International Journal on Gastroenterology and Liver Disorders

Splanchnic Inflammatory System – Expert Opinion

Mini Review

Volume 3 Issue 2- 2023

Author Details

Richard C Semelka MD[1], Miguel Ramalho MD[2]*

[1]*Richard Semelka Consulting, PLLC, USA*
[2]*Department of Radiology, Hospital da Luz, Portugal*

*Corresponding author

Richard Semelka, Richard Semelka Consulting, PLLC, Chapel Hill, North Carolina, USA

Article History

Received: November 7, 2023 Accepted: November 11, 2023 Published: : November 14, 2023

Mini Review

Metabolic Syndrome, Irritable Bowel Syndrome (IBS), and leaky gut describe inflammatory processes of organs in the splanchnic system. Our current work with MRI has shown that imaging findings of inflammation of segments of the upper GI tract (lower esophagus, stomach, duodenum, and jejunum) and hepatosteatosis are commonly observed in individuals with these clinical conditions and in obese patients presenting with abdominal pain. One initial consideration is that these conditions, and other similar ones, are all aspects of the same clinical picture, which may be best termed Splanchnic Metabolic Syndrome. There may be various underlying causes, but the essential findings are inflammation of the upper GI tract and hepatosteatosis, recognized as a widespread finding [1], with the root cause being the entry point to the system from the exterior, the upper GI tract.

Beyond the liver and GI tract, inflammation of all organs and tissues in the splanchnic system can be observed. The only imaging approach that can define the full extent of splanchnic inflammation and related disease is Gadolinium-based Contrast agent (GBCA) enhanced MRI. On imaging, this condition can be termed Splanchnic Inflammatory Syndrome (SIS). Our approach for imaging the splanchnic system involves imaging on modern MR systems, using conventional MR sequences, which include: 1. short duration fat-suppressed T2-weighted; 2. In and Out-of-Phase T1 gradient echo sequence (Dixon may be optimal); 3. noncontrast T1 fat-suppressed (or similar dark-fat) gradient echo; late arterial and 5-minute post-GBCA gradient echo. Figure 1 illustrates the imaging findings demonstrable on MRI of the SIS.

Based on our initial observations and experience of over 1,000 subjects, many conditions in the splanchnic system that are inflammatory and may reflect, and probably most commonly reflect, SIS, as these other findings are commonly (possibly invariably) related to co-existent upper GI inflammation and hepatosteatosis. These include:

a. acute acalculous cholecystitis
b. biliary dysfunction of all forms
c. pancreatic steatosis
d. small pancreatic cysts, including side-branch IPMN.
e. mesenteric panniculitis.

Many benign and malignant lesions in these organ systems may also arise as a result of chronic inflammation of the splanchnic system.

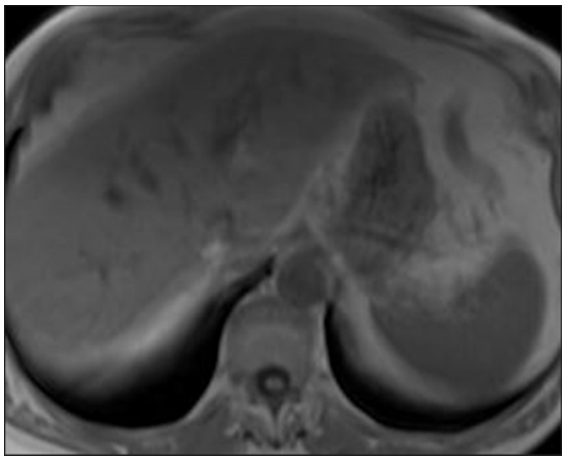

Figure 1: 1(a).

Image Interpretation

Liver and pancreatic steatosis are optimally evaluated using in-and-out-phase and comparable (e.g., Dixon) strategies of all imaging approaches. Fat content can be quantified with this approach [2]. Gallbladder and biliary dysfunction, small pancreatic cysts, and IPMNs are well recognized on MRI, and many of these sequences are used in concert [3,4].

Mesenteric panniculitis is best shown on out-of-phase images supplemented by other sequences [5]. Inflammation of the GI tract is shown in the combination of late arterial and 5-minute post-contrast images [6]. Early enhancement is consistent with significant acute inflammation, and late enhancement reflects prolonged interstitial dwell time in the extracellular matrix (ECM) seen in acute and chronic inflammation.

The least generally understood imaging interpretation is the increased enhancement of the upper GI tract, which should follow the established approach for imaging Crohn's of the terminal ileum, Crohn's, ulcerative colitis, and infective colitis in the colon [6]. The great majority of body MR radiologists to the present time do not have the experience to interpret upper GI tract inflammation well. This may partly reflect that inflammation is common and historically misinterpreted as normal.

The first understanding is that the normal bowel should enhance approximately the same intensity as the paraspinal and other muscles. The greater the extent of inflammation of the bowel, the closer the bowel enhances to the signal of normal renal cortex and pancreas on arterial phase images and renal parenchyma on 5-minute post-contrast images. The most accessible segment to interpret as inflamed is the distal esophagus; the most challenging is the jejunum. Three patterns of abnormal jejunal enhancement are observed: diffusely increased enhancement (observed on arterial phase or both phases), patchy jejunal enhancement, and 5-minute increased serosal enhancement. This last appearance is often seen alone but invariably is present with mesenteric panniculitis and, when seen together, usually reflects more long-standing mesenteric inflammation. The ileum may also be shown to have a small caliber tubular, relatively featureless appearance with moderately intense mural enhancement that should be described.

We observe that many individuals with acute acalculous cholecystitis have the MR appearance of intense inflammation of the proximal duodenum associated with a gallbladder that shows progressively intense mural enhancement. A similar appearance is observed in cases of biliary dyskinesia.

Discussion

Obesity has become the most important disease condition in the American population and is progressively a worldwide phenomenon [1]. The Metabolic Syndrome, therefore, may be one of the most common conditions, yet it is under-reported [7-10]. We consider this partly because the designation is too broad and, hence, confusing. What of the many Metabolisms? A condition associated with Type II diabetes and dyslipidemia should be termed the Splanchnic Metabolic Syndrome, as other organ system metabolic conditions have come to be described according to the organs involved. Based on our considerable imaging experience, the findings of what has been called the Metabolic Syndrome, IBS, and leaky gut all have essentially identical MR findings. Hence, we believe the Splanchnic Metabolic Syndrome and the bowel manifestation represent a leaky gut early on and IBS as inflammation intensifies. Our opinion is that there is one umbrella condition, the Splanchnic Metabolic Syndrome, with likely multiple causes and bowel findings that reflect a prominent component of bowel inflammation and biliary findings a prominent component of biliary disease.

It may be particularly important to draw attention to the fact that individuals at present who have RUQ pain and normal appearance of the GB at sonography will undergo cholecystectomy. Studies show that clinical failure is expected because a sizable percentage have persistent RUQ. This reflects that the source of pain all along is the proximal duodenum, and inflammation of the gallbladder is a sympathetic response to bowel inflammation. Continued research into associated conditions in the Metabolic Syndrome has shown that multiple findings are evident: upper GI inflammation is common [11-13], fatty change of the pancreas is common [7,14,15], and mesenteric panniculitis is not uncommon [16]. This is identical to what we have observed in the MR-based imaging designation of the SIS.

At present, the full spectrum of SIS findings is not reported in MR studies due to the lack of experience of most readers. However, this may currently be the most common disease present in MR studies and may be invariably present in adult obese patients with abdominal pain. A final word on treatment. The primary treatment for SIS (and Splanchnic Metabolic Syndrome) is careful dietary modification. Our opinion is that individuals often experience intolerance to various foods, and this should be evaluated. Gluten may be a common cause, but many other food sources could be the cause. Also important is to determine if hyperchlorhydria or hypochlorhydria is a prominent component of upper GI inflammation, which may be most efficiently performed as a week-long apple vinegar trial. SIS is very common; possibly only on MRI can the full extent be observed. Improved knowledge of interpreting upper GI findings also needs to be expanded.

Mild SIS As Shown on MRI

In-phase (a) and out-of-phase [Figure 2] (b) T1 gradient-echo images show normal relation mild higher signal of liver than spleen on in-phase (a) with signals becoming equivalent on out-of-phase (b) reflecting mild hepatosteatosis (measured fat fraction 11%). 5 min post-GBCA image of the esophagus [Figure 3] (c) shows mild increased enhancement of the esophagus (arrow, c) with a 4 mm thick wall (within normal measurement) consistent with mild distal esophagitis. Duodenum shows mild increased enhancement with 5 mm mural thickness, as shown on the late hepatic arterial phase image [Figure 4] (d). Late arterial [Figure 5] (e) and 5 min [Figure 6] (f) images at a lower anatomic level show mild increased enhancement of a portion of jejunum (arrow, e, f).

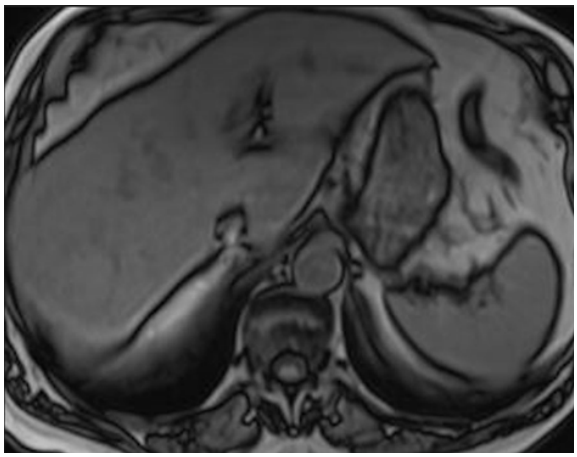

Figure 2: 1(b).

A mild level of upper GI inflammation and mild hepatosteatosis is the most common presentation of SIS. This is commonly observed in obese individuals with a clinical history of abdominal pain, which can be generalized, RUQ, or LUQ. The location of pain often reflects the location of the bowel, which is most inflamed.

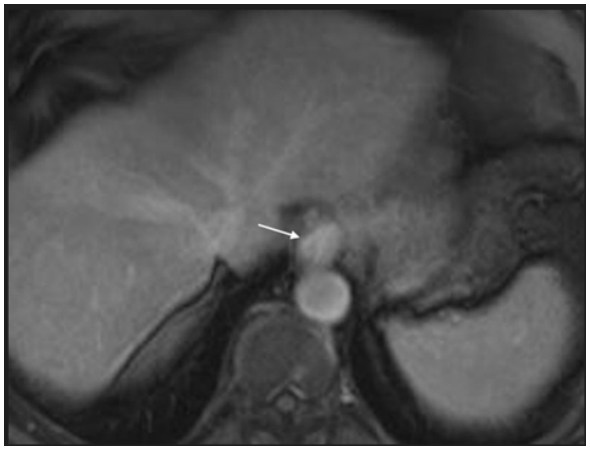

Figure 3: 1(c).

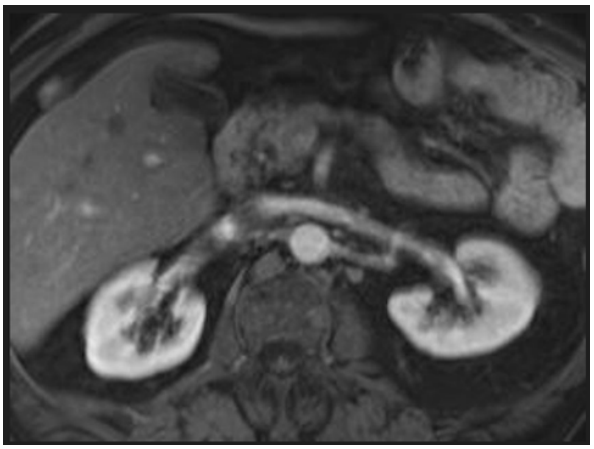

Figure 4: 1(d).

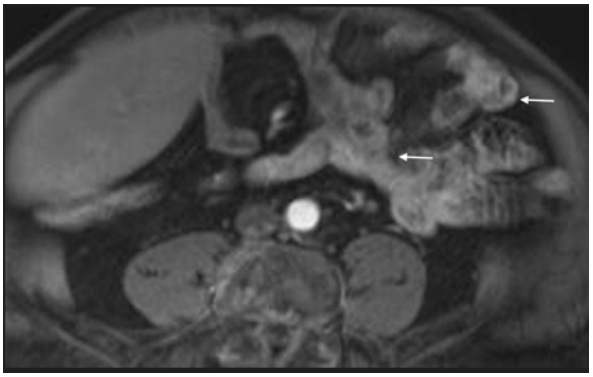

Figure 5: 1(e)**.**

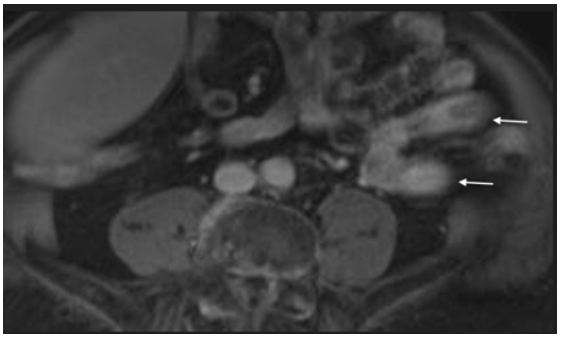

Figure 6: 1(f)**.**

References

1. Mitra S, De A, Chowdhury A, (2020) Epidemiology of nonalcoholic and alcoholic fatty liver diseases. Transl Gastroenterol Hepatol 5: 16.
2. Starekova J, Hernando D, Pickhardt PJ, Reeder SB, (2021) Quantification of Liver Fat Content with CT and MRI: State of the Art Radiology 301(2): 250-262.
3. Altun E, Semelka RC, Elias J, Jr Braga L, Voultsinos V, et al. (2007) Acute cholecystitis: MR findings and differentiation from chronic cholecystitis. Radiology 244(1): 174-83.
4. Harrington KA, Shukla-Dave A, Paudyal R, Do RKG, (2020) MRI of the Pancreas. J Magn Reson Imaging 53(2): 347-359.
5. Buragina G, Magenta Biasina A, Carrafiello G, (2019) Clinical and radiological features of mesenteric panniculitis: a critical overview. Acta Biomed 90(4): 411-422.
6. Maccioni F, Bruni A, Viscido A, Colaiacomo MC, Cocco A, et al. (2006) MR imaging in patients with Crohn's disease: the value of T2- versus T1-weighted gadolinium-enhanced MR sequences with use of an oral superparamagnetic contrast agent. Radiology 238(2) :517-530.
7. Ballester-Valles C, Flores-Mendez J, Delgado-Moraleda J, Ballesteros Martin-Portugues A, Merino-Torres JF, et al. (2020) Hepatic and pancreatic fat as imaging biomarkers of metabolic syndrome. Radiologia 62(2): 122-130.
8. Graffy PM, Pickhardt PJ, (2016) Quantification of hepatic and visceral fat by CT and MR imaging: relevance to the obesity epidemic, metabolic syndrome, and NAFLD. Br J Radiol 89(1062): 20151024.
9. Neeland IJ, Ross R, Despres JP, Matsuzawa Y, Yamashita S, et al. (2019) Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. Lancet Diabetes Endocrinol 7(9): 715-725.
10. Wagner R, Eckstein SS, Yamazaki H, Gerst F, Machann J, et al. (2022) Metabolic implications of pancreatic fat accumulation. Nat Rev Endocrinol 18(1): 43-54.
11. Sogabe M, Okahisa T, Kimura T, Okamoto K, Miyamoto H, et al. (2016) Influence of metabolic syndrome on upper gastrointestinal disease. Clin J Gastroenterol 9(4): 191-202.
12. Lee YC, Yen AM, Tai JJ, S-H Chang, J-T Lin, et al. (2009) The effect of metabolic risk factors on the natural course of gastro-oesophageal reflux disease. Gut 58: 174-81.
13. Yamamoto S, Watabe K, Takehara T, (2012) Is obesity a new risk factor for gastritis?. Digestion 85(2): 108-110.
14. Rugivarodom M, Geeratragool T, Pausawasdi N, Charatcharoenwitthaya P, (2022) Fatty Pancreas: Linking Pancreas Pathophysiology to Nonalcoholic Fatty Liver Disease. J Clin Transl Hepatol 10(6): 1229-1239.
15. Bi Y, Wang JL, Li ML, Zhou J, Sun XL, (2019) The association between pancreas steatosis and metabolic syndrome: A systematic review and meta-analysis. Diabetes Metab Res Rev 35(5): e3142.
16. Gunes SO, Akturk Y, Guldogan ES, Yilmaz KB, Ergun O, et al. (2021) Association between mesenteric panniculitis and non-neoplastic disorders. Clin Imaging 79: 219-224.