computed tomography perfusion imaging enables quantitative assessment of hepatic hemodynamics in patients with primary biliary cholangitis–autoimmune hepatitis overlap syndrome. Increased arterial flow may reflect hemodynamic alterations associated with fibrotic remodeling, even before cirrhosis develops.

Introduction

Autoimmune cholestatic liver diseases represent a rare group of hepatobiliary disorders characterized by progressive immune-mediated inflammation and destruction of the bile ducts. The most common condition in this category is primary biliary cholangitis (PBC), which can progress to secondary biliary cirrhosis and its related complications.[1]

PBC, previously termed primary biliary cirrhosis, is a chronic autoimmune liver disease that causes gradual damage to the small intrahepatic bile ducts, ultimately resulting in fibrosis, cirrhosis, and liver failure.[2] Patients with PBC usually exhibit a biochemical pattern of chronic cholestasis, with alkaline phosphatase (ALP) levels elevated to at least twice the upper normal limit and remaining high for six months or longer. Gamma-glutamyl transferase (GGT) levels are typically increased up to five times the upper limit of normal. Total bilirubin generally stays within the normal range during early stages but rises as the disease advances. Aminotransferases

may show mild elevation. The serological hallmark of PBC is the presence of anti-mitochondrial antibodies (AMA), which are highly specific and diagnostic once other intrahepatic and extrahepatic causes of cholestasis are excluded. Approximately 5% of patients with clinical and histological features of PBC are AMA-negative.[3]

Around 10–20% of individuals with PBC may also develop features of autoimmune hepatitis (AIH), either concurrently or sequentially, and a minority of AIH patients may later present with PBC characteristics. This condition is referred to as the PBC–AIH variant (previously known as overlap syndrome).[4]

Diagnosing the PBC–AIH variant remains difficult in daily clinical practice, as no modern consensus criteria currently exist. The Paris criteria, introduced about two decades ago, are still commonly applied due to their reasonable sensitivity. Liver biopsy remains an essential diagnostic method for confirming the PBC–AIH variant and is recommended by both national and international guidelines. However, because biopsy is invasive and carries risks such as pain and, in rare cases, severe complications, there is a strong demand for non-invasive diagnostic tools for assessing and monitoring chronic liver diseases.[5]

Computed tomography (CT) perfusion has emerged as a promising non-invasive imaging technique capable of identifying early pathological changes in the liver—even before cirrhosis develops. CT perfusion parameters can help assess disease severity and hemodynamic alterations. Previous research has demonstrated the utility of CT perfusion in chronic viral hepatitis B and C, showing its effectiveness in detecting perfusion abnormalities related to fibrosis and cirrhosis. However, data on its use in autoimmune liver diseases are still limited.[6,7]

Given these considerations, CT perfusion imaging could play an important role in the non-invasive evaluation of autoimmune liver diseases. Perfusion parameters such as arterial flow (AF), portal flow (PF), and perfusion index (PI) may reflect the degree of fibrosis and the severity of liver dysfunction.

The aim of the present study was to evaluate and compare CT perfusion parameters (AF, PF, and PI) in patients with fibrosis and cirrhosis due to PBC–AIH variant syndrome and those in a control group consisting of potential liver donors.

Materials and methods

This study was approved by the Ethics Committee of the National Scientific Surgery Center named after A.N. Syzganov ((Approval No. 4 (92) dated 10th of November, 2023).

Study design and population This single-center prospective study was conducted between December 2023 and September 2025 and included 18 patients with PBC-AIH overlap related liver fibrosis and 12 patients with PBC-AIH overlap related liver cirrhosis. The control group consisted of 20 potential related living liver donors.

Inclusion criteria (AIH-PBC overlap group): patients of both genders, over 18 years, with serologically, biochemically and histologically confirmed liver fibrosis or cirrhosis secondary to AIH-PBC overlap syndrome before therapy.

Exclusion criteria (AIH-PBC overlap group): viral hepatitis, drug-induced hepatitis, other types liver disease (AIH, primary sclerosing cholangitis (PSC), and another overlap with AIH syndromes, Wilson's disease, hemochromatosis, non-alcoholic steatohepatitis), focal liver lesions, hepatic vein abnormalities (thrombosis, cavernous transformation), history of splenectomy, dysfunction of vital organs (cardiac, renal, or respiratory failure), and refusal to participate.

Inclusion criteria (control group): potential donors for living donor liver transplantation with clinically and radiologically confirmed healthy livers.

Exclusion criteria (control group): donors with hepatic steatosis >5% confirmed by biopsy, focal liver lesions (cysts or hemangiomas), and refusal to participate.

A flowchart illustrating the patient selection process is presented in Figure 1.

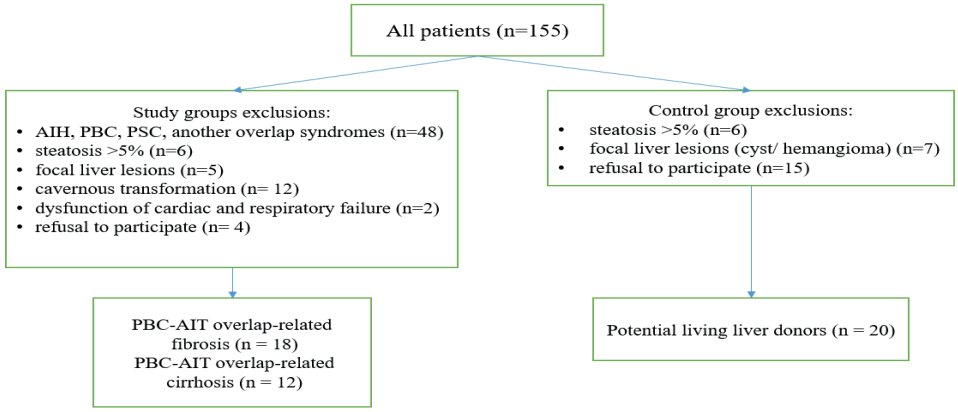


Figure 1.
Flowchart of study exclusion criteria

The patients were diagnosed based on laboratory, immunological, serological, diagnostic and clinical data. The severity of AIH-PBC overlap syndromein cirrhosis stages were assessed utilising the Child-Pugh score. Electronic medical records were reviewed to collect the data on patients' characteristics, including age, sex, body mass index (BMI), laboratory and serological findings.

CT acquisition protocol

CT perfusion imaging was performed in the cranio-caudal direction using a 160-slice MDCT scanner (*Aquilion Prime SP, Canon Medical Systems, Japan*). Scan parameters included a tube voltage of 80 kVp, tube current of 30 mAs, gantry rotation time of 0.5 s, and slice thickness of 0.5 mm, with a detector coverage of 8 cm. Patients were instructed to maintain shallow breathing without deep inspiration or breath-holding during image acquisition.

Initially, an unenhanced scan was acquired for anatomical localization of abdominal organs. Intravenous contrast medium was then administered via a 20-G cubital vein catheter using a dual-syringe automated power injector (*Ulrich, Germany*). A non-ionic iodinated contrast agent (*Ultravist 370 mgI/mL; Schering, Berlin, Germany*) was injected at a dose of 50 mL; in patients with obesity the dose was increased to 60 mL. The injection rate was 4.5-5.0 mL/s. The dynamic perfusion scan lasted 92 seconds and consisted of a single pre-contrast acquisition, followed by 20 consecutive scans at 4-second intervals and additional three scans at 5-second intervals.

Image post-processing

Two radiologists with 7 and 12 years of experience in abdominal radiology, respectively, who were blinded to the clinical and histopathological data, independently performed image post-processing. Perfusion analysis was conducted using the dedicated workstation Vitrea (*Canon Medical Systems, USA*) with the "4D Dual Input Liver" application.

Perfusion values were calculated using a dual-input maximum slope model. Regions of interest (ROIs) were manually placed in the abdominal aorta, portal vein, hepatic parenchyma, and spleen, with a graph generated to produce time-density curves (TDC), Figure2.

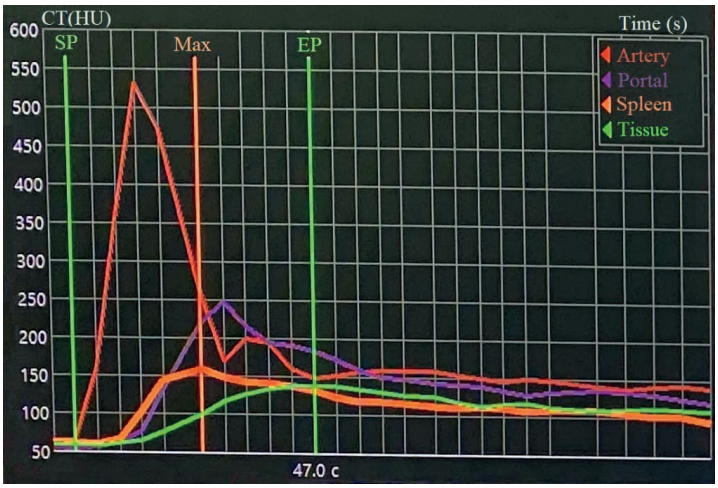


Figure 2.
Dual-input, maximum-slope liver perfusion model with time density curve for aorta, portal vein, spleen, andliver

For the liver, ROIs were positioned within segments III, IV, VI, VIII, carefully avoiding peripheral areas and large vessels. The ROI size was set to >1.0 cm². Perfusion parameters: arterial flow (AF) ml/min/100 ml, portal flow (PF) ml/min/100 ml and perfusion index (PI; HAF/(HAF + PVF)) % were calculated on perfusion maps (Figure 3).

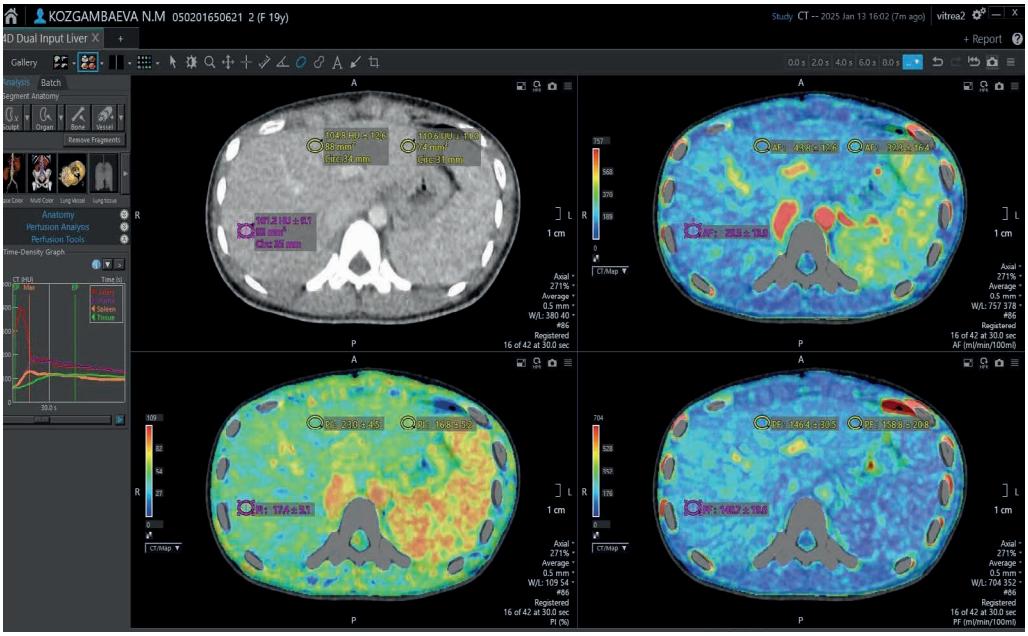


Figure 3. Perfusion maps: arterial flow (AF) – right top, portal flow (PF) – right bottom, perfusion index (PI) – left bottom

Ultrasound examination and liver biopsy

All patients, including those in the control group (potential liver donors), underwent abdominal ultrasound examination (*Hitachi, Japan*) by a radiologist with 15 years of experience in order to exclude focal liver lesions, hepatic steatosis, and hepatic venous pathology (thrombosis, occlusion, or cavernous transformation).

Subsequently, percutaneous liver biopsy was performed under ultrasound guidance in patients with liver fibrosis and cirrhosis secondary to AIH-PBC overlap syndrome 10-14 days before CT perfusion. Before the operation, blood routine examination and blood clotting time detection were conducted. Under local anesthesia and after aseptic preparation of the surgical field, a puncture needle was advanced into the peritoneal cavity through the right intercostal space. The passage of the needle through the liver capsule and into the parenchyma (segment VII) was confirmed by both real-time ultrasound visualization and tactile feedback. Tissue sampling was conducted using an automated biopsy device (*Pro-Mag™ Ultra, Canada*) equipped with a 16-G × 20 cm needle. A single liver core biopsy specimen was obtained, measuring 19 mm in length.

Histopathologic analysis

Tissue samples were fixed in 10% neutral buffered formalin. The specimens were then dehydrated using a standard protocol in a closed-system tissue processor (*Thermo SCIENTIFIC Excelsior AS, USA*) and embedded in paraffin. Paraffin sections 4-5 μm thick were cut on a rotary microtome (Sakura Accu-Cut SRM 200, Japan). For routine examination, the sections were stained with hematoxylin using the automated stainer (*Thermo SCIENTIFIC Gemini AS, USA*). Histochemical analysis was performed with the following staining methods: Masson's trichrome, Schiff's reagent, Perls' Prussian blue, orcein, and silver impregnation.

Prepared slides were examined under microscopes (ZEISS AXIO Imager Z2, Germany) equipped with an Axiocam 506 color camera and ZEISS ZEN Imaging Software.

The assessment of fibrosis, bile duct loss, chronic cholestasis, and necroinflammatory activity (cholangitis and hepatitis) was performed using the Nakanuma score and inflammatory activity was performed using the modified Ishak

classification and the Batts-Ludwig system, with correlation to the Meta-analysis of Histological Data in Viral Hepatitis (METAVIR) score.

Statistical analysis Descriptive statistics are reported as mean ± standard deviation (SD) for normally distributed variables and as median with interquartile range (IQR) for non-normally distributed variables. The normality of distribution was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Interobserver agreement between the two radiologists was evaluated using the interclass correlation coefficient (ICC), which was calculated on the full dataset with 95% confidence intervals (CI). Group comparisons of CT perfusion parameters were performed using one-way ANOVA test followed by the post hoc test or the Kruskal-Wallis test. The diagnostic value of CT perfusion parameters for AIH-fibrosis and AIH-cirrhosis was evaluated using the receiver operating characteristic (ROC) curve analysis, with calculation of the area under the curve (AUC), sensitivity, and specificity. A p value < 0.05 was considered to indicate statistical significance. All statistical analyses of the data were performed using IBM SPSS statistics (version 27).

Results

Biopsy results Among AIH-PBC fibrosis patients the stage F1 was observed in 7 patients, F2 in 8 patients, F3 in 3 patients, and F4 (AIH- cirrhosis) in 12 patients. The degree of inflammatory activity was A1 in 13 patients, A2 in 11 patients, A3 in 4 patients, and A4 in 3 patients.

Laboratory data Among AIH-cirrhosis patients according to the Child-Pugh score, Class A was identified in 4 patients, Class B in 6 patients, and Class C in 2patient.

No statistically significant differences were observed in age or gender distribution among the groups (p = 0.510 and p = 0.820, respectively). Both fibrosis and cirrhosis groups showed elevated levels of alkaline phosphatase (ALP) and **γ**-glutamyl transferase (GGT), reflecting the cholestatic pattern of liver injury. However, there were no significant differences in ALP (p = 0.794) or GGT (p = 0.055) values between the two subgroups.

The majority of patients in both groups were AMA-positive and IgG-positive, with positivity rates exceeding 80%, and no significant differences were found between the groups (p = 0.317 and p = 0.564, respectively).

Baseline demographic and biochemical characteristics of patients with AIH–PBC overlap syndrome (fibrosis and cirrhosis groups) and control subjects are summarized in Table 1.

Table 1.
Baseline characteristics of study groups

Parameters	AIH-PBC fibrosis	AIH-PBCcirrhosis	Control group	p- value
Age (years)	48.7±10.5	47.0±11.1	37.9±8.6	0.510
Gender (m:f)	2:16	2:10	9:11	0.820
ALP (U/L)	257.7±171.3	292.±151.2	N/A	0.794
γ-glutamyltransferase (U/L)	376.5±136.1	391.0±140.1	N/A	0.055
AMA positive (%)	89.7	94.8	N/A	0.317
IgG positive (%)	77.4	81.2	N/A	0.564

PBC: primary biliary cholangitis; ALP: alkaline phosphatase; AMA: anti-mitochondrial antibodies. Data are presented as mean ± standard deviation.

To assess interobserver variability, CT perfusion parameters — AF and PF— were measured independently by two radiologists (Radiologist 1 and Radiologist 2) with different levels of experience. The paired Student's t-test revealed no statistically significant differences between the measurements of Radiologist 1 and Radiologist 2 for AF (p = 0.871; ICC = 0.873) and PF (p = 0.681; ICC = 0.837) in patients with fibrosis and cirrhosis due to PBC–AIH overlap syndrome.

In the control group (liver donors), ICC values were also high: AF: 0.885, 95% CI (0.790-0.939) and PF: ICC 0.722, 95% CI (0.299-0.890). Subsequently, the mean values of AF and PF were used for further analysis.

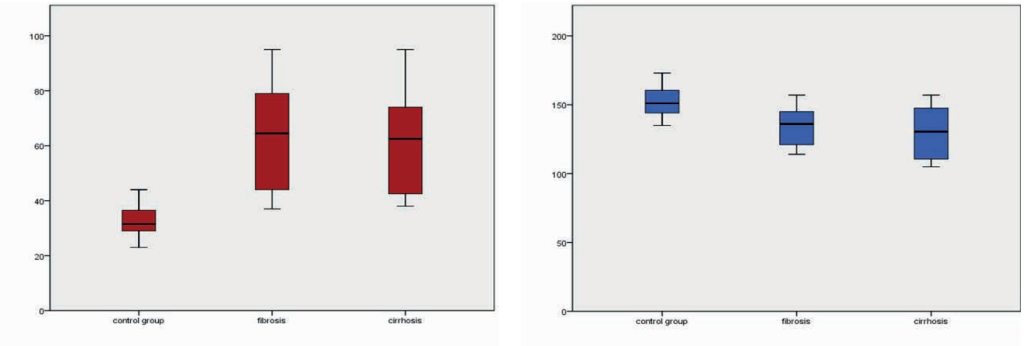
A one-way analysis of variance (ANOVA) revealed a statistically significant increase in AF values in patients with fibrosis ($p < 0.001$) and cirrhosis ($p < 0.001$) compared with the control group, while no significant difference in AF was observed between the fibrosis and cirrhosis groups ($p = 0.900$) in the setting of PBC–AIH overlap syndrome.

The PF parameter was significantly decreased in patients with fibrosis ($p < 0.001$) and cirrhosis ($p = 0.005$) compared with the control group, with no significant difference in PF between the fibrosis and cirrhosis groups ($p = 0.982$) in PBC–AIH overlap syndrome (Table 2, Figure 4).

Table 2.
CT perfusion parameters of the liver in patients with fibrosis and cirrhosis due to PBC–AIH overlap syndrome and in healthy liver parenchyma of potential donors

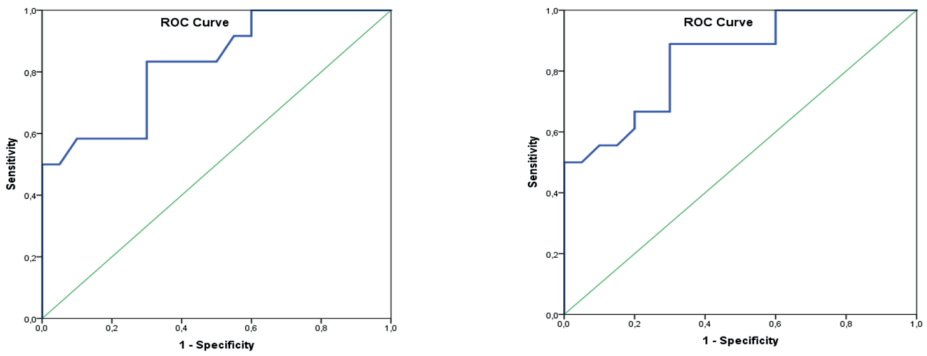
CT Perfusion Parameter	Fibrosis (n = 21)	Cirrhosis (n = 17)	Control Group (n = 20)	IntergroupDifferences
AF (ml/100 ml/min) Median 25th percentile 75th percentile	61.50 43.00 78.00	62.50 41.75 76.50	32.00 29.00 37.00	Fibrosis > Control ($p < 0.001$)* Cirrhosis> Control ($p < 0.001$)* Fibrosis = Cirrhosis ($p = 0.900$)
PF (ml/100 ml/min) Median 25th percentile 75th percentile	141.50 130.00 146.50	130.50 109.75 147.75	154.00 146.50 164.00	Cirrhosis< Control ($p = 0.005$)* Fibrosis < Control ($p < 0.001$)* Cirrhosis = Fibrosis ($p = 0.793$)
PI (%) Median 25th percentile 75th percentile	31.45 22.68 37.24	31.86 27.08 34.48	21.45 17.32 26.53	Control < Fibrosis ($p < 0.001$)* Control <Cirrhosis ($p = 0.001$)* Fibrosis = Cirrhosis ($p = 0.982$)
AF-arterial flow; PF-portal flow; PI- portal Index; * P value ⩽ 0.05 was considered statistically significant				

Figure 4.
Boxplot illustrating AF and PF values in patients with liver fibrosis and cirrhosis, as well as in the control group, in the context of AIH



An ROC curve was constructed to differentiate PF values between patients with PBC–AIH overlap syndrome and the control group (Figure 5).

Figure 5.
Receiver operating characteristic curve for differentiating the PF parameter in the AIH–PBC overlap fibrosis and cirrhosis groups from the control group



The AUC for patients with liver fibrosis due to AIH–PBC overlap was 0.842 (95% CI: 0.719–0.965), with a cut-off value of 154.5, sensi-

tivity of 89%, and specificity of 60%. The AUC for patients with liver cirrhosis due to AIH–PBC overlap was 0.825, with a cut-off value of 155.5, sensitivity of 92%, and specificity of 60%.

Discussion

The overlap variant of PBC and AIH represents a clinically significant, though relatively uncommon, form of autoimmune liver disease characterized by the coexistence of cholestatic and hepatitic features. Diagnosis of this hybrid phenotype remains challenging due to the absence of a modern international consensus definition. In current clinical practice, the Paris criteria remain the most widely applied diagnostic tool, showing reasonable sensitivity and specificity, although they were developed more than two decades ago.[8]

Liver biopsy continues to play a central role in the diagnosis of overlap syndrome, especially in seronegative (AMA-negative) variants or in cases with suspected interface hepatitis. Histological evaluation allows confirmation of both bile duct injury and inflammatory activity/fibrosis staging. However, biopsy is invasive and associated with procedural risks, underscoring the clinical need for reliable non-invasive biomarkers and functional imaging. Current guidelines on autoimmune liver diseases also recommend liver biopsy in cases of uncertain serology or suspected overlap syndromes.[3,8]

From a clinical and prognostic perspective, patients with PBC–AIH overlap syndrome often experience a more aggressive disease course and a higher risk of portal hypertension and need for transplantation compared to isolated PBC or AIH. Therefore, early and accurate disease stratification is critical for optimizing management and outcomes. Recent cohort studies and systematic reviews confirm the heterogeneity of disease trajectories and emphasize the importance of individualized therapeutic approaches.[9]

Treatment of overlap syndrome typically combines anticholestatic therapy—mainly ursodeoxycholic acid (UDCA)—with immunosuppressive agents, especially when AIH features (interface hepatitis, elevated transaminases) are predominant. Meta-analyses and systematic reviews have shown that combination therapy (UDCA + corticosteroids ± azathioprine) provides better biochemical remission rates and transplant-free survival compared with monotherapy, although evidence is mostly derived from retrospective cohort studies, and randomized trials remain scarce.[10]

In the present study, analysis of hepatic perfusion parameters (AF, PF, PI) using CT perfusion revealed consistent shifts toward increased arterial contribution and decreased portal venous flow in patients with fibrosis and cirrhosis compared to controls. These findings are consistent with prior reports showing that CT perfusion parameters change in chronic diffuse liver diseases and correlate with fibrosis stage and portal hypertension. Thus, CT perfusion imaging emerges as a promising non-invasive technique for quantitative assessment of hepatic hemodynamic alterations in autoimmune and cholestatic liver diseases.[11]

Despite encouraging results, several limitations should be acknowledged. First, most published CT perfusion studies have focused on viral or post-necrotic fibrosis, whereas data specifically addressing autoimmune overlap syndromes are still scarce, limiting generalizability. Second, methodological variability—including perfusion software algorithms, ROI selection, scanning parameters, and contrast protocols—complicates direct comparisons across studies and highlights the need for protocol standardization. Finally, radiation exposure and limited availability of CT perfusion in routine clinical practice remain important considerations for future implementation.[12]

In our study, quantitative CT perfusion parameters were assessed in patients with fibrosis and cirrhosis secondary to AIH-PBC overlap syndrome and compared with a control group of potential living liver donors. In AIH-PBC overlap syndrome patients, AF and PI values were significantly higher, while PF was significantly lower compared with controls. No statistically significant differences in AF, PF, and PI were found between the fibrosis and cirrhosis subgroups. These findings indicate that PF reduction and compensatory AF increase occur as early as the fibrosis stage.

Traditional serological and biochemical markers often fail to reliably differentiate fibrosis stages in PBC–AIH overlap, as demonstrated in our cohort. Integration of quantitative imaging biomarkers—such as CT perfusion, spectral or dynamic CT, MR perfusion, and elastography—may improve risk stratification and monitoring of fibrosis progression.

Future prospective multicenter studies should aim to: (a) validate perfusion threshold values for clinically meaningful outcomes; (b) compare the diagnostic performance of CT perfusion with established non-invasive methods (elastography, serum fibrosis indices); and (c) assess how perfusion metrics change in response to therapy (UDCA ± immunosuppression) and correlate with histological improvement.[7]

Limitations The present study has several limitations. The sample size was relatively small, which may reduce the statistical power and generalizability of the findings. Furthermore, although CT perfusion provides valuable quantitative information, it involves radiation exposure and requires standardized acquisition and analysis protocols.

What's known? PBC and AIH represents a clinically important but relatively uncommon autoimmune liver disease characterized by the coexistence of cholestatic and hepatitic features. Liver biopsy remains the gold standard for diagnosis, particularly in AMA-negative variants or when serology and biochemistry are inconclusive. Histology allows confirmation of interface hepatitis and small bile duct destruction, supporting a definitive diagnosis. However, because liver biopsy is invasive and carries risks such as bleeding and pain, there is a growing need for reliable non-invasive tools for diagnosis, staging, and disease monitoring.

What's new? This study demonstrated that CT perfusion imaging can detect hemodynamic alterations in patients with chronic autoimmune liver diseases such as PBC–AIH overlap. Specifically, arterial flow (AF) was significantly increased, while portal flow (PF) was significantly decreased in patients with fibrosis and cirrhosis compared with controls, consistent with the known hemodynamic shift from portal to arterial dominance in chronic liver disease. No significant difference in perfusion parameters (AF, PF, PI) was observed between fibrosis and cirrhosis, suggesting that CT perfusion changes occur early and may reflect functional impairment before advanced morphologic alterations develop.

Conclusion

In conclusion, the PBC–AIH overlap syndrome is a clinically important autoimmune liver disease with variable presentation and potentially poorer prognosis than isolated PBC or AIH. A pronounced increase in CT perfusion parameter AF and a decrease PBC–AIH overlap syndrome hepatitis.

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Author Contributions: B.B., Zh.Zh., N.A.: were responsible for the study concept, design, supervision, and approval of the final manuscript version; B.G., K.Y.: contributed to data collection, preparation, and primary verification; B.D., A.B., M.A.: performed statistical analysis and drafted the manuscript sections on Materials and Methods and Results; G.A., K.A., M.A.: prepared the Introduction and Discussion sections. All authors reviewed and approved the final version of the manuscript.

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