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2016 ASCPaLM

The 14th Asian Society for Clinical Pathology and Laboratory Medicine Congress (2016)

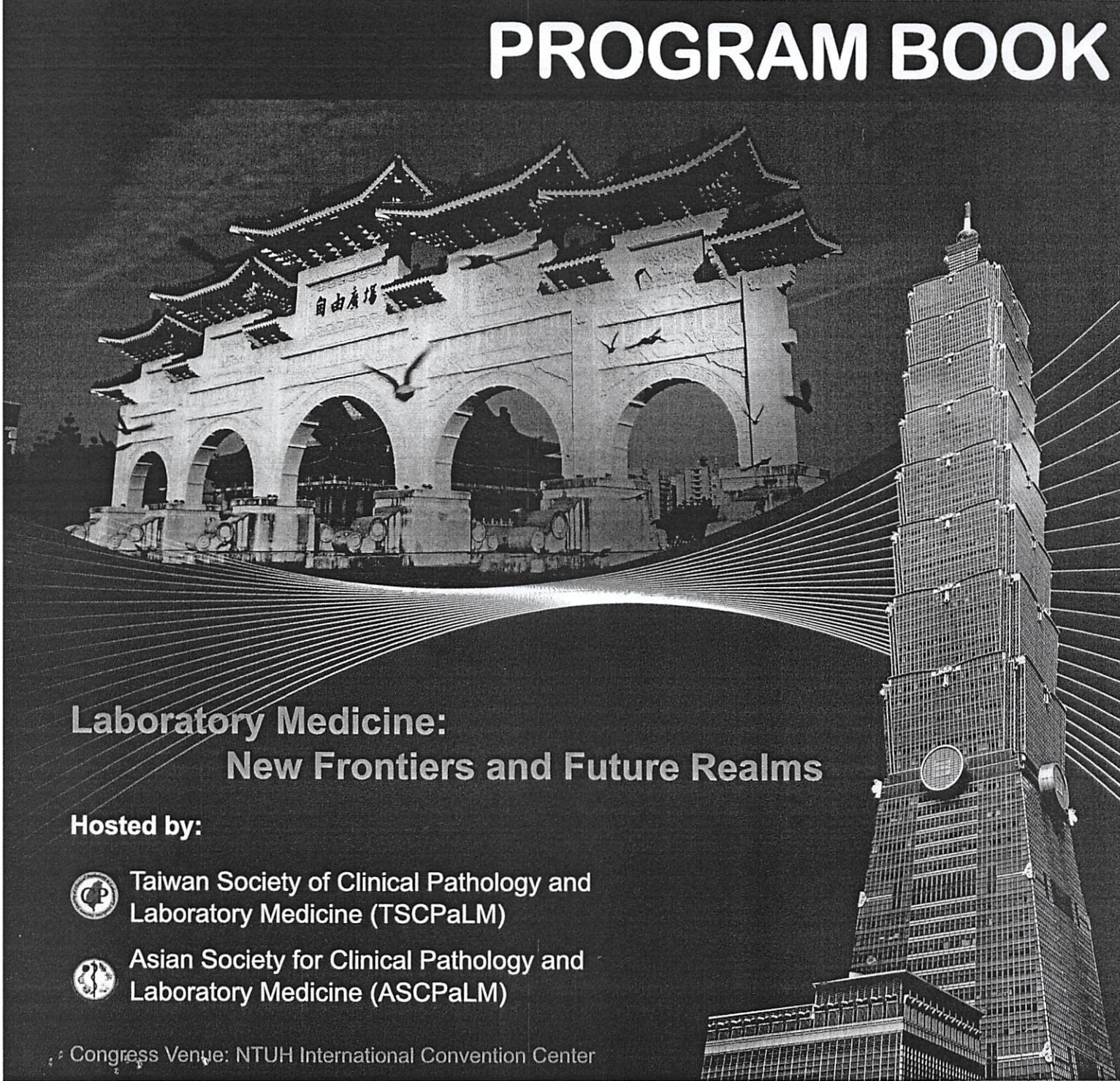
TAIPEI, TAIWAN

March 25-27, 2016

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Poster Internat

PROGRAM BOOK



**Laboratory Medicine:
New Frontiers and Future Realms**

Hosted by:



Taiwan Society of Clinical Pathology and
Laboratory Medicine (TSCPALM)



Asian Society for Clinical Pathology and
Laboratory Medicine (ASCPaLM)

Congress Venue: NTUH International Convention Center

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Welcome Message from Asian Society for Clinical Pathology and Laboratory Medicine (ASCPaLM)

On behalf of ASCPaLM, I would like to take this opportunity to express my heartfelt welcome for your participating in the 14th ASCPaLM congress which be held in the convention center of National Taiwan University Hospital on 25-27 March 2016. The previous 3rd (1994) and 7th ASCPaLM (2002) Congress were held in Taiwan as well. The activity is organized every other year in the selected sites in the rotating manner amongst the cities in Japan, Korea, Taiwan, Indonesia and Mongolia, respectively. This activity was arranged to serve all clinical pathologists in the region to join together and promote camaraderie.

Please take one minute to ponder what we have done and yet to be done, we can see a tremendous improvement in the field of clinical pathology and Laboratory medicine over the last 20 years, spanning from classical clinical pathology to molecular biology such as clustered regularly-interspaced short palindromic repeats, liquid biopsy; next generation sequencing; standardization and harmonization ; accreditation; risk management; one test for many disorders by using GC and LC mass chromatography; one-fit-for-all to precise and personal medicine.

Finally, all of you will have our best wishes during this congress and do take this occasion as the platform for sharing, caring and establishing anything with the fellow clinical pathologists from any institutions in the regions. Let's enjoy this unforgettable encounter and share the avant-garde program altogether!

I wish you have a wonderful stay in Taipei.

Best Wishes,



Tjin-Shing Jap, M.D.

President, Asian Society for Clinical Pathology and Laboratory Medicine (ASCPaLM)

<http://www.ascpalm.org/>

Welcome Message from ASCPaLM 2016

Dear Colleagues and Friends,

Welcome to Taiwan!

With 2 years of anticipation, it is our great honor and privilege to welcome your participation at the 14th Asian Society of Clinical Pathology and Laboratory Medicine Congress (2016) (the 14th ASCPaLM 2016) from March 25 to March 27, 2016 in Taipei, Taiwan.

Over the past years, ASCPaLM has rapidly grown to become one of the most important congress in the field of clinical pathology and laboratory medicine and in Taipei we expect more than 300 delegates from all over the world not only from member countries. The Program Committee brings forward an inspiring 3-day scientific program, inclusive of 4 plenary lectures, 8 keynote speeches, 8 symposia, as well as oral and poster sessions. Under the theme of "Laboratory Medicine: New Frontiers and Future Realms", our amazing lineup of speakers will share their latest research results and discuss important issues. Also, we encourage your active participation in both industry seminars and exhibitions where companies present new equipment and diagnostics for large and small laboratories. We believe ASCPaLM 2016 will be a congress not merely for learning and exchanging ideas but also for us to reconnect with old friends and build lasting friendships with new ones.

National Taiwan University Hospital International Convention Center (THCC), conveniently located at the heart of Taipei City, is well-connected to major attractions through nearby Mass Rapid Transit (MRT) stations. Please take your time to immerse yourself in amazing Taipei and we guarantee that you will bring back home with loads of memories.

Once again, we would like to express our whole-hearted gratitude to all of you for your valued participation to the continued success of this important biannual congress. Enjoy it!

Best Regards,



Jang-Jih Lu, MD, PhD

Chairman, Organizing Committee, ASCPaLM 2016



Po-Ren Hsueh, MD

Chairman, Program Committee, ASCPaLM 2016

Committees

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Chairman, Organizing Committee

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Chairman, Program Committee

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Fang-Yeh Chu

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Committee

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Invited Speakers and Moderators In alphabetical order of last name

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Chung, Wen-Hung	Taiwan	Palma, John	United States
Doljoo, Zolzaya	Mongolia	Partakusuma, Lia G	Indonesia
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Enkhsaikhan, L.	Mongolia	Ru, Kun	China
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Guan, Ming	China	Shen, Ming-Ching	Taiwan
Guo, Wei	China	Song, Junghan	South Korea
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Harper, Jacqueline	Australia	Sukartini, Ninik	Indonesia
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Hsieh, Wen-Shyang	Taiwan	Sung, Heungsup	South Korea
Hsueh, Po-Ren	Taiwan	Suryaatmadja, Marzuki	Indonesia
Hur, Mina	South Korea	Tristina, Nina	Indonesia
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Ichikawa, Daisuke	Japan	Tsai, Keh-Sung	Taiwan
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Jap, Tjin-Shing	Taiwan	Tzen, Chin-Yuan	Taiwan
Kang, So Young	South Korea	Wan, Hsiang-Lin	Taiwan
Kim, Jeong-Ho	South Korea	Wang, Cheng-Bin	China
Kim, Sollip	South Korea	Wang, Hualiang	China
Kwon, Oh Hun	South Korea	Wang, Xue-Feng	China
Lee, Li-Na	Taiwan	Wei, Cui	China
Li, Chien-Feng	Taiwan	Wu, Jiunn-Jong	Taiwan
Li, Li	China	Yamamoto, Masaru	Japan
Lin, Chih-Pei	Taiwan	Yan, Jing-Jou	Taiwan
Lin, Dong-Tsamn	Taiwan	Ying, Bin-Wu	China

Acknowledgements

The Organizing Committee would like to take this opportunity to acknowledge, with sincere appreciation, the generous contributions and support of the following sponsors and collaborating organizations:

Sponsors and Exhibitions



Singular Sponsors In alphabetical order of company name



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Collaborating Societies



General Information

Abstract USB

Please visit at least 6 exhibitors to collect 6 stamps on the coupon attached to your name badge. Once you collect them, please return your coupon at registration counter during opening hours in exchange for an abstract USB.

Badges

All participants are required to wear a badge to attend sessions or enter the exhibition area during the Congress period for recognition.

Camera and Recording

Any electronic recording and photograph taking are **NOT** allowed in all sessions.

Certificate of Attendance

Certificate of attendance will be provided with the name badge.

Congress Materials

Program book and name badge with certificate of attendance will be distributed with the congress bag.

Emergencies

For emergencies, dial 110 (police) or 119 (fire) from any phone.

Exhibition

Industrial exhibition will be open as follows:

Friday, March 25, 2016	15:00-18:30
Saturday, March 26, 2016	08:30-16:30
Sunday, March 27, 2016	08:30-15:30

Food and Beverage

Light meals are available at the Garden Café on the 1st floor of THCC on individual's own expense.

Housing

Any changes to hotel reservations should be made directly with the hotel.

Information (Lost & Found)

Information counter will be in service in the registration area and provides the relevant information on the congress and Taipei.

Any missing or unattended personal belongings will be taken to the same counter.

Internet Service

Free WiFi will be available throughout the congress venue. Search for user name **ZyXEL** with password: **77240109** and open a browser for connection.

Language

The official language of the congress is English (except Mandarin forum scheduled on March 25) . Simultaneous translation will NOT be provided.

Liability and Insurance

The ASCPaLM 2016 Organizing Committee and ASCPaLM shall not be liable for personal accidents, illness, losses or damage to private property of registered delegates of the congress. It is, therefore recommended that delegates and accompanying persons arrange appropriate travel and health insurance before traveling.

Message Board

Message board will be set up on the 1st floor for participants to exchange messages or obtain the congress updates and future meeting notice.

Mobile Phone

Switch your mobile phone to vibrate mode or switch it off in the session rooms.

Registration Counters

Onsite registration is accepted. Service hours are as follow.

Friday, March 25, 2016	08:00-18:30 (Credit card payment available from 15:00)
Saturday, March 26, 2016	07:30-17:00
Sunday, March 27, 2016	07:30-17:00

Only TWD (aka NTD: New Taiwan Dollar) cash and credit cards (VISA, Master and JCB) will be accepted for payment.

Secretariat Office/Speaker Ready Room

Secretariat Office/Speaker Ready Room (#205) will be open during service hours below. Speakers/Presenters may preview their presentation one day prior to presentation.

Friday, March 25, 2016	08:00-18:30
Saturday, March 26, 2016	07:30-17:00
Sunday, March 27, 2016	07:30-17:00

Tour

Overseas delegates, who have already purchased half-day Congress city tour, **MUST** check in at tour counter during the service hours before **12:00, Sunday, March 27, 2016** and will receive a notice for boarding information.

Venue

National Taiwan University Hospital International Convention Center (THCC)

Add : No. 2, Xuzhou Road, Zhongzheng District, Taipei City

Tel: +886-2-7724-0109

Nearby MRT stations : NTU Hospital Station (Tamsui-Xinyi Line), Shandao Temple Station (Bannan Line)

Poster Session

*Presenting authors are underlined.

Saturday, March 26, 2016

10:00-10:30

Room 101 & Room 102

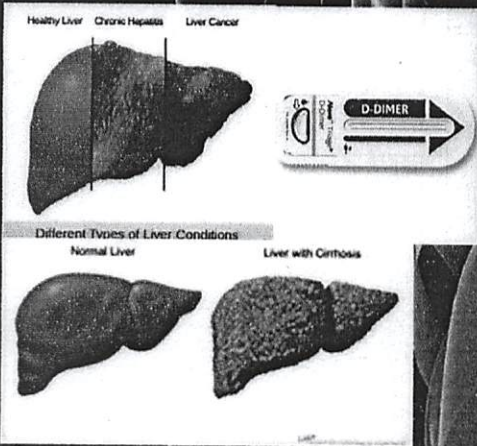
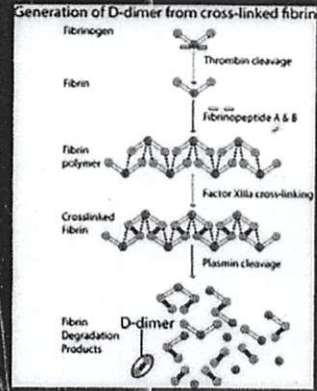
- PS-001 Molecular Basis D-dimer in Chronic Hepatitis and Liver Cirrhosis**
Indranila Samsuria (Indonesia), Purwanto Adhipireno (Indonesia), Imam Budiwiyono (Indonesia), Heniah Asti (Indonesia), Edward Kurnia (Indonesia)
- PS-002 Overexpression of *IRS2* Associated with Disease Progression in Intrahepatic Cholangiocarcinoma**
Wan-Ting Huang (Taiwan)
- PS-003 Detection of BRAF Mutation in the Cyto centrifugation Supernatant Fluid from Fine-needle Aspiration of Thyroid Lesions Enhancing the Diagnostic Yield**
Chung-Che (Jeff) Chang (Taiwan), Ashley Brown (United States), Khin Lim (United States), George Corpus (United States), Martha Hustek (United States), Anh Tran (United States)
- PS-004 Establish GeneScan to Improve the Clonality Detection Rate in T Cell Receptor Gene Rearrangement**
Wan-Li Chen (Taiwan), Yi-Lin Chen (Taiwan), Min-Ching Lin (Taiwan), Shu-Ching Yang (Taiwan), Chung-Liang Ho (Taiwan), Wenya Huang (Taiwan)
- PS-005 Competitive Allele-specific Taqman PCR (Cast-PCR) Assay for BRAF V600E Mutation Detection in Papillary Thyroid Carcinoma**
Adhitya Bayu Perdana (Indonesia), Bayu Brahma (Indonesia), Rizky Ifandriani Putri (Indonesia), Fahreza Saputra (Indonesia)
- PS-006 Down-expression of Matriptase-2 Correlates with Tumor Progression and Clinical Prognosis in Oral Squamous Cell Carcinoma**
Ming-Fang Cheng (Taiwan), Kan-Tai Hsia (Taiwan)
- PS-007 The Potential of SH3BGL3 Protein as a Prognostic Biomarker for Urothelial Carcinoma**
Nan-Haw Chow (Taiwan)
- PS-008 Multiplex Polymerase Chain Reaction Assay Developed to Diagnose Adult Bacterial Meningitis in Taiwan**
Cheng-Chuan Su (Taiwan), Chi-Tsung Lee (Taiwan), Kuang-Ming Hsiao (Taiwan), Jin-Cherng Chen (Taiwan)
- PS-009 Diagnostic Validity of Simple/Rapid Screening Test for HIV-1 Antibodies Using Human Immunodeficiency Virus Proviral DNA as Gold Standard**
Efrida Efrida (Indonesia), Andani Ekaputra (Indonesia), Zelly Dia Rofinda (Indonesia), Almurdi Almurdi (Indonesia), Dian Pertiwi (Indonesia)
- PS-010 Hepatitis C Virus Genotyping Using Next-generation Sequencing — An Efficient and Cost-effective Alternative to Sanger Sequencing in the Hospital Laboratory Setting**
Chung-Che (Jeff) Chang (Taiwan), Camoron Wales (United States), George Corpus (United States)

Molecular Basis D-Dimer Increase In Chronic Hepatitis and Liver Cirrhosis

Indranila KS, Purwanto AP, Imam BW, Herniah AW, Edward KL
 Department of Clinical Pathology Medical Faculty of Diponegoro University,
 Kariadi Hospital Semarang

Introduction:

Chronic hepatitis and liver cirrhosis is a chronic liver disease resulting in hepatic dysfunction as hemostasis. Chronic hepatitis causes liver cirrhosis complications, as hiperfibrinolisis events marked by an increase in D-dimer in the incidence molecular basis of bleeding. D-dimer in chronic hepatitis and cirrhosis examined and analyzed the difference between chronic hepatitis and cirrhosis. The research objective is to distinguish the levels of D-dimer in chronic hepatitis and cirrhosis.

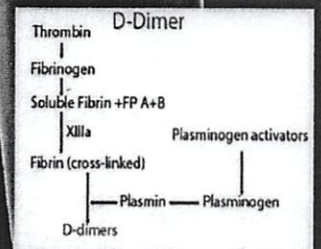


Methods:

A cross sectional study in 16 patients with chronic hepatitis and cirrhosis in dr. Kariadi Hospital, in periode March-May 2014. Levels of D-dimer used the latex enhance turbidimetric assay. Data analysis using Mann Whitney test for D-dimer in chronic hepatitis and cirrhosis.

Results:

The median D-dimer in chronic hepatitis are $190 \pm 82.30 \mu\text{g/L}$ and in the cirrhosis are $4860 \pm 57 \mu\text{g/L}$. The results of different test levels of D-dimer significantly between chronic hepatitis and cirrhosis with $p = 0.00$.



Conclusion:

There is a significant difference in the levels of D-dimer in chronic hepatitis and cirrhosis.

Keywords: Hepatitis, Cirrhosis, Bleeding, Hemostasis.

Daftar Pustaka:

1. Ricardo R, Rui TM, Miguel S. Classification and Staging of Chronic Liver Disease from Multimodal Data. IEEE Trans Biomed Eng. 2012;60(5):1336-44.
2. Park W, Keefe EB. Diagnosis and Treatment of Chronic Hepatitis B. Minervagastroenterol Dietol. 2004;50:289-303.
3. Islamuddin. Hubungan Peningkatan Kadar D-Dimer dengan Perdarahan Varises Esofagus pada Sirosis Hati Stadium Dekompensata. Padang: Universitas Andalas. 2011

Molecular basic D-dimer in chronic hepatitis and liver cirrhosis

Indranila KS, Purwanto AP, Imam BW, Herniah AW, Edward KL

Department of clinical pathology medical faculty Diponegoro University/ Dr. Kariadi Hospital Semarang

Abstract

Introduction: Chronic Hepatitis and liver cirrhosis is a chronic liver disease resulting in hepatic dysfunction as hemostasis. Chronic hepatitis causes liver cirrhosis complications as hiperfibrinolisis events marked by an increase in D-dimer in the incidence molecular basic of bleeding. D-dimer in chronic hepatitis and cirrhosis examined and, analyzed the differences. The research objective is to distinguish the levels of D-dimer in chronic hepatitis and cirrhosis.

Methods: A cross sectional study in 16 patients with chronic hepatitis and cirrhosis in hospital dr. Kariadi, in periode March-May 2014. Level of D-dimer used the latex enhance turbidimetric assay. Data analysis using Mann Whitney test for D-dimer in chronic hepatitis and cirrhosis.

Results: The median D-dimer in chronic hepatitis are 190 ± 82.30 $\mu\text{g/L}$ and in the cirrhosis are 4860 ± 57 $\mu\text{g/L}$. the results of different test levels of D-dimer significantly between chronic hepatitis and cirrhosis with $p=0.00$

Conclusions : there is a significant difference in the levels of D-dimer in chronic hepatitis and cirrhosis

Keywords : Hepatitis, cirrhosis, bleeding, hemostasis.

Background. Heart disease is a disease of the liver due to various causes within a period of 6 months. These diseases include chronic hepatitis and cirrhosis. This disease has a mortality and morbidity were significantly increased in developing countries mainly caused by hepatitis virus B and C. Initial of chronic hepatitis stage is usually asymptomatic.

Hepatitis can be cured, inactivated hepatitis can develop into chronic and is known as chronic hepatitis. Inflammation of the liver in hepatitis make liver damage and destruction of liver cells are characterized by biomarker liver function tests. Hepatitis B is an infection of hepatitis B virus (HVB), can be acute or chronic. Chronic hepatitis B infection can be detected by the presence of HBsAg positive for more than 6 months.

Infection with hepatitis C virus (HCV) can be acute or chronic, with symptoms are asymptomatic, so people do not feel sick. Hepatitis C is chronic if anti-HCV or HCV- RNA was detected positive for more than 6 months. The degree of severity of HCV infection varies among individuals, but all provide direction to the development of heart failure.

Liver cirrhosis is a chronic disease which is a chronic end-stage liver disease. Cirrhosis hepatis anatomically according to Sherlock is a fibrosis that extends to the formation of nodules in all parts of

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the liver, and fibrosis not only in one lobe. Cirrhosis is a chronic liver disease in which the damage occurred continuously, and nodular regeneration occurs, as well as the proliferation of connective tissue to prevent diffuse parenchymal necrosis or in the onset of inflammation. Any chronic condition occurs in the liver can lead to cirrhosis of the liver. approximately 80-90 percent of heart disease suffer from the damage before clinical symptoms of liver failure appeared.

Liver disease increased disease in the hole of the European Union, the researchers report in the Journal of Hepatology. WHO (world health organization) found that 170,000 deaths each year are caused by cirrhosis hepatitis. The main causes of liver disease are excessive alcohol consumption, viral infections and obesity. infection with hepatitis B and C according to Elzouki et al (2013) experienced by people aged 21-29 years and males more than females.

Cirrhosis and chronic liver disease is a common cause of death in the United States in 2002, some 27 257 deaths (9.5 per 100,000 population) dominated by men. In Asia, cases of hepatitis occurred about 9.98 million cases to about 585 800 deaths in 2011. Indonesia is in ranks third in patients with hepatitis in the world, after India and China, whose estimated number of 30 million people. Indonesia, including areas with high endemicity and in the high prevalence of more than 8%, according to WHO criteria. A total of 10 391 sera were examined and found positive HBsAg prevalence of 9.4% in 2007. Bandung is an area that have a moderate prevalence of hepatitis B virus, which is 4-5%. Number of people living in Bandung in 2010 an estimated 2 million people, meaning the prevalence of the above, there are approximately 100,000 people with HBsAg.

WHO estimates there are 54,000 deaths and 955,000 disability connecting it with a long life associated with acute hepatitis C virus infection. HVC infection becomes chronic infection, 3-4 million people have HVC infections each year, 170 million people are chronically infected and develop into chronic liver diseases, cirrhosis and liver cancer. While 350,000 people die every year because of this HVC.

Liver is an important organ in the primary and secondary hemostasis. Liver damage associated with coagulation disorder that worsens as the heart damage. Liver failure on chronic liver disease resulting in an increase fibrinolysis. Hiperfibrinolysis in cirrhosis of the liver is indicated by elevated levels of tissue plasminogen activator (tPA) and plasminogen activator inhibitor-1 (PAI-1), and decreased levels of plasminogen, antiplasmin, and factor XIII. tPA levels increase due to increased acquisition by the endothelium due to reduced clearance of the liver. PAI-1 levels increased, but not as high levels of tPA. This situation resulted in an increasing degradation products of fibrinogen and D-dimer.

D-dimer plasma level is an accurate sign of fibrinolysis activity, which indicates the activity of plasmin and thrombin. D-dimer test is also used to determine the diagnosis Disseminated intravascular coagulation (DIC) in chronic liver disease, especially patients with liver cirrhosis. Anticipated increase in fibrinolysis (hiperfibrinolysis) resulting in fatal bleeding incidents.

The theory above is in accordance with the results of Islamuddin (2011) found elevated levels of D-dimer associated with the occurrence of bleeding esophagus in psien hepatic cirrhosis. Dhanumjaya et al (2013) writes that peningkatana fibrinolytic activity becomes an important factor responsible for the tendency of bleeding in liver disease.

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D- dimer become an important parameter to assess the status of fibrinolysis in chronic liver disease, Li (2011) wrote that the D-dimer can be used as effective indicators at different degrees of liver disease. Pan et al (2006) wrote that the levels of D-dimer in hepatic cirrhosis in a significant rise higher than the chronic hepatitis.

Measurement of levels of D-dimer mostly performed on patients cirrhosis of the liver, and still little is done in patients with chronic hepatitis. D-dimer difference to both diseases are not much discussed in most studies. The usefulness of D-dimer examination theoretically been known chronic liver disease, good to see the risk of bleeding and DIC. This study will measure the difference of D-dimer in patients with chronic hepatitis and cirrhosis of the liver, so it can be differences in the levels of D-dimer in both these circumstances.

The research question : is there a difference between the levels of D-dimer chronic hepatitis with cirrhosis of the liver? The aim of the research objectives are: to analyze the differences between the levels of D-dimer chronic hepatitis and cirrhosis of the liver.

D-dimer levels are parameters that have been widely studied in hepatic cirrhosis, among others, to look at the incidence of bleeding and assessment of disease progression. Both the D-dimer in chronic hepatitis is still hard to find in previous studies that the research needs to be done about it.

Our studies interested in conducting research on the D-dimer in chronic hepatitis compared to cirrhosis of the liver. Chronic hepatitis taken on this research that chronic liver inflammation caused by infection with hepatitis B and C, which is different from the research that has been done had dedicated chronic hepatitis due to hepatitis B virus infection. Cirrhosis of the liver is taken from this research is that only patients suffering liver failure with a history of viral infections hepatitis B and C.

Hepatitis

Chronic hepatitis is a liver disease histologically patterned as necrosis, inflammation and fibrosis of hepatocytes in various weight levels, light for more than 6 months. The most common cause of chronic hepatitis is viral infection. Hepatitis virus infection plays a role in heart most is the hepatitis virus B (HBV) and C (HCV). Chronic persistent hepatitis have histopathologic features are localized inflammatory infiltrasi, and the border area between cells portal. Chronic lobular hepatitis have histopathologic virus features are accompanied by portal inflammatory focal necrosis and inflammation in the liver lobular that resembles acute hepatitis improved. chronic active hepatitis have histopathologic there is erosion in periportal hepatocytes by inflammatory cells (necrosis metal piece or interface hepatitis), usually accompanied periportal connective tissue that extends into the heart lobular. As seen in table 1.

Table 1. Classification of chronic hepatitis

classification	contemporary classification	
	levels (activity)	stage (fibrosis)
chronic persistent hepatitis;	minimal or mild	no or mild

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Chronic lobular hepatitis	mild or moderate	light
chronic active hepatitis	mild, moderate, severe	mild, moderate, severe

D-dimer

D-dimer is formed through crosslinking of factor XIII and fibrin monomer hydrolysis by plasmin and is a marker for early diagnosis of thrombosis, as well as an indicator of abnormal coagulation and fibrinolysis. D-dimer concentration will increase with impaired hepatic function.

In the process of abnormal clot formation, a fibrin clot formed at the last stage of the coagulation process. Fibrin generated by the activity of thrombin that breaks fibrinogen into fibrin monomers. Fibrinogen is a glycoprotein with a formula $A\alpha, B\beta, \gamma$. Consists of three pairs of polypeptide chains are not identical and mutually plait namely 2 chain $A\alpha$, 2 $B\beta$, and 2 γ . Fibrinogen molecule is bound dimeric by disulfide bond at the terminal end. Couple chain $A\alpha$ and $B\beta$ chains have fibrinopolipeptida a small one, at the terminal called fibrinopolipeptida A and B.

Process of change fibrinogen into fibrin consists of three phases: Enzymatic, polymerization and stabilization. At the stage of enzymatic, 2 molecules of fibrinopeptide A and 2 molecules of fibrinopeptide B are broken down and fibrinogen is converted by thrombin into fibrin monomer soluble. Phase polymerization, fibrinopolipeptida A removable which will cause the aggregation side to side, followed by the release of fibrinopeptide B into contact with monomer units with more powerful and forming clots unstable. The next stage is the stabilization, in which the addition of thrombin, factor XIII A and calcium ion (Ca^{2+}) to form insoluble stable fibrin. Thrombin causes the activation factor XIII which acted as transamidase. Factor XIIIa causes cross-linked of fibrin monomer which adjacent to form stable covalent bonds (fibrin Mesh). Chains α and γ plays a role in the formation of a stable fibrin insoluble. the flow of cross-linked fibrin formation can be seen in Fig.1

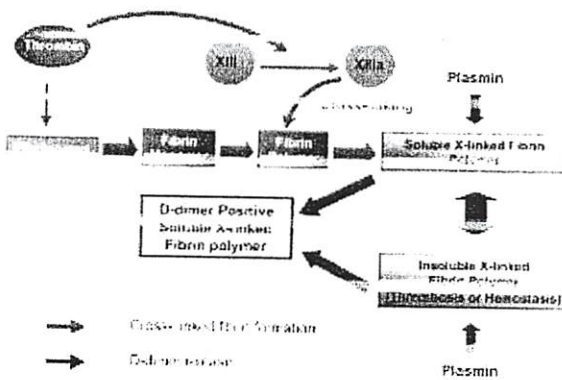


Figure 1. D- Dimer Formation by Soheir, et al

Plasminogen is normally present in the plasma will be absorbed by fibrin. When in fibrin, plasminogen is converted by tissue plasminogen activator (tPA) into plasmin derived from tPA-plasminogen complex -

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fibrin. Plasmin is fibrinolytic enzyme is the main function breaks down fibrinogen and fibrin which produce a variety of products degeneration fibrinogen (fibrin degradation products). If plasmin lyse fibrin insoluble, it will increase the amount of soluble fibrin degradation products. Fibrin degradation product (FDP) that is produced in the form of fragments X, Y, D, and E. Two fragments D and one fragmen E of the fragment binds strongly affecting the D-dimer. The dynamics of the formation of D-dimer derived from fibrin can be seen in Figures 1 and D- dimer formation scheme shown in Figure 2.

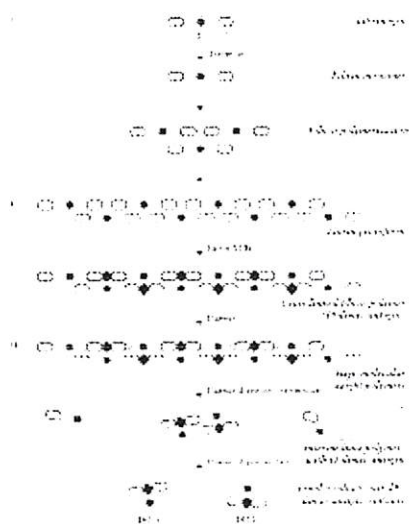


Figure 2. schematic d- dimer by Soheir et al

D-dimer examination principle is to use monoclonal antibodies that recognize epitopes on the D-dimer fragment. There are several methods of inspection are enzyme linked immunosorbent assay (ELISA), latex agglutination (LA) and whole blood agglutination (WBA).

Latex agglutination method used in this study using the antibody coated on latex particles. Agglutination macroscopically visible if there is an increase in D-dimer in plasma. This method is less sensitive to the screening, the test is not expensive but easy to do, but in some studies indicate that this method has less sensitivity to mendeteksi D-dimer in pulmonary embolism and acute venous thrombosis.

the method has a sensitivity in the range of 80-100% and a negative predictive value of 90% depending on the researcher. Latex agglutination modified by using automatic analysis can be used to measure the quantitative D-dimer. for example latex enhanced turbidimetric test. The principle of this method is the formation of covalent bonds polystyrene particles on a monoclonal antibody against cross-linkage region daru D-dimer. Cross-linkage has a structure of stereometrik. Agglutination reaction that occurs detected using turbidimetry. This method results comparable to conventional ELISA.

Methods study : The design of this research is descriptive analytic cross sectional approach. the scope of the research was conducted in a poly medicine and inpatient ward dr. Kariadi Semarang and examination of serum levels of D-dimer in Laboratory Installation. RS. dr. Kariadi. Research time of examination of samples up to the presentation of the results is in March and May 2014. The disciplines studied are clinical pathology and subpart hepatology and hematologi. Population research targets are patients who come to the clinic in internal medicine dr. Kariadi Semarang. Population is affordable chronic and patients with liver disease, cirrhosis of the liver with a history of chronic hepatitis who

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come to the clinic medicine and hospitalization in internal medicine hospital dr. Kariadi Semarang. The subject research is conducted done by purposive sampling to meet the inclusion and exclusion criteria. A cross sectional study in 16 patients with chronic hepatitis and cirrhosis in hospital dr. Kariadi . Level of D-dimer used the latex enhance turbidimetric assay. Data analysis using Mann Whitney test for D-dimer in chronic hepatitis and cirrhosis.

Inclusion criteria were patients aged ≥ 21 years, not using drugs that cause coagulation disorders such as aspirin, heparin or warfarin. Not using contraception, not pregnant, and without a history of malignancy of the liver or other organs. Without a history of coronary heart disease or being exposed to the disease, with no history of stroke or being exposed to the disease, do not have an infection, do not experience joint disease, no history of autoimmune disease or being exposed to the disease. willing to participate in research. Exclusion criteria: lipemic sample and hemolysis.

Materials and research reagents composed of D-dimer reagents innovance, D- dimer reagents accelerator, and D-dimer innovance reconstitution medium. Examination of the workings of D-dimer: 1) there is no special preparation for the examination of D-dimer. Principle probes D-dimer is a polystyrene particle formation of covalent bonds on a monoclonal antibody against cross- linkage of D-dimer. 2) The specimen used is blood plasma with the anticoagulant sodium citras 3.2% .3) Put all the reagents, standards of work, and spesimen.4) Blood homogenized, centrifuged at 3000 rpm for 5 minutte .5) Supernatant was taken and stored temperature $< - 20^{\circ}$ C is stable until 2 months , while at room temperature can be stable till 8 hours. 6) The levels of D-dimer is checked using the tools and reagents from coagulometer Sysmex CA-1500 with latex Enhance turbidimetric test method. 7) D-dimer normal value of 0-500 $\mu\text{g} / \text{L}$.

Data collected included interviews, physical examinations and laboratory tests. Collected data is done by editing, coding, and entered into a computer programme. Data D- dimer in chronic hepatitis and cirrhosis Mann Whitney test and significance declared at $p < 0.05$. Across the studies that met the inclusion and exclusion criteria, requested approval of informed consent. Permit research done by asking ethical clearance from the ethics committee of health research Diponegoro University School of Medicine / dr. Kariadi Semarang No. 070 / EC / FK-RSDK / 2014

Results:

Research conducted on 32 patients consisted of 16 patients with chronic hepatitis and 16 patients with cirrhosis of the liver. the control patients in hospitals and hospitalization. patient characteristics are shown in Table 2.

Table 2. patient characteristics

patient characteristics	variable	
	chronic hepatitis	cirrhosis Hepatis
age (years)		
median \pm SE	40,50 \pm 3,30	51,50 \pm 2,29
min-max value	22-67	35-62

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long suffered from hepatitis (month),		
min-max value	7-252	108-168
long-suffering liver cirrhosi (months)		
min-max value	-	3-72

The median D-dimer in chronic hepatitis are $190 \pm 82.30 \mu\text{g/L}$ and in the cirrhosis are $4860 \pm 57 \mu\text{g/L}$. The results of different test levels of D-dimer significantly between chronic hepatitis and cirrhosis with $p=0.00$

Conclusions : there is a significant difference in the levels of D-dimer in chronic hepatitis and cirrhosis

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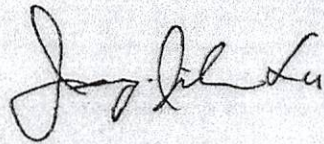
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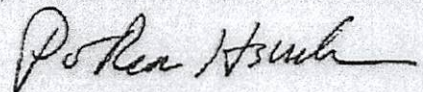
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