



INDONESIAN JOURNAL OF CLINICAL PATHOLOGY AND MEDICAL LABORATORY (IJCPML)

[PERHIMPUNAN DOKTER SPESIALIS PATOLOGI KLINIK INDONESIA](#)

✦ [P-ISSN : 08544263](#) < > [E-ISSN : 24774685](#)



0

Impact Factor



650

Google Citations



Sinta 2

Current Accreditation

[Google Scholar](#) [Garuda](#) [Website](#) [Editor URL](#)

History Accreditation

2016 2017 2018 2019 2020 2021 2022 2023 2024 2025 2026

[Garuda](#) [Google Scholar](#)

[Immunogenicity Assessment on Clinical Trials of SARS-CoV-2 Vaccines](#)

[Indonesian Association of Clinical Pathologist and Medical laboratory](#) [INDONESIAN JOURNAL OF CLINICAL PATHOLOGY AND MEDICAL LABORATORY Vol 28, No 2 \(2022\) 202-208](#)

📅 2022 📄 DOI: [10.24293/ijcpml.v28i2.1975](#) 🏆 [Accred : Sinta 2](#)

[Concordance Test of Various Erythrocyte Indices for Screening of Beta Thalassemia Carrier](#)

[Indonesian Association of Clinical Pathologist and Medical laboratory](#) [INDONESIAN JOURNAL OF CLINICAL PATHOLOGY AND MEDICAL LABORATORY Vol 28, No 2 \(2022\) 137-142](#)

📅 2022 📄 DOI: [10.24293/ijcpml.v28i2.1842](#) 🏆 [Accred : Sinta 2](#)

[Gene Expression of SOX2, OCT4, and Nanog by Small Molecule Compound VC6TFZ on Peripheral Blood Mononuclear Cell](#)

[Indonesian Association of Clinical Pathologist and Medical laboratory](#) [INDONESIAN JOURNAL OF CLINICAL PATHOLOGY AND MEDICAL LABORATORY Vol 28, No 2 \(2022\) 115-120](#)

📅 2022 📄 DOI: [10.24293/ijcpml.v28i2.1759](#) 🏆 [Accred : Sinta 2](#)

[Correlation between Inflammatory Markers of Platelet Index and Vitamin D with Body Mass Index](#)

[Indonesian Association of Clinical Pathologist and Medical laboratory](#) [INDONESIAN JOURNAL OF CLINICAL PATHOLOGY AND MEDICAL LABORATORY Vol 28, No 2 \(2022\) 161-164](#)



INDONESIAN JOURNAL OF
**Clinical Pathology and
 Medical Laboratory**

p-ISSN : 0854-4263
 e-ISSN : 2477-4685

Published by
 Indonesian Association of Clinical
 Pathologist and Medical Laboratory

Majalah Patologi Klinik Indonesia dan Laboratorium Medik

HOME ABOUT LOGIN REGISTER SEARCH CURRENT ARCHIVES ANNOUNCEMENTS AUTHOR GUIDELINE EDITORIAL TEAM

Home > Archives > Vol 26, No 3 (2020)

Vol 26, No 3 (2020)

Table of Contents

Articles

- | | |
|--|-----------------------------|
| <p> The Association of Syphilis Infection and Other Risk Factors with Immunity of Patients with HIV on Anti-Retroviral Therapy</p> <p><i>Pik Siong, B. Rina A. Sidharta, Amiroh Kurniati</i></p> | <p> PDF
Pg. 257-265</p> |
| <p> Identification of Risk Factors Characteristics of Transfusion Reaction</p> <p><i>Eko Putri Rahajeng, Raehana Samad, Rachmawati Muhiddin</i></p> | <p> PDF
Pg. 266-271</p> |
| <p> Analysis of Smear Microscopy and Culture Conversion Results in Multidrug-Resistant Tuberculosis Patients with and without Type 2 Diabetes Mellitus</p> <p><i>Henny Fauziah, Aprianti S, Handayani I, Kadir NA</i></p> | <p> PDF
Pg. 272-276</p> |
| <p> The Difference of Lymphocyte, hs-CRP, and Electroencephalogram with and without Simvastatin in Acute Ischemic Stroke</p> <p><i>Chairil Amin Batubara, Aldy Safruddin Rambe, Nindia Sugih Arto</i></p> | <p> PDF
Pg. 277-281</p> |
| <p> Comparison of 25-Hydroxyvitamin D Levels in Acute Coronary Syndrome</p> <p><i>Mirna Rahmafindari, Leonita Anniwati, Muh. Aminuddin, Ferdy R. Marpaung</i></p> | <p> PDF
Pg. 282-288</p> |
| <p> Cytokines, Autoantibodies, and Complements in Active Systemic Lupus Erythematosus Patients from Javanese Population</p> <p><i>Yuliasih Yuliasih, Lita Diah Rahmawati, Putu Ayu Niken Amrita, Setiati Widyaningrum, Dodi Kriswanto</i></p> | <p> PDF
Pg. 289-296</p> |
| <p> Diagnostic Test of Serum Pregnancy-Associated Plasma Protein-A Level as Biomarker for Early Diagnosis of Acute Myocardial Infarction</p> <p><i>Novida Dwi Astuti, JB. Suparyatmo, Amiroh Kurniati</i></p> | <p> PDF
Pg. 297-302</p> |
| <p> The difference of Reticulocyte Hemoglobin Equivalent Pre- and Post-Ultrafiltration Hemodialysis in Patients with Chronic Kidney Disease</p> <p><i>Ni Made Rindra Hermawathi, Arifoel Hajat, Yetti Heraningsih, Widodo Widodo</i></p> | <p> PDF
Pg. 303-306</p> |
| <p> Prevalence of Expanded Dengue Syndrome in Patients with Dengue Virus Infection at the Dr. Soetomo Hospital Surabaya in 2017 – 2018</p> | <p> PDF
Pg. 307-311</p> |

SUBMIT



ISSN

p-ISSN: 0854-4263

e-ISSN: 2477-4685

LINK

Template

Statement Letter

Copyright Transfer Form

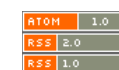
INDEX



USER

Username
 Password
 Remember me

CURRENT ISSUE




JOURNAL CONTENT

Search
 Search Scope
 All

Browse

[By Issue](#)[By Author](#)[By Title](#)

Avina Fimas Adissadah, Aryati Aryati, Suhintam Pusarawati

 [The Correlation between Serum Growth Differentiation Factor-15 Levels and Post-Acute Myocardial Infarction Acute Heart](#)

 PDF

Pg. 312-316


Kenny Mayavani, JB. Suparyatmo, Dian Ariningrum

 [Bacterial Profile and Antibiogram of Hospital-Acquired Pneumonia and Ventilator-Associated Pneumonia Patients in ICU of Raden Mattaher Hospital Jambi](#)

 PDF

Pg. 377-311


Sotianingsih Sotianingsih, Samsirun H, Lipinwati Lipinwati

 [Assessment of Small Dense Low-Density Lipoprotein and Apolipoprotein B/Apolipoprotein A-I Ratio to Predict the Peripheral Arterial Disease in Patients with Hypertension](#)

 PDF

Pg. 322-327

Kurniawan Prihutomo, MID. Pramudianti, Amiroh Kurniati

 [Diagnostic Value of Determination Blast Cell Population Lineage Using WPC Scattergram Hematology Analyzer](#)

 PDF

Pg. 328-333


Nina Ratnasari, Arifoel Hajat, S. Ugroseno Yudho Bintoro

 [The Association of Parathyroid Hormone Related Protein and Vitamin D Level with Serum Calcium Ion in Acute Leukemia Patients](#)

 PDF

Pg. 334-339


Ninie Wiendyanthi, MI. Diah Pramudianti, Yuwono Hadisparto

 [Hemoglobin, Hematocrit, Leukocyte, and Platelet Changes Due To Ultrafiltrationhemodialysis in Chronic Kidney Disease Patients](#)

 PDF

Pg. 340-343


Nathalya Dwi Kartikasari, Paulus Budiono Notopuro, Widodo Widodo, Yeti Hernaningsih

 [AST, ALT and Albumin Level in Chronic Hepatitis B Patients with and without Complications of Cirrhosis and Hepatocellular Carcinoma](#)

 PDF

Pg. 344-349


Viky Nafi'ah Rahma Maulidia, Puspa Wardhani, Bagus Setyoedi

 [The Effects of the Supplementation with Snakehead Fish and Sea Cucumber Extracts on Platelet-Derived Growth Factor in Post-Operative Patients](#)

 PDF

Pg. 350-355


Otniel Wendy Wahono, JB. Suparyatmo, Dian Ariningrum

 [The Diagnostic Value of Neutrophil-Lymphocyte Ratio in Diagnosis of Autism Spectrum Disorders](#)

 PDF

Pg. 356-361


Nurmalia Purnama Sari, Yudianita K, M. Rafii Haiqal, M. Fadill Akbar

 [Serum Copeptin as the Predictor for Acute Heart Failure Complication of Acute Myocardial Infarction in Patients with ST-Segment Elevation](#)

 PDF

Pg. 362-368

Ardi Putranto Ari Supomo, JB. Suparyatmo, Dian Ariningrum



 [Correlation between CRP Level and Neutrophil Extracellular Traps in Malignancy Patients with Neutropenic Fever](#)

 PDF



Pg. 369-374



Phey Liana, Kemas M. Yakub, Eny Rahmawati, Berliana Agustin

Literature Review

 Congenital Hypothyroidism: Incidence, Etiology and Laboratory Screening	 PDF
Liong Boy Kurniawan	Pg. 375-380

Case Report

 Laboratory Diagnostic Approach and Interpretation in Gram-Negative Bacterial Infection: A-Case Report of Sepsis in Secondary Hospital Settings	 PDF
Adhi Kristianto Sugianli, Ida Parwati	Pg.

 Thyroid Crisis and Septic Suspected Sepsis in the First Trimester of Pregnancy	 PDF
Mahmudah Hidayati, Banundari Rachmawati	Pg.



Front Matter

 Cover	 PDF
	Pg.

 Contents	 PDF
	Pg.

Back Matter

 Author and Subjects Index	 PDF
	Pg.

 Author Guideline and Subscribe Form	 PDF
	Pg.

Contact Us:

Laboratorium Patologi Klinik RSUD Dr. Soetomo
 Jl. Mayjend. Prof. Dr. Moestopo 6-8 Surabaya.
 Telp/fax (031-5042113, 085733220600
 E-mail: majalah.ijcp@yahoo.com
 Website: <https://indonesianjournalofclinicalpathology.org>

ACCREDITED No. 36a/E/KPT/2016, 23 Mei 2016
OAI Address: <https://indonesianjournalofclinicalpathology.org/>

Find us on:



Indonesian Journal of Clinical Pathology and Medical Laboratory is
 licensed under a [Creative Commons Attribution-ShareAlike 4.0](https://creativecommons.org/licenses/by-sa/4.0/)
 International License.

00541820

[View My Stats](#)
[View My Old Stats](#)



INDONESIAN JOURNAL OF Clinical Pathology and Medical Laboratory

p-ISSN : 0854-4263
e-ISSN : 2477-4685

Published by
Indonesian Association of Clinical
Pathologist and Medical Laboratory

Majalah Patologi Klinik Indonesia dan Laboratorium Medik

HOME ABOUT LOGIN REGISTER SEARCH CURRENT ARCHIVES ANNOUNCEMENTS AUTHOR GUIDELINE EDITORIAL TEAM

Home > About the Journal > Editorial Team

Editorial Team

Editor-in-chief

Puspa Wardhani, Editor-in-chief Indonesian Journal of Clinical Pathology and Medical Laboratory, Indonesia, Indonesia

Editorial Boards

Ida Parwati, Department of Clinical Pathology, Faculty of Medicine, Padjajaran University, Bandung, Indonesia, Indonesia

Anak Agung Wiradewi Lestari, Department of Clinical Pathology, Faculty of Medicine, Udayana University/Sanglah Hospital, Bali, Indonesia, Indonesia

Yulia Nadar Indrasari, Department of Clinical Pathology, Faculty of Medicine, Universitas Airlangga/Dr. Soetomo Hospital, Surabaya, Indonesia, Indonesia

Nuri Diah Indrasari, Department of Clinical Pathology, Faculty of Medicine, Indonesia University/Dr. Cipto Mangunkusumo, Jakarta, Indonesia, Indonesia

Efrida Efrida, Department of Clinical Pathology, Faculty of Medicine, Andalas Univesity/M.Djamil Hospital, Padang, Indonesia, Indonesia

Puspa Wardhani, Editor-in-chief Indonesian Journal of Clinical Pathology and Medical Laboratory, Indonesia, Indonesia

Nyoman Suci Widiastuti, Department of Clinical Pathology, Faculty of Medicine, Diponegoro University/Dr. Kariadi, Semarang, Indonesia, Indonesia

Ulung Bahrun, Department of Clinical Pathology, Faculty of Medicine, Hasanuddin University/Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia, Indonesia

Budi Mulyono, Department of Clinical Pathology, Faculty of Medicine, Gajah Mada University/Dr. Sardjito Hospital, Yogyakarta, Indonesia, Indonesia

Editorial Assistant

Dian Wahyu Utami, Indonesian Journal of Clinical Pathology and Medical Laboratory, Indonesia, Indonesia

Peer-Reviewers

Tony Badrick, Royal College of Pathologists of Australia Quality Assurance Programs, Australia

Che Maraina Bt Che Hussin, Universiti Sains Malaysia, Malaysia

Zifalil Bin Alwi, Universiti Sains Malaysia, Malaysia

Wendy Erber, The University of Western Australia, Australia

Hans Vrieliink, Sanquin Blood Supply, Plesmanlaan 125, 1066 CX Amsterdam, the Netherlands, Netherlands

Ni Kadek Mulyantari, Department of Clinical Pathology, Faculty of Medicine, Udayana University/Sanglah General Hospital Bali, Indonesia, Indonesia

Adi Koesoema Aman, Department of Clinical Pathology, Faculty of Medicine, Sumatera Utara University/Adam Malik Hospital, Medan, Indonesia, Indonesia

Basti Andriyoko, Department of Clinical Pathology, Faculty of Medicine, Padjajaran University/Dr. Hasan Sadikin Hospital, Bandung, Indonesia, Indonesia

Paulus Budiono Notopuro, Department of Clinical Pathology, Faculty of Medicine, Universitas Airlangga/Dr. Soetomo Hospital, Surabaya, Indonesia, Indonesia

Tonang Dwi Ardyanto, Department of Clinical Pathology, Faculty of Medicine, Sebelas Maret University/Dr. Moewardi Hospital, Surakarta, Indonesia, Indonesia

Agustin Iskandar, Faculty of Medicine, Brawijaya University, Indonesia

Tenri Esa, Department of Clinical Pathology, Faculty of Medicine, Hasanuddin University/Dr. Wahidin Sudirohusodo, Makassar, Indonesia, Indonesia

Rachmawati Adiputri Muhiddin, Department of Clinical Pathology, Faculty of Medicine, Hasanuddin University/Dr. Wahidin Sudirohusodo, Makassar, Indonesia, Indonesia

Liong Boy Kurniawan, Department of Clinical Pathology, Faculty of Medicine, Hasanuddin University/Wahidin Sudirohusodo Hospital, Makassar, Indonesia, Indonesia

Andaru Dahesih Dewi, Department of Clinical Pathology, Faculty of Medicine, Gajah Mada University/Dr. Sardjito Hospital, Yogyakarta, Indonesia, Indonesia

Umi Solekhah Intansari, Department of Clinical Pathology, Faculty of Medicine, Gajah Mada University/Dr. Sardjito Hospital, Yogyakarta, Indonesia, Indonesia

Banundari Rachmawati, Department of Clinical Pathology, Faculty of Medicine, Diponegoro University/Dr. Kariadi Hospital, Semarang, Indonesia, Indonesia

Agnes Rengga Indrati, Department of Clinical Pathology, Faculty of Medicine, Padjajaran University/Hasan Sadikin Hospital, Bandung, Indonesia, Indonesia

Rikarni Rikarni, Department of Clinical Pathology, Faculty of Medicine, Andalas University/M.Djamil Hospital, Padang, Indonesia, Indonesia

Hani Susianti, Department of Clinical Pathology, Faculty of Medicine, Brawijaya University/Saiful Anwar Hospital, Malang, Indonesia, Indonesia

[SUBMIT](#)



[ISSN](#)

p-ISSN : 0854-4263

e-ISSN : 2477-4685

[LINK](#)

[Template](#)

[Statement Letter](#)

[Copyright Transfer Form](#)

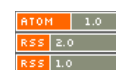
[INDEX](#)



[USER](#)

Username
Password
 Remember me

[CURRENT ISSUE](#)



[JOURNAL CONTENT](#)

Search
Search Scope
All

[Adhi Kristianto Sugianli](#), Department of Clinical Pathology, Faculty of Medicine, Padjadjaran University/Dr. Hasan Sadikin Hospital, Bandung, Indonesia, Indonesia

[Yetti Hernaningsih](#), Department of Clinical Pathology, Faculty of Medicine, Universitas Airlangga/Dr. Soetomo Hospital, Surabaya, Indonesia, Indonesia

[Yuyun Widaningsih](#), Department of Clinical Pathology, Faculty of Medicine, Hasanuddin University/Wahidin Sudirohusodo Hospital, Makassar, Indonesia, Indonesia

[Rismawati Yaswir](#), Department of Clinical Pathology, Faculty of Medicine, Andalas University/M. Djamil Hospital, Padang, Indonesia, Indonesia

[Maimun Zulhaidah Arthamin](#), Department of Clinical Pathology, Faculty of Medicine, Brawijaya University/Saiful Anwar Hospital, Malang, Indonesia, Indonesia

[Aryati Aryati](#), Department of Clinical Pathology, Faculty of Medicine, Airlangga University/Dr. Soetomo Hospital, Surabaya, Indonesia, Indonesia

[Rahajuningsih Dharma](#), Department of Clinical Pathology, Faculty of Medicine, Indonesia University/ Ciptomangunkusumo Hospital, Jakarta, Indonesia

[Mansyur Arif](#), Department of Clinical Pathology, Faculty of Medicine, Hasanuddin University/Wahidin Sudirohusodo Hospital, Makassar, Indonesia

[July Kumalawati](#), Department of Clinical Pathology, Faculty of Medicine, Indonesia University/Ciptomangunkusumo Hospital, Jakarta, Indonesia, Indonesia

[Purwanto AP](#), Department of Clinical Pathology, Faculty of Medicine, Diponegoro University/Dr. Kariadi Hospital, Semarang, Indonesia, Indonesia

[Jusak Nugraha](#), Department of Clinical Pathology, Faculty of Medicine, Universitas Airlangga/Dr. Soetomo Hospital, Surabaya, Indonesia, Indonesia

[Edi Widjayanto](#), Department of Clinical Pathology, Faculty of Medicine, Brawijaya University/Saiful Anwar Hospital, Malang, Indonesia, Indonesia

[Ninik Sukartini](#), Department of Clinical Pathology, Faculty of Medicine, Indonesia University/Dr. Ciptomangunkusumo Hospital, Jakarta, Indonesia, Indonesia

[Kusworini Handono](#), Department of Clinical Pathology, Faculty of Medicine, Brawijaya University/Dr. Saiful Anwar Hospital, Malang, Indonesia, Indonesia

[Ranti Permatasari](#), Department of Clinical Pathology, Faculty of Medicine, Sumatera Utara University/Adam Malik Hospital, Medan, Indonesia, Indonesia

[Browse](#)[By Issue](#)[By Author](#)[By Title](#)**Contact Us:**

Laboratorium Patologi Klinik RSUD Dr. Soetomo
Jl. Mayjend. Prof. Dr. Moestopo 6-8 Surabaya.
Telp/fax (031-5042113, 085733220600
E-mail: majalah.ijcp@yahoo.com
Website: <https://indonesianjournalofclinicalpathology.org>

ACCREDITED No. 36a/E/KPT/2016, 23 Mei 2016**OAI Address:** <https://indonesianjournalofclinicalpathology.org/>**Find us on:**

Indonesian Journal of Clinical Pathology and Medical Laboratory is
licensed under a [Creative Commons Attribution-ShareAlike 4.0](#)
International License.

00541817[View My Stats](#)
[View My Old Stats](#)

AST, ALT and Albumin Level in Chronic Hepatitis B Patients with and without Complications of Cirrhosis and Hepatocellular Carcinoma

Viky Nafi'ah Rahma Maulidia¹, Puspa Wardhani², Bagus Setyoboedi³

¹ Medical Student, Faculty of Medicine, Airlangga University, Surabaya, Indonesia

² Department of Clinical Pathology, Airlangga University/Dr. Soetomo Hospital in Surabaya, Indonesia. E-mail: puspa_pk@yahoo.co.id

³ Department of Pediatrics, Airlangga University/Dr. Soetomo Hospital Surabaya, Indonesia

ABSTRACT

Complications of Chronic Hepatitis B (CHB) infection are liver cirrhosis and Hepatocellular Carcinoma (HCC). Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), and albumin may be used as indicators of hepatocyte damage. This study aimed to determine differences between AST, ALT, and albumin in CHB patients without complications with CHB patients with cirrhosis and HCC complications. An analytical cross-sectional study was conducted in March-May 2019 on 62 CHB patients with or without cirrhosis or HCC complications using the total sampling method. AST and ALT were calculated using the Siemens Dimension device and International Federation of Clinical Chemistry (IFCC) method. The data were analyzed using independent samples T-test. Albumin in CHB patients without complications was higher than CHB patients who had complications of liver cirrhosis ($p=0.002$). The AST and ALT were not significantly different. Aspartate aminotransferase in CHB with cirrhosis complications differed from CHB patients who had HCC complications ($p=0.015$), however, not different in ALT and albumin. Aspartate aminotransferase, ALT, and albumin in CHB patients without complications were different from those with HCC complications. Albumin in CHB patients without complications was different from CHB patients with cirrhosis complications. Aspartate aminotransferase in CHB patients with cirrhosis complications was different from CHB patients who had HCC complications. As a result of these differences, an integrated approach to intervening liver damage may be needed to prevent the progression of the disease from becoming more severe.

Keywords: Aspartate aminotransferase, alanine aminotransferase, albumin, chronic hepatitis B, cirrhosis, hepatocellular carcinoma

INTRODUCTION

Hepatitis B Virus (HBV) infection is one of the significant health problems globally, especially Indonesia.¹ Two billion people worldwide had been infected by hepatitis B virus, and 240 million of them had Chronic Hepatitis B (CHB).² Around the world, around 650 thousand people died every year due to CHB complications. Forty-five percent of CHB patients had HCC complications, while thirty percent of CHB patients had cirrhosis complications.³ Hepatocellular carcinoma is one of the leading causes of death in males, especially in Southeast Asia. In Asia, the rates of hepatocellular carcinoma and cirrhosis are low in the age range of 35-40 years but increase with age.⁴

The prevalence of chronic HBV infection varies geographically, from high prevalence (> 8%), medium (2-7%) to low (< 2%).⁵ Hepatitis B is endemic in developing regions with large populations such as Southeast Asia, China, sub-Saharan Africa, and the Amazon Valley, where at least 8% of the population

are chronic carriers of HBV. In these areas, 70-95% of the population shows serological evidence of past or present from HBV infection. Most infections occur during infancy or childhood, most of which occur without symptoms.³

Indonesia is the second-highest endemicity country of hepatitis B in Southeast Asia after Myanmar in 2013.⁶ The data of Riskesdas 2017 said that 7.1% of Indonesia's population had hepatitis B.⁷ In 2013, around 2.8 million people in Indonesia were infected with hepatitis B and C, including the potential to become chronic and of the chronic 1.4 million had the possibility to suffer from hepatocellular carcinoma.⁶

Aspartate aminotransferase increase is more significant than ALT in CHB, and albumin levels have decreased in CHB.⁸ Liver function test is done to see the liver damage, especially liver enzyme tests. This enzyme is secreted by hepatocytes when cells are damaged. Aminotransferase is a specific indicator of hepatocyte necrosis. Aspartate aminotransferase is one of the enzymes used as indicators of liver tissue

damage by the HBV. But, AST is less specific as a marker of liver damage. Its concentration also increases in the deterioration of other body tissues such as heart, kidney, striated muscles, and brain. Furthermore, ALT is more reliable and specific than AST to indicate liver damage because its increase is produced only by hepatocytes.⁹

Serum albumin levels will decrease if there is impaired function of albumin synthesis in hepatocytes, and chronic liver cell lesions occur.^{10,11} Low albumin serum levels found in patients with severe chronic liver disease such as cirrhosis are mostly caused by decreased synthesis of albumin.⁹

Clinicians must choose non-invasive indices in the patient's serum quickly and know their interpretations to prevent the progression of HBV infection to a more severe phase, death, and transmission to others around hepatitis B sufferers. Non-invasive tests used to assess the severity of liver disease are serum biochemical parameters, such as the ratio of AST to ALT, fibrosis score-4 (FIB-4), AST to platelet ratio index (APRI), spleen-platelet ratio, index of splenic-platelet index Forns, and the Hui index.¹² Therefore, this study aimed to know the differences in AST, ALT, and albumin levels in CHB patients based on the presence or absence of cirrhosis complications and hepatocellular carcinoma complications in Dr. Soetomo Hospital, Surabaya.

METHODS

This study was a cross-sectional analytical study conducted in March-May 2019 after obtaining approval from the Dr. Soetomo Hospital Ethical Committee with the ethical clearance number of 1062/KEPK/III/2019. The samples were taken by a total sampling method. The data were obtained from medical records of CHB patients at Dr. Soetomo Hospital, Surabaya, in 2017-2018. The number of samples in this study was 62 CHB patients with details of 25 CHB patients without complications of liver cirrhosis and hepatocellular carcinoma, 25 CHB patients with complications of liver cirrhosis, and 12 CHB patients with complications of hepatocellular carcinoma. Other diseases, which might affect AST, ALT, and albumin levels were excluded such as pancreatitis which increases blood levels of AST ALT, myocardial infarction, which increases AST levels, chronic kidney disease and renal impairment, which decreases albumin levels, bile duct obstruction, liver abscess and other liver diseases that affect blood levels of ALT. Aspartate aminotransferase and ALT levels in the blood were calculated using the Siemens

Dimension device and IFCC method. Albumin levels in the blood were calculated using the Siemens Dimension device and BCG (Bromocresol Green) method.

Data of AST, ALT, and albumin level in CHB patients with or without complications of cirrhosis or HCC were compared by independent samples T-test, with a significance level of $p < 0.05$. Statistical analysis was done using SPSS 25.0 software.

RESULTS AND DISCUSSION

The samples were 62 patients in total with details of 25 CHB patients without complications and 37 CHB patients with complications (25 patients with complications of liver cirrhosis and 12 patients with complications of hepatocellular carcinoma). Age 46.40 years was the average age of CHB patients without complications; age 51.16 years was the average age of CHB patients with cirrhosis complications, and age 48 was the average age of CHB patients with HCC complications). The average AST, ALT, and albumin levels of CHB patients without complications were 60.84 IU, 42.08 IU, and 3.36 IU. The average AST, ALT, and albumin levels of CHB patients with cirrhosis complications were 125.96 IU, 88.32 IU, and 2.9 IU, respectively. The average AST, ALT, and albumin levels of CHB patients who had HCC complications were 390.16 IU, 114.16 IU, and 3.3 IU. The patients in all groups (CHB without complications, CHB with cirrhosis complications, and CHB with HCC complications) were mostly male (Table 1).

The prevalence of HBV-related liver disease was also observed to be higher in males than in females due to the mechanism of sex hormones of androgen and estrogen.¹³ The liver is considered a sexually dimorphic organ that expresses androgen receptors and estrogen receptors α .¹⁴ The liver is responsive to sex hormones for differences found in gene expression patterns, immune responses, and xenobiotic metabolism between males and females.¹⁵ HBV X protein (HBx) increases hepatic androgen receptor activity by androgen-dependent mechanisms, strengthening sex differences in liver tissue in male patients infected with HBV.¹⁶ Androgen Receptors (AR) increase HBV transcription, including all viral mRNA. Two Androgen-Responsive Elements (ARE) that are tightly bound to AR are found in the HBV genome nucleotide in enhancer I. These two ARE cause an increase in HBV mRNA levels caused by androgen receptors. Thus, AR increases the transcription of HBx. High HBx is directly proportional to the rise of ARE in the HBV genome.

Thus, the ARE in the HBV genome causes a loop of reciprocal relations between androgen receptors and HBx. ARE is the one responsible for the increment in HBV transcription regulation. Therefore, in male HBV patients, viral replication is more active, and the oncogenic potential is higher.¹³

Albumin serum levels in CHB patients without complications differed from CHB patients who experienced cirrhosis complications (p=0.002), while not different in AST (p=0.078) and ALT levels (p=0.173) (Table 2). This finding was in line with other studies conducted in China, involving 201 CHB patients. The result was that patients in the CHB with liver cirrhosis group had lower albumin levels compared to the CHB without complications group with a p-value <0.001.¹⁷

Aspartate aminotransferase and ALT are enzymes that catalyze the transfer of α-amino groups from aspartic and alanine to the α-keto ketoglutaric acid groups to produce oxalic and pyruvic acids, which are important contributors to the citric acid cycle.¹⁸ Both enzymes require pyridoxal-5'-phosphate (vitamin B6) to carry out this reaction, although the effect of pyridoxal-5'-phosphate deficiency is higher on ALT activity than on AST. Both of these enzymes are highly concentrated in the liver. Aspartate

aminotransferase is also diffuse in the heart, skeletal muscles, kidneys, brain, and red blood cells, and ALT has low concentrations in skeletal and kidney muscles.¹⁹ Therefore, an increase in ALT levels is more specific for liver damage. In the liver, ALT is localized only in the cellular cytoplasm, whereas AST is in the cytosol and mitochondria.²⁰ The third zone of liver acini has a higher AST concentration, and the damage of this zone can cause more significant changes in AST levels.²¹ Patients with advanced liver cirrhosis always have hypoalbuminemia caused by decreased albumin synthesis by hepatocytes. Water and sodium retention affect the albumin content in the extracellular space. Other factors are likely to contribute to hypoalbuminemia, an increase in the transcapillary rate.²²

Liver cirrhosis is divided into two stages, namely compensated cirrhosis and decompensated liver cirrhosis. In the asymptomatic phase of the disease, commonly referred to as compensated liver cirrhosis, patients may have a good quality of life and be undetected for several years. In the asymptomatic phase of the disease, commonly referred to as compensated liver cirrhosis, patients may have a good quality of life and be undetected for several years. Decompensated liver cirrhosis is characterized

Table 1. Characteristics of patients

Variables	CHB (n)	CHB with cirrhosis (n)	CHB with HCC (n)
Number of patients	25	25	12
Age	46.40±13.33	51.16±9.74	48.00±12.17
Gender			
Male patients	21 (84)	21 (84)	8 (66.7)
Female patients	4 (16)	4 (16)	4 (33.3)
AST	60.84±49.85	125.96±170.58	390.16±309.23
ALT	42.08±22.06	88.32±163.37	114.16±93.18
Albumin ^a	3.36±0.49	2.90±0.49	3.03±0.33

Non-parametric variables are expressed as average ± standard deviation

b Categorical variables are expressed as No. (%)

Table 2. The differences between AST, ALT, and albumin in CHB patients without complications from those who experienced cirrhosis complication

Variables	CHB without Complication x̄±SD	CHB with Cirrhosis x̄±SD	p-value*
AST	60.84 ± 49.85	125.96 ± 170.58	0.078
ALT	42.08 ± 22.06	88.32 ± 163.37	0.173
Albumin	3.36 ± 0.49	2.90 ± 0.49	0.002

Note: *Independent samples T-test

x̄ = average

SD = Standard Deviation

by clinical symptoms, including ascites, bleeding, encephalopathy, and jaundice. This disease usually develops faster towards death.²³ Once decompensated liver cirrhosis has occurred, cirrhosis becomes a systemic disease, with multi-organ or organ system dysfunction.²⁴ In compensated liver cirrhosis, AST and ALT levels tend to be normal. In decompensated liver cirrhosis, levels of AST and ALT in the blood tend to increase.²⁵

In this study, there were no differences in levels of AST and ALT in research subjects suffering from CHB without complications, and CHB patients who experienced cirrhosis complications might be the cause of several factors, such as is the phase of CHB disease and prognosis of the disease.

Aspartate aminotransferase, ALT, and albumin levels in CHB patients without complications differed from CHB patients with HCC complications with p-values of 0.004, 0.022, and 0.020, consecutively (Table 3). This finding was in line with other studies, which reported that AST and albumin levels in CHB patients without complications with CHB patients who experience HCC complications have significant differences with p-values of 0.043 and 0.001, respectively.²⁶

Hepatocellular carcinoma generally develops from chronic hepatitis or liver cirrhosis. In those two conditions, inflammation and regeneration of hepatocytes occur continuously.²⁷ Chronic liver injury can cause permanent hepatocyte damage, resulting in compensation by cell proliferation. Because of this massive compensation, fibrosis and cirrhosis develop in the process of continuous liver remodeling. There are two main mechanisms in hepatocarcinogenesis, namely cirrhosis and the presence of DNA mutations that cause an imbalance of oncogenesis from cells, which ultimately leads to neoplastic cell development.²⁸ Increased levels of the aminotransferase enzyme in hepatocellular carcinoma reflect damage to adjacent hepatocytes as a direct result of tumor growth or further damage

to liver cells caused by disruption of blood supply or venous drainage. This may also be due to ongoing liver cell necrosis in those who have concomitant active cirrhosis or chronic active hepatitis.²⁹

Tumor size affected albumin levels. In HCC patients, the higher the diameter of the tumor, the lower the albumin levels. Low albumin levels reflect damage to the liver parenchyma due to tumor growth. Decreased albumin levels are associated with a poor prognosis for hepatocellular carcinoma.³⁰ The majority of samples of CHB patients with HCC complications had low albumin levels. In this study, albumin levels of study subjects who had CHB without complications with those who experienced HCC complications appeared to be different.

Low albumin levels or high levels of AST and ALT in patients with hepatocellular carcinoma were directly proportional to high HBV DNA levels. High levels of HBV DNA caused poor prognosis of hepatocellular carcinoma patients.³¹ In this study, AST and ALT levels of study subjects who had CHB without complications with those experiencing HCC complications appeared different according to statistical tests.

Aspartate aminotransferase serum levels in CHB with cirrhosis complications differed from CHB patients who had HCC complications (p=0.015), while not different in ALT (p=0.615) and albumin levels (p=0.371) (Table 4). This finding was in line with other studies that reported that increased AST, ALT, and decreased albumin levels were associated with CHB, with cirrhosis complications.³² Another study said that 90% of people with hepatocellular carcinoma had elevated AST levels and an abnormally reduced level of albumin in the blood.³³ Elevated ALT levels were strongly associated with the incidence of hepatocellular carcinoma.³⁴ In this study, there were differences in albumin levels between CHB with cirrhosis complications patients and CHB with hepatocellular carcinoma complications patients.

Table 3. The differences between AST, ALT, and albumin in CHB patients without complications from those who had hepatocellular carcinoma complication

Variables	CHB without Complication	CHB with HCC	p-value
	$\bar{x} \pm SD$	$\bar{x} \pm SD$	
AST	60.84±49.85	390.16±309.23	0.004
ALT	42.08±22.06	114.16±93.18	0.022
Albumin	3.36±0.49	3.03±0.33	0.020

Note: *Independent samples T-test
 \bar{x} = average
 SD = Standard Deviation

Table 4. The differences between AST, ALT, and albumin in CHB patients with cirrhosis complications from those who experienced hepatocellular carcinoma complications

Variables	CHB without Complication	CHB with HCC	p-value
	$\bar{x} \pm SD$	$\bar{x} \pm SD$	
AST	125.96±170.58	390.16±309.23	0.015
ALT	88.32±163.37	114.16±93.18	0.615
Albumin	2.90±0.49	3.03±0.33	0.371

Note: *Independent samples T-test \bar{x} = average SD = Standard Deviation

CONCLUSION AND SUGGESTION

Aspartate aminotransferase, ALT, and albumin levels in CHB patients without complications were higher than those with hepatocellular carcinoma complications. Albumin levels in CHB patients without complications were more elevated than CHB patients with cirrhosis complications. Aspartate aminotransferase levels in CHB patients with cirrhosis complications were lower than CHB patients who had HCC complications. As a result of these differences, an integrated approach to intervening liver damage may be needed to prevent the disease's progression from becoming more severe.

REFERENCES

1. Perhimpunan Peneliti Hati Indonesia. Panduan tata laksana infeksi hepatitis B kronik. Jakarta, Perhimpunan Peneliti Hati Indonesia. 2006 [cited at April 15, 2018]. Available at <http://www.pphi-online.org/>
2. Khumaedi A, Gani R, Hasan I. Pencegahan transmisi vertikal hepatitis B: Fokus pada penggunaan antivirus antenatal. *Jurnal Penyakit Dalam Indonesia*, 2017; 3(4): 225.
3. Alter M. Epidemiology of hepatitis B in Europe and worldwide. *Journal of Hepatology*, 2003; 39: 64-69.
4. World Health Organization. Guidelines for the prevention, care, and treatment of persons with chronic hepatitis B infection. Geneva, World Health Organization. 2015 [cited at April 15, 2018]. Available at <https://www.who.int/hepatitis/publications/hepatitis-b-guidelines/en/>
5. Hou J, Liu Z, Gu, F. Epidemiology and prevention of hepatitis B virus infection. *International Journal of Medical Sciences*, 2005; 2(1): 50-57.
6. Pusat Data dan Informasi Kementerian Kesehatan Republik Indonesia. InfoDATIN hepatitis. Jakarta Selatan, Pusat Data dan Informasi Kementerian Kesehatan Republik Indonesia. 2014 [cited at April 15, 2018]. Available at <http://www.pusdatin.kemkes.go.id>.
7. Kementerian Kesehatan Republik Indonesia. 150 ribu orang potensial mengalami hepatitis B kronis.

Biro Komunikasi dan Pelayanan Masyarakat, Kementerian Kesehatan Republik Indonesia. 2017 [cited at April 15, 2018]. Available www.depkes.go.id/article/view/17072800006/150-ribu-orang-potensial-alami-hepatitis-kronis.html

8. Betharina N, Hendriyono FX, Mashuri. Perbedaan hasil laboratorium penderita hepatitis B dan C kronis dengan derajat fibrosis hati. *Berkala Kedokteran*, 2017; 13(1): 41-46.
9. Thapa B, Walia A. Liver function tests and their interpretation. *The Indian Journal of Pediatrics*, 2007; 74(7): 663-671.
10. Sherlock S, Dooley J. Diseases of the liver and biliary system. 11th Ed., United States of America, Blackwell Publishing, 2002; 32-34.
11. Dufour DR. Liver disease: Clinical chemistry and molecular diagnostics. 4th Ed, Missouri, Elsevier, 2006; 1777-1827.
12. Asian Pacific Association for the study of liver. Asian-Pacific clinical practice guidelines on the management of hepatitis B. *Hepatology International*, 2016; 10(1): 1-98.
13. Wang SH, Yeh SH, Lin WH, Wang HY, Chen DS, Chen PJ. Identification of androgen response elements in the enhancer I of hepatitis B virus: A mechanism for sex disparity in chronic hepatitis B. *Hepatology*, 2009; 50(5): 1392-1402.
14. Ahlbory - Dieker DL, Stride BD, Leder G, Schkoldow J, Trölenberg S, *et al.* DNA binding by estrogen receptor - alpha is essential for the transcriptional response to estrogen in the liver and the uterus. *Mol. Endocrinol*, 2009; 23(10): 1544-1555.
15. Waxman DJ, Holloway MG. Sex differences in the expression of hepatic drug metabolizing enzymes. *Mol. Pharmacol*, 2009; 76(2): 215-228.
16. Yang WJ, Chang CJ, Yeh SH, Lin WH, Wang SH, *et al.* Hepatitis B virus X protein enhances the transcriptional activity of the androgen receptor through c - Src and glycogen synthase kinase - 3 beta kinase pathways. *Hepatology*, 2009; 49(5): 1515-1524.
17. Jia W, Qi X, Ji YY, Xun YH, Wang H, *et al.* Low serum hepatitis B surface antigen level predicts compensated cirrhosis caused by chronic hepatitis B. *Hepat Mon*, 2015; 15(8): e30385.
18. Dufour DR, Lott JA, Nolte FS, Gretch DR, Koff RS, Seeff

- LB. Diagnosis and monitoring of hepatic injury. I. Performance characteristics of laboratory tests. *Clinical Chemistry*, 2000; 46(12): 2027-2049.
19. Wroblewski F. The clinical significance of alterations in transaminase activities of serum and other body fluids. *Advances in Clinical Chemistry*, 1958; 1: 313-351.
 20. Rej R. Aminotransferases in disease. *Clinical Laboratory Medicine*, 1989; 9(4): 667-687.
 21. Kamimoto Y, Horiuchi S, Tanase S, Morino Y. Plasma clearance of intravenously injected aspartate aminotransferase isozymes: Evidence for preferential uptake by sinusoidal liver cells. *Hepatology*, 1985; 5(3): 367-375.
 22. Henricksen JH, Siemssen O, Krintel JJ, Malchow-Møller A, Bendtsen F, Ring-Larsen H. Dynamics of albumin in plasma and ascites fluid in patients with cirrhosis. *Journal of Hepatology*, 2001; 16: 53-60.
 23. D'Amico G. The clinical course of cirrhosis. Population-based studies and the need of personalized medicine. *Journal of Hepatology*, 2014; 60(2): 241-242.
 24. Bernardi M, Moreau R, Angeli P, Schnabl B, Arroyo V. Mechanisms of decompensation and organ failure in cirrhosis: From peripheral arterial vasodilation to systemic inflammation hypothesis. *Journal of Hepatology*. 2015; 63(5): 1272-1284.
 25. Elsing C, Harenberg S, Stremmel W, Hermann T. Serum levels of soluble Fas, nitric oxide, and cytokines in acute decompensated cirrhotic patients. *World J Gastroenterol*, 2007; 13(3): 421-425.
 26. Chen S, Jia J, Gao Y, Li H, Fang M, *et al*. Clinical evaluation of hepatitis B core-related antigen in chronic hepatitis B and hepatocellular carcinoma patients. *International Journal of Clinical Chemistry*, 2018; 486: 237-244.
 27. Lemoine A, Saffroy R, Debuire B. Mechanisms of hepatocarcinogenesis. *Atlas Genet Cytogenet Oncol Haematol*, 2007; 11(1): 65-70.
 28. Bertino G, Di Carlo I, Ardiri A, Calvagno GS, Demma S, *et al*. Systemic therapies in hepatocellular carcinoma: Present and future. *Future Oncol*, 2013; 9(10): 1533-1548.
 29. Domingo EO, Lingao AL, Lao JY, *et al*. The significance of common laboratory tests in hepatocellular carcinoma. *Philippine Journal of Internal Medicine*, 2013; 51: 1-3.
 30. Carr B, Guerra V. Serum albumin levels in relation to tumor indis in hepatocellular carcinoma patients. *The International Journal of Biological Markers*, 2017; 32(4): 391-396.
 31. Witjes C, Ijzermans J, van der Eijk A, Hansen B, Verhoef C, *et al*. Quantitative HBV-DNA and AST are strong predictors for survival after HCC detection in chronic HBV patiens. *The Netherlands Journal of Medicine*, 2011; 54: 508-513.
 32. Merza M. Characteristics of chronic hepatitis B virus patients related liver cirrhosis in a tertiary care referral hospital, Duhok, Iraqi Kurdistan. *Journal of Gastroenterology, Pancreatology & Liver Disorders*, 2017; 4(5): 1-5.
 33. Lopez JB, Balasegaram M, Thambyrajah V, Timor J. the value of liver function test in hepatocellular carcinoma. *The Malaysian Journal of Pathology*, 1996; 18(2): 95-99.
 34. Ishiguro S, Inoue M, Tanaka Y, Mizokami M, Iwasaki M, Tsugane S. Serum aminotransferase level and the risk of hepatocellular carcinoma: A population-based cohort study in Japan, *Eur J Cancer Prev*, 2009; 18(1): 26-31.