View this email in your browser

Liver Digest

A weekly update of PLRC happenings

January 9, 2020

PITTSBURGH LIVER RESEARCH CENTER
A partnership of University of Pittsburgh & UPMC

www.livercenter.pitt.edu

Featured Faculty - Dr. Abhinav Humar

In this issue

PLRC Seminar

PLRC Seminar Series

Tue, 1/28/2020

12:00 to 1:00 pm

S123 BST

Suthat Liangpunsakul, MD (click on name to go to bio page)

Professor of Medicine

Professor of Biochemistry and Microbiology

Indiana University School of Medicine

Alterations in circadian machinery and ω-oxidation of fatty acids in the pathogenesis of alcohol-induced liver injury

Pizza will be provided.

This activity has been approved for AMA PRA Category 1 credit #6242-Liver Center Seminars.

The full schedule of Enrichment activities is posted on **https://www.livercenter.pitt.edu/events**.

Faculty Highlights

PLRC members collaborating on manuscripts are noted in red.

Case Report:

Gluskin AB, Dueker JM, El Hag M, Puthenpurayil KJ, **Bataller R**. Alcoholic hepatitis masquerading as tumor infiltration: Reversibility after abstinence. Clin Case Rep. 2019 Sep 30;7(11):2174-2176. doi: 10.1002/ccr3.2448. eCollection 2019 Nov. PMID: 31788273

ABSTRACT

A subset of patients with alcoholic hepatitis present with atypical imaging resembling hepatic tumor infiltration. Our case involves a patient who was initially thought to have multiple large hepatic metastases, ultimately found to have alcoholic hepatitis. It is essential to ask about alcohol use when clinical suspicion is high.

For full text, please **click here**.

Invited Commentary:

Satdarshan P. Monga. No Zones Left Behind: Democratic Hepatocytes Contribute to Liver Homeostasis and Repair. Cell Stem Cell; Volume 26, Issue 1, 2 January 2020, Pages 2-3.

ABSTRACT

Despite minimal turnover, liver cells possess immense regenerative capacity. Some studies suggest existence of a hepatocyte subset with such unique capabilities. However, in the current issue of Cell Stem Cell, three independent studies (Chen et al., 2020, Matsumoto et al., 2020, and Sun et al., 2020) demonstrate an equitable homeostatic and reparative potential of all hepatocytes, irrespective of their lobular location or ploidy status.

For full text, please **click here**.

Original Article:

Cannella R, Minervini MI, **Rachakonda V**, Bollino G, **Furlan A**. Liver stiffness measurement in patients with nodular regenerative hyperplasia undergoing magnetic resonance elastography. Abdom Radiol (NY). 2019 Dec 13. doi: 10.1007/s00261-019-02367-8. PubMed PMID: 31834457.

ABSTRACT

PURPOSE: Nodular regenerative hyperplasia (NRH) may mimic cirrhosis at imaging. We aim to investigate the effect of NRH on liver stiffness measurement (LSM) obtained with magnetic resonance elastography (MRE).

METHODS: This retrospective, Institutional Review Board-approved study included 37 subjects with NRH (Group 1) and no or minimal fibrosis (F0-F1), a control group (Group 2) made of 30 subjects with non-advanced fibrosis (F0-F2), and a control group (Group 3) made of 30 subjects with advanced fibrosis (F3-F4), all with available MRE. LSM was measured in each subject along with assessment of hepatic morphological features of cirrhosis and signs of portal hypertension. The significance of the difference in mean LSM between Group 1 and 2 and between Group 1 and 3 was evaluated using the Mann-Whitney U test. The difference in distribution of imaging features among groups was assessed using the Pearson $\chi 2$ or Fisher exact test.

RESULTS: The mean ± SD LSM in Group 1 (3.56 ± 1.10 kPa) was significantly higher compared to Group 2 (2.91 ± 0.52 kPa, $P = 0.019$) and significantly lower compared to Group 3 (7.18 ± 2.08 kPa, $P < 0.001$). Twelve (32%) patients with NRH had LSM ≥ 4.11 kPa, and 6 (16%) patients had LSM ≥ 4.71 kPa. Surface nodularity ($P = 0.032$) and caudate lobe hypertrophy ($P = 0.004$) were more commonly visualized

in Group 1 than in Group 2. At least one feature of portal hypertension was observed in 16 (43%) NRH subjects.

CONCLUSION: NRH may increase the LSM obtained with MRE and may represent a confounding factor when using liver stiffness for the non-invasive diagnosis of fibrosis.

For full text, please **click here**.

Brief Report:
Butt AA, Yan P, Aslam S, Abou-Samra AB, Sherman KE, **Shaikh OS**. Liver Fibrosis Progression and Mortality in Hepatitis B- and C-Coinfected Persons Treated With Directly Acting Antiviral Agents: Results From ERCHIVES. Clin Infect Dis. 2019 Dec 16. pii: ciz1097. doi: 10.1093/cid/ciz1097. PubMed PMID: 31840746.

ABSTRACT
For persons with baseline Fibrosis-4 1.46-3.25, cirrhosis incidence/1000 patient-years was 49.3 among hepatitis B virus (HBV)/hepatitis C virus (HCV) coinfected and 18.2 among HCV monoinfected (P = .03). Cirrhosis risk was numerically higher but statistically nonsignificant among HBV/HCV coinfected (hazards ratio [HR] 1.51; 95% confidence intervals [CI], .37-6.05) but lower among those who attained sustained virologic response (HR, .52; 95% CI, .42-.63).

For full text, please **click here**.

Review Article:
Christine E. Dolin, **Gavin E. Arteel**. The Matrisome, Inflammation, and Liver Disease. Semin Liver Dis. 2020 Jan 7. doi: 10.1055/s-

0039-3402516. [Epub ahead of print] PubMed PMID: 31910448.

ABSTRACT
Chronic fatty liver disease is common worldwide. This disease is a spectrum of disease states, ranging from simple steatosis (fat accumulation) to inflammation, and eventually to fibrosis and cirrhosis if untreated. The fibrotic stage of chronic liver disease is primarily characterized by robust accumulation of extracellular matrix (ECM) proteins (collagens) that ultimately impairs the function of the organ. The role of the ECM in early stages of chronic liver disease is less well-understood, but recent research has demonstrated that several changes in the hepatic ECM in prefibrotic liver disease are not only present but may also contribute to disease progression. The purpose of this review is to summarize the established and proposed changes to the hepatic ECM that may contribute to inflammation during earlier stages of disease development, and to discuss potential mechanisms by which these changes may mediate the progression of the disease.

For full text, please **click here**.

Funding Opportunity

Research Grants

PSC Partners Seeking a Cure

Our mailing address is:
Pittsburgh Liver Research Center
200 Lothrop St. | Pittsburgh, PA 15261