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Direct cost analysis of rapid MRI in the emergency department evaluation of patients suspected of having acute ischemic stroke

Adam de Havenon, Chris Orlando, Alen Delic, more...
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Article information

Results

We included 408 brain attack activations (mean ± SD age 62.1 ± 17.6 years, 47.8% male): 257 in the CTP cohort and 151 in the MRI cohort. Discharge diagnosis was ischemic stroke in 193/408 (47.3%). After patient matching, we found significant reductions for the MRI cohort in total cost (−18.7%, 95% CI −35.0, −2.4, p = 0.02) and hospital LOS (−17.0%, 95% CI −31.2, −2.8, p = 0.02), with no difference in ED LOS (p = 0.74) as compared to the CTP cohort.

Conclusion

Although these results are preliminary and hypothesis-generating, we found that the use of a rapid MRI protocol in emergency department brain attacks was associated with a 18.7% reduction in total direct cost and 17% reduction in hospital length of stay.

Effect of short MRI protocol on stroke diagnosis in the ED

Measure	CT perfusion cohort	Short MRI cohort	p-value
Hospital length of stay in days	3.5	1.9	<0.001
ED length of stay in minutes	206.2	234	0.026

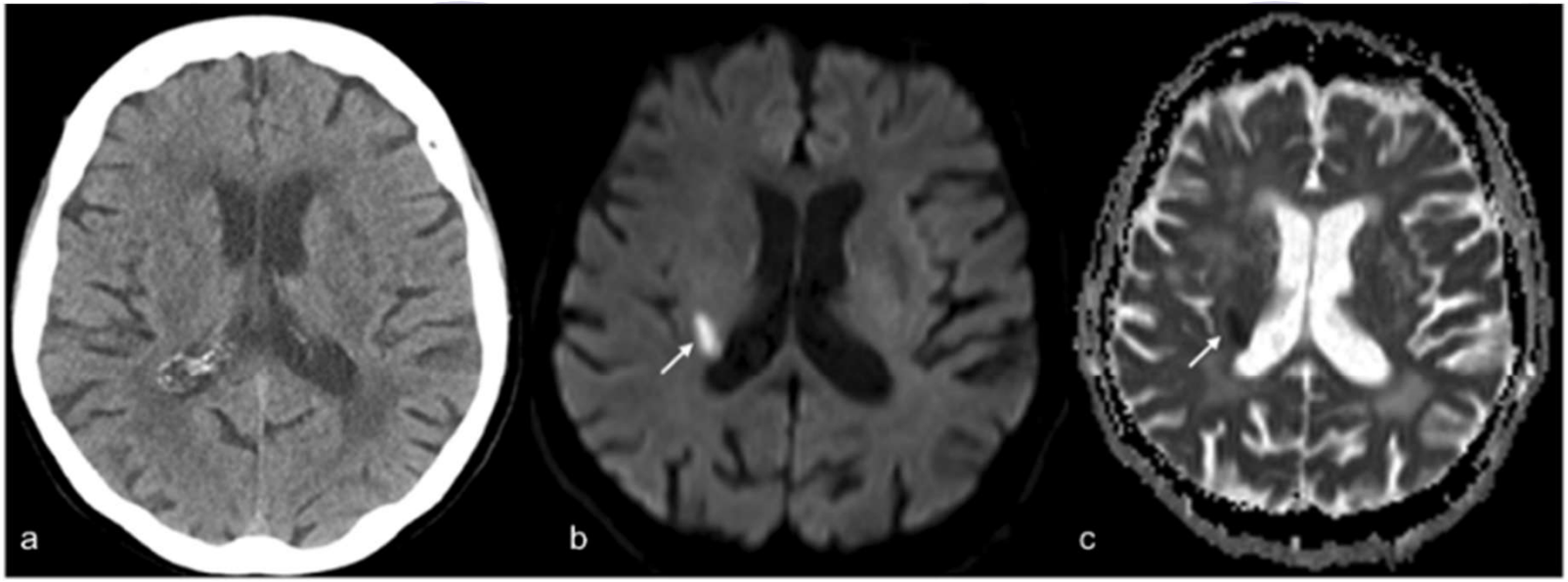

Fig. 1 Representative CT-occult minor stroke in a patient presenting with transient left-sided mild hemiparesis. Non-contrast CT (**a**) did not show any signs of ischemic stroke. Diffusion-weighted imaging (**b**, b1000) and the corresponding apparent diffusion coefficient map (ADC map, **c**) from the short MRI protocol revealed an ischemic lesion in the posterior limb of the right internal capsule (arrow)

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Quantification of Hepatic Steatosis by Ultrasound: Prospective Comparison to MRI Proton-Density Fat Fraction as Reference Standard

Jonathan R. Dillman, MD, MSc[1,2], Samjhana Thapaliya, MD[1], Jean A. Tkach, PhD[1,2] andAndrew T. Trout, MD[123]

ABSTRACT :

Background: Multiple ultrasound platforms now provide quantitative measures of hepatic steatosis. One such measure is the ultrasound-derived fat fraction (UDFF), which combines attenuation and backscatter quantification.

Objective: To characterize agreement between UDFF and MRI proton-density fat fraction (PDFF) measurements.

Methods: This prospective cross-sectional study enrolled 56 overweight and obese adolescents and adults (≥16 years) who underwent investigational ultrasound (ACUSON Sequoia; deep abdominal transducer) and MRI examinations of the liver during a single visit from August 2020 to October 2020. Ultrasound examinations included three UDFF acquisitions of five measurements each (15 measurements total), and an overall median of medians was computed (UDFFoverall). MRI examinations included three PDFF acquisitions with calculation of an overall median PDFF. Spearman rank-order correlation was computed between UDFF and MRI PDFF measurements. Intraclass correlation coefficients and Bland-Altman difference plots were used to assess agreement. ROC curves were used to assess diagnostic performance of UDFF for detecting MRI PDFF ≥5.5%.

Results: Median participant age was 32.5 years (interquartile range [IQR]: 24-39 years); 40 participants were female, 16 were male. A total of 34 (60.7%) participants had MRI PDFF ≥5.5%. UDFFoverall was 10.5% (IQR, 5.0-20.0%); median MRI PDFF was 6.1% (IQR, 3.4-13.7%). UDFFoverall was positively associated with MRI PDFF [rho=0.82; p<.001; ICC, 0.84 (95% CI: 0.59-0.93)]. Mean bias between UDFF and PDFF was 4.0% (95% limits of agreement: -7.9, 15.9%) with similar bias if summarizing UDFF by the first 5 (4.4%), 3 (4.4%), or 1 (4.6%) measurements. UDFF had AUC of 0.90 (95% CI: 0.79-0.96) for MRI PDFF ≥5.5%; AUC was not significantly different based on the number of UDFF measurements (p=.11-.97 for all pairwise AUC comparisons). UDFFoverall cutoff of >5% had sensitivity of 94.1% and specificity of 63.6% for diagnosing MRI PDFF ≥5.5%.

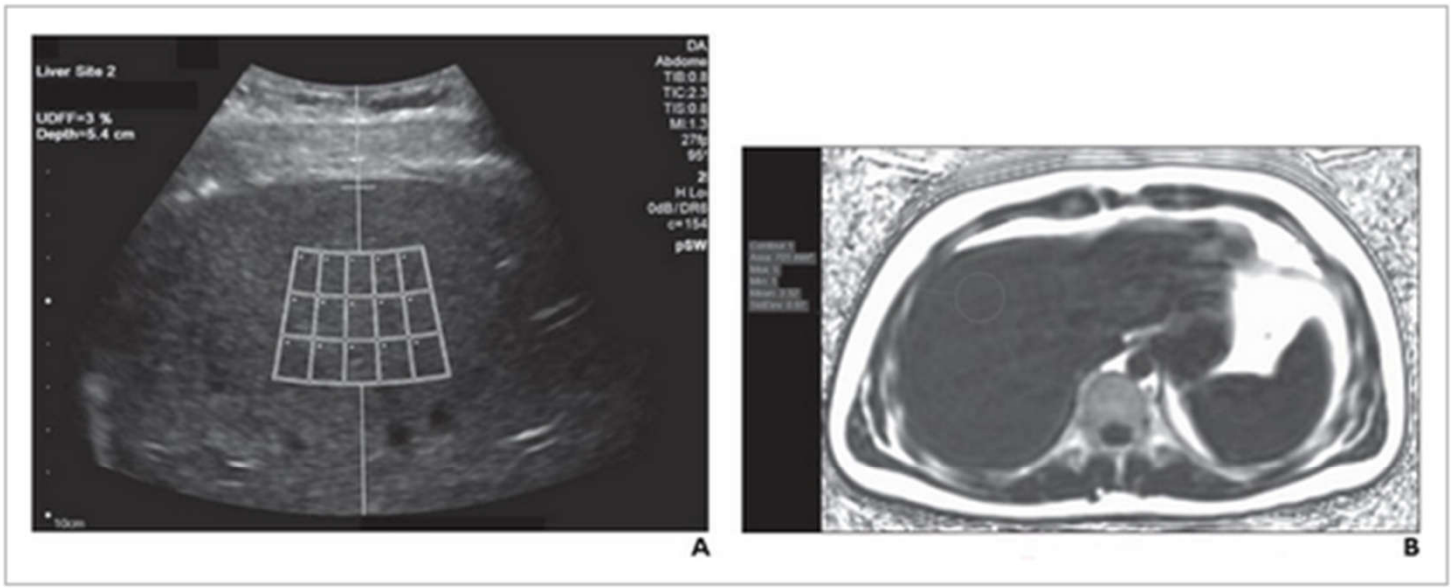
Conclusion: Measurements of hepatic steatosis using UDFF show strong agreement with measurements by MRI PDFF. A UDFF cutoff of >5% provides high AUC and sensitivity, albeit low specificity, for detection of MRI PDFF ≥5.5%.

Clinical Impact: UDFF may have a clinical role in detection of hepatic steatosis. A reduced number of individual measurements is likely sufficient for determining an overall UDFF value.

https://www.eurekalert.org/multimedia/937163 90%

23-Year-Old Man with Body Mass Index of 25.1 (IMAGE)

AMERICAN ROENTGEN RAY SOCIETY



CAPTION

Left: UDFF—operator places crossbar at liver capsule, with sample ROI fixed 1.5 cm deep from crossbar, to ensure measurement obtained sufficiently deep to liver capsule. Overall UDFF was 3%. Right: Median MRI PDFF from three acquisitions was 3%, demonstrating agreement.

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VIRAL HEPATITIS IN 2021

Vaccination at the forefront of the fight against hepatitis B and C

Manal H. El-Sayed and Jordan J. Feld

Vaccination is a key intervention for the elimination of hepatitis B virus (HBV) and hepatitis C virus (HCV) infections to fulfil the WHO's 2030 global elimination goal. Innovations in 2021 promise to curb HBV transmission by reducing mother-to-child transmission and enhancing vaccine immunogenicity in at-risk adult groups. Additionally, an HCV vaccination trial was conducted, and there were also advances in our understanding of the immunology underpinning the lack of protection against HCV reinfection.

Key advances

- In the Democratic Republic of the Congo, combining hepatitis B virus (HBV) screening and treatment for pregnant women, as well as infant birth-dose immunization, with existing platforms for prevention of mother-to-child transmission of HIV, has proven practical and been widely accepted[3].
- A multicentre, randomized controlled trial demonstrated that the tri-antigenic HBV vaccine elicited a robust immune response in people aged >45 years, including those with chronic disease and an impaired immune response[4].
- A phase I/II randomized trial evaluated two consecutive hepatitis C virus (HCV) vaccines in adults with a history of injection drug use; the vaccine regimen produced HCV-specific T cell responses but did not prevent chronic HCV infection[7].
- Transcriptomic data from a cohort treated with direct-acting antivirals revealed a molecular signature of T cell exhaustion in HCV-specific $CD8^+$ T cells that remained as a chronic 'scar' even after chronic antigen stimulation was stopped[10].

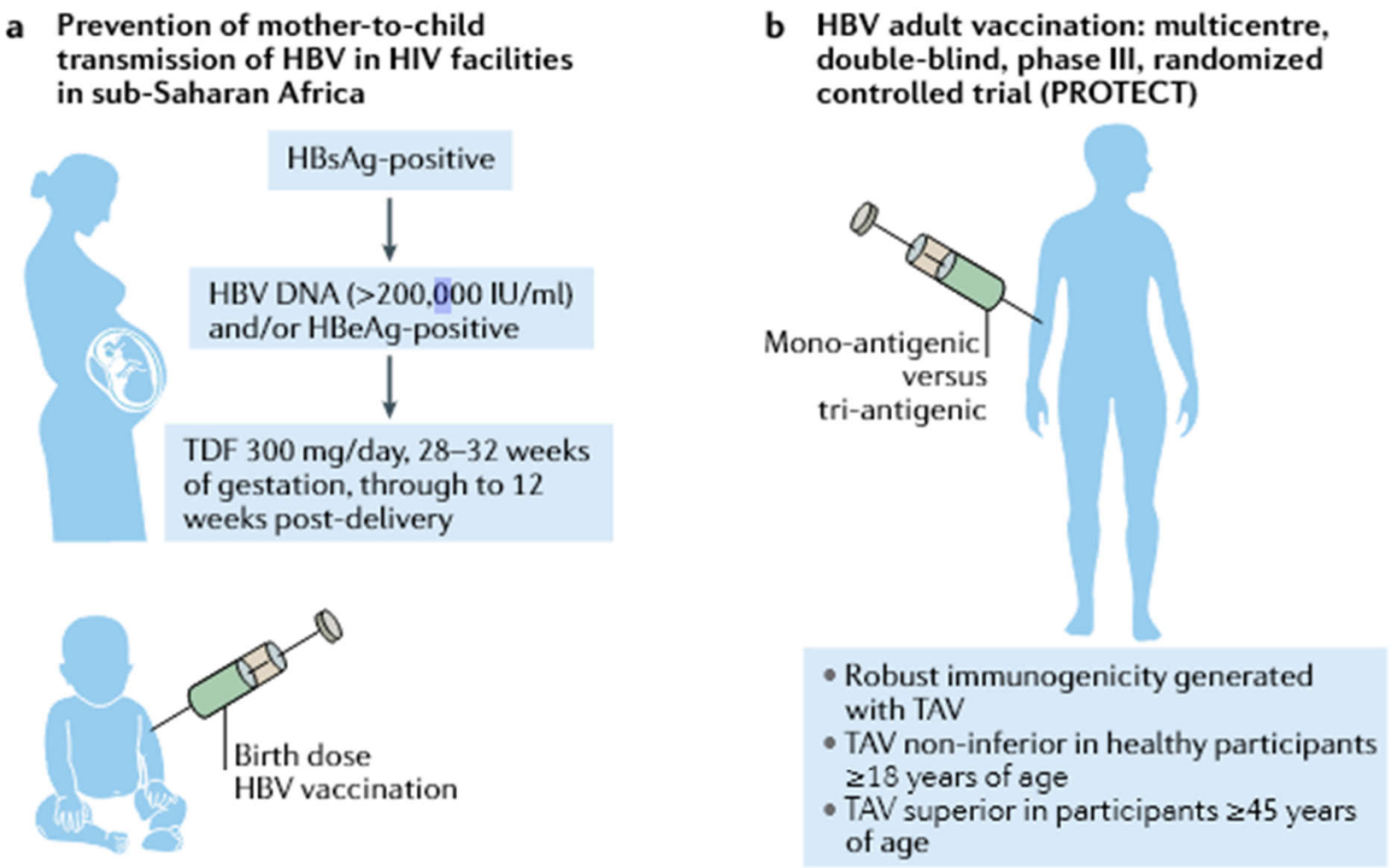

Fig. 1 | **Advances in HBV vaccination in 2021. a** | Integrating hepatitis B virus (HBV) screening and treatment for pregnant women, as well as birth-dose vaccination, into existing facilities for prevention of mother-to-child transmission of HIV[3]. **b** | A multicentre, randomized controlled trial (PROTECT) of TAV (a tri-antigenic recombinant HBV vaccine) versus MAV (mono-antigenic yeast-derived alum-adjuvanted vaccine)[4]. HBeAg, hepatitis B e antigen; HBsAg, hepatitis B surface antigen.

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Postinfection Irritable Bowel Syndrome

Uday C Ghoshal

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Following acute gastroenteritis (AGE) due to bacteria, viruses, or protozoa, a subset of patients develop new onset Rome criteria positive irritable bowel syndrome (IBS), called postinfection IBS (PI-IBS). The pooled prevalence of PI-IBS following AGE was 11.5%. PI-IBS is the best natural model that suggests that a subset of patients with IBS may have an organic basis. Several factors are associated with a greater risk of development of PI-IBS following AGE including female sex, younger age, smoking, severity of AGE, abdominal pain, bleeding per rectum, treatment with antibiotics, anxiety, depression, somatization, neuroticism, recent adverse life events, hypochondriasis, extroversion, negative illness beliefs, history of stress, sleep disturbance, and family history of functional gastrointestinal disorders (FGIDs), currently called disorder of gut-brain interaction. Most patients with PI-IBS present with either diarrhea-predominant IBS or the mixed subtype of IBS, and overlap with other FGIDs, such as functional dyspepsia is common. The drugs used to treat non-constipation IBS may also be useful in PI-IBS treatment. Since randomized controlled trials on the efficacy of drugs to treat PI-IBS are rare, more studies are needed on this issue. **(Gut Liver 2022;16:331-340)**

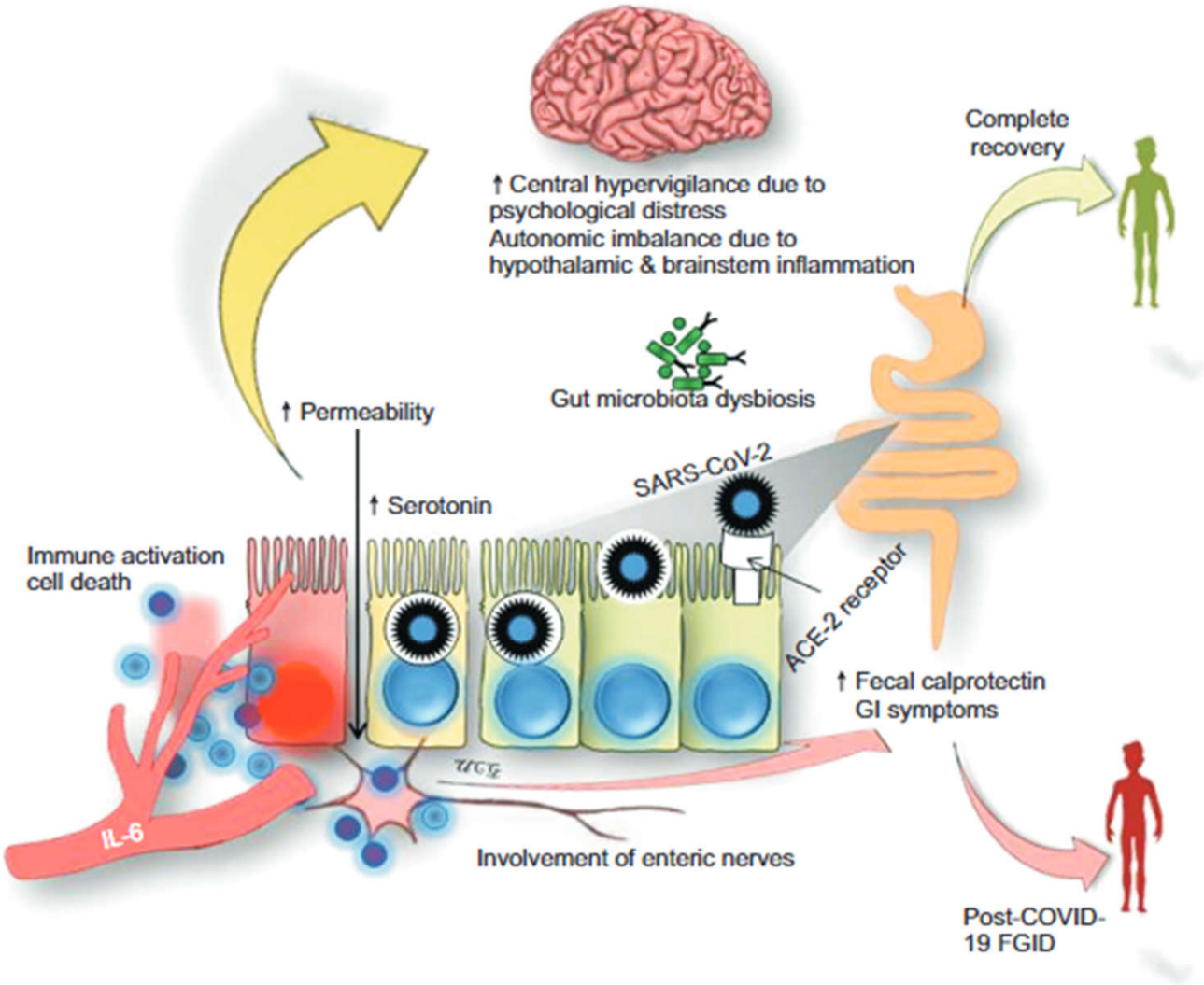

Fig. 1. Putative pathophysiological mechanism of post-coronavirus disease 2019 (COVID-19) functional gastrointestinal disorders (FGIDs), currently called disorders of the gut-brain interaction (DGBI). Severe acute respiratory distress syndrome coronavirus-2 (SARS-CoV-2) affects the gastrointestinal (GI) tract after entering via the angiotensin-converting enzyme-2 (ACE-2) receptor, which leads to immune activation (including increase in inflammatory markers such as interleukin-6 [IL-6]), damage to intestinal mucosa associated with GI symptoms, the presence of SARS-CoV-2 ribonucleic acid in feces, abnormal intestinal permeability, increased fecal calprotectin, increased mucosal serotonin, and gut microbiota dysbiosis. Increased visceral sensitivity at the gut level due to enteric nervous system involvement and at the central level due to psychological stress may contribute to the development of post-COVID-19 FGIDs/DGBI.

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120%

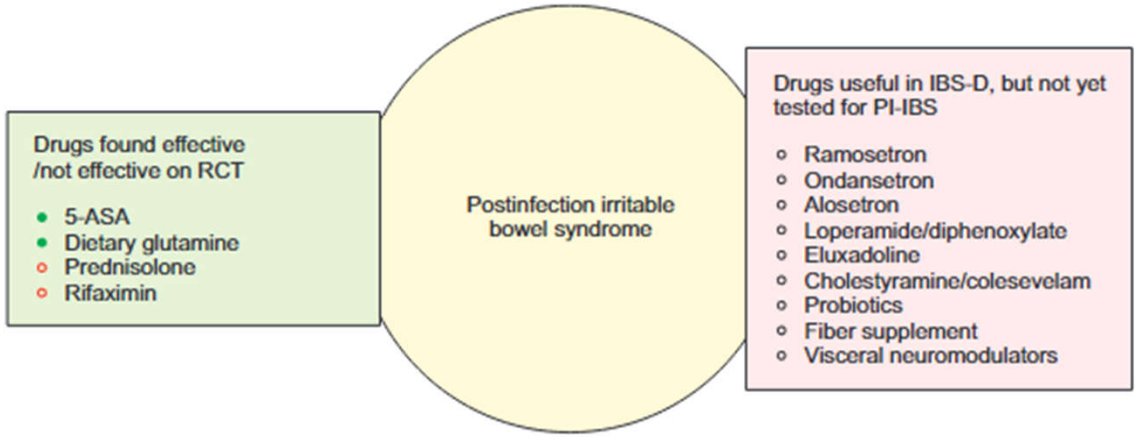
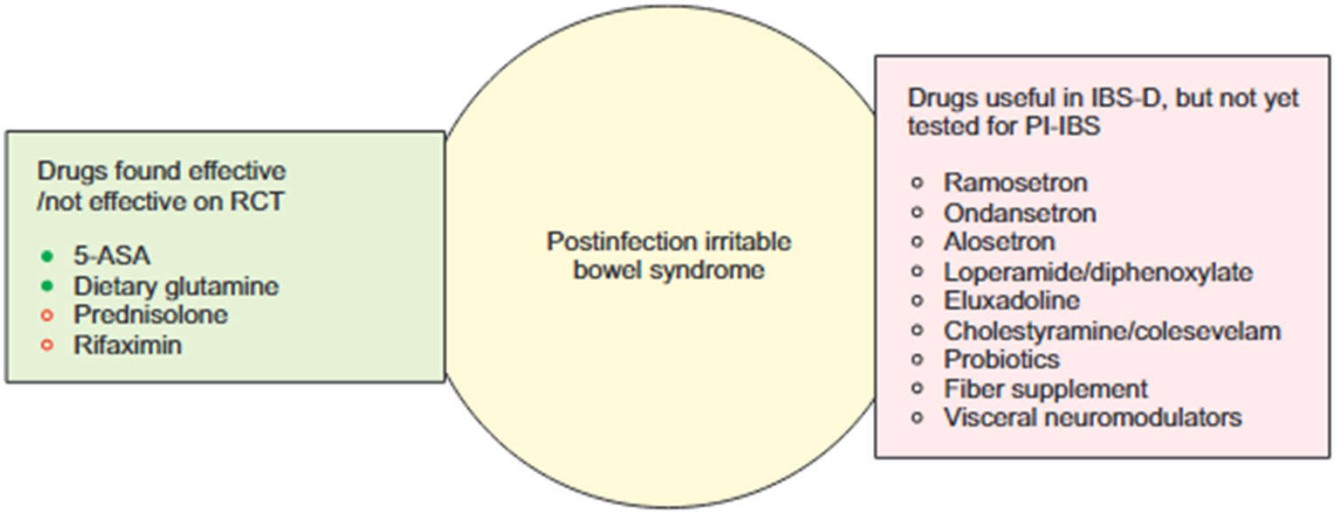


Fig. 2. Potentially useful drugs in the treatment of PI-IBS. In the light green box, the drugs that have been evaluated in RCTs are listed (filled green circles indicate the drugs that were found to be effective in the RCTs, and empty red circles show those that were found to be ineffective). The pink box lists the drugs that were found to be effective in diarrhea-predominant IBS but have not yet been evaluated in PI-IBS.
5-ASA, 5-aminosalicylic acid; RCT, randomized controlled trial; IBS, irritable bowel syndrome; IBS-D, diarrhea-predominant IBS; PI-IBS, postinfection IBS.

Table 3. Drugs (Including the Dosages) Used in the Treatment of Diarrhea-Predominant Irritable Bowel Syndrome (IBS) That May Also Be Useful in Postinfection IBS

Drug class	Agents	Dose
Opiates	Loperamide	2–4 mg, 4 times per day
	Diphenoxylate	2.5–5 mg, 4 times per day
	Eluxadoline	100 mg twice daily
Bile acid modulators	Cholestyramine	4 g daily or up to 4 times per day
	Colestipol	4 g daily or up to 4 times per day
	Colesevelam	1,875 mg up to twice daily
	Obeticholic acid	25 mg per day
5HT-3 receptor antagonists	Alosetron	0.5–1 mg twice daily
	Ondansetron	2–8 mg twice daily
	Ramosetron	5 μg per day



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	Eluxadoline	100 mg twice daily
Bile acid modulators	Cholestyramine	4 g daily or up to 4 times per day
	Colestipol	4 g daily or up to 4 times per day
	Colesevelam	1,875 mg up to twice daily
	Obeticholic acid	25 mg per day
5HT-3 receptor antagonists	Alosetron	0.5–1 mg twice daily
	Ondansetron	2–8 mg twice daily
	Ramosetron	5 μg per day
Gut microbiota manipulators		
Probiotics	*Lactobacillus* sp., *Saccharomyces boulardii*	
Antibiotics	Rifaximin	550 mg thrice daily for 14 days
Fiber supplements	Calcium polycarbophil	5–10 g daily
	Psyllium	10–20 g daily
	Pectin	2 Capsules daily before meal

CONCLUSION AND FUTURE DIRECTIONS

PI-IBS is a disorder of gut-brain interaction (previously called FGID), which undoubtedly proved that some patients with FGIDs are not entirely functional.[3,4] PI-IBS may occur following bacterial, protozoal, and viral AGE.[10] Two recent studies, one from Bangladesh and the other from India showed that cholera may be followed by the development of PI-IBS.[32,33] A pooled prevalence of PI-IBS was 11.5% after an episode of AGE.[40] Most patients with PI-IBS either present as IBS-D or mixed subtype of IBS.[40] Overlap of PI-IBS with other FGIDs, mainly, functional dyspepsia is common.[32] One-fifth of patients with Rome criteria positive PI-IBS may have PI-malabsorption, popularly known as tropical sprue.[32] It is believed that the development of PI-IBS may follow COVID-19.[7] However, studies are urgently needed to evaluate the frequency, spectrum, and risk factors for the development of PI-IBS, if any, following COVID-19. Since most patients with PI-IBS presents with the non-constipation type of IBS, the drugs used to treat non-constipation IBS may also be useful in PI-IBS treatment. However, most of these drugs have not been evaluated for the treatment of IBS. Of the four drugs evaluated by randomized controlled trial, prednisolone and rifaximin were found ineffective in PI-IBS treatment.[81-83] Dietary glutamine was found useful in the treatment of PI-IBS in a Chinese study.[80] 5-Aminosalicylic acid undergoing randomized controlled trial was not found useful in IBS-D patients.[79] However, in the subgroup of patients with PI-IBS, 5-aminosalicylic acid was found useful in IBS-D patients.[79] More studies are needed to evaluate the efficacy of various drugs in the treatment of PI-IBS.

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Ann Acad Med Singap. 2017 Jul;46(7):282-286.

Routine Staging Using Chest Computed Tomography in Workup of Treatment-Naïve Hepatocellular Carcinoma Prior to Locoregional Therapy: Is There a Need?

Po Wey Leong [1], Uei Pua, Kian Soon Lim

Affiliations + expand

Alpha fetoprotein plays a critical role in promoting metastasis of hepatocellular carcinoma cells

Yan Lu [a, b, #], Mingyue Zhu [a, b, #], Wei Li [a, b, #], Bo Lin [a, b], Xu Dong [a, b], Yi Chen [a, b], Xieju Xie [a, c], Junli Guo [a, b, *], Mengsen Li [a, b, d, *]

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[b] Key Laboratory of Molecular Biology, Hainan Medical College, Haikou, Hainan Province, China
[c] Department of Pathophysiology, Hainan Medical College, Haikou, Hainan Province, China
[d] Institution of Tumor, Hainan Medical College, Haikou, Hainan Province, China

In conclusion, this report is the first time to demonstrate that AFP promoted HCC cells metastasis directly. AFP overexpression in HCC cells was related to metastatic characteristics in human HCC patients. Overexpression of AFP plays a critical role in promoting invasion and distant metastasis of HCC cells through up-regulating expression of metastasis-related proteins. The molecular mechanism of AFP promoted metastasis of HCC cells *via* activating PI3K/AKT signal pathway. Thus, AFP could be applied as a novel therapeutic target for treating HCC patients.

XÉT NGHIỆM MÁU

MEDIC

I. HUYẾT HỌC / ĐÔNG MÁU - HEMATOLOGY / COAGULATION

NFS(C.B.C)(CÔNG THỨC MÁU)	*	
WBC	8.41	(4.0-10.0)10^9/L
% Neu	68.7	(40 - 74 %)
% Lym	21.9	(19 - 48 %)
% Mono	6.6	(3 - 9 %)
% Eos	1.5	(0 - 7 %)
% Baso	0.8	(0 - 1.5 %)
# Neu	5.77	(1.7 – 7.0) 10^9/L
# Lym	1.84	(1.0 – 4.0) 10^9/L
# Mono	0.55	(0.1 – 1.0) 10^9/L
# Eos	0.13	(0 – 0.5) 10^9/L
# Baso	0.07	(0 – 0.2) 10^9/L
RBC	5.28	(3.80 - 5.60)10^12/L
Hb	15.1	(12 - 18 g/dL)
Hct	45.9	(35 - 52 %)
MCV	86.9	(80 - 97 fL)
MCH	28.7	(26 - 32 pg)
MCHC	33.0	(31 - 36 g/dL)
RDW	15.4	(11.0 - 15.7%)
PLT	143	(130 - 400)10^9/L
MPV	9.1	(6.30 - 12.0 fL)

Uric Acid/Serum	4.65	(1
hs CRP	9.50 H	(≤
Độ Lọc Cầu Thận (CKD-EPI)	*	
Creatinin/Serum	0.869	(1

IV. MIỄN DỊCH - IMMUNOLOGY

B.N.P	32.50	(≤ 100 pg/mL)	QTMD12
TSH u.sensitive (3rd G)[1]	1.04	(0.32 -5 µIU/ml)	QTMD00
Free T4	0.960	(0.71 - 1.85 ng/dl)	QTMD03
A.F.P[1]	16525 H	(< 20 ng/ml)	QTMD00
C.E.A[1]	3.29	(< 5 ng/mL)	QTMD00
Pro GRP	41.10	(< 65 pg/mL)	QTMD17
HBsAg (Định tính, qualitative)	POS S/CO 4932	(Index <1; S/Co <1)	QTMD01
Anti HBs (Định lượng, quantitative)	0.420	(≥ 10 mIU/mL)	QTMD12
Anti HBc Total (IgG+IgM)	POS S/CO 8.66	(S/Co < 1, Index < 0.5)	QTMD12
Anti HCV (Định tính, qualitative)	NEG S/CO 0.130	(S/Co < 1; Index < 1)	QTMD01
CYFRA 21-1	5.81 H	(< 3.3 ng/mL)	QTMD03
IgE	2016 H	(< 130 UI/mL)	
Troponin-I hs (Abbott)[1]	1.90	(M < 34.2 ng/L, F < 15.6 ng/L)	QTMD01

V. SINH HỌC PHÂN TỬ - MOLECULAR BIOLOGY

HBV-DNA	Positive	(NEGATIVE)	

III. SINH HOÁ - BIOCHEMISTRY

Glucose (FPG)[1]	4.93	(3.90 - 5.90 mmol/L)	QTSH00
GGT[1]	309.6 H	(M < 55 U/L; F < 36 U/L)	QTSH00
CPK-MB (Abbott)	2.10	(< 7 ng/ml)	QTSH07
SGOT (AST)[1]	72.17 H	(< 35 U/L)	QTSH00