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**INTERNATIONAL JOURNAL OF  
 ADVANCED RESEARCH (IJAR)**

Article DOI:10.21474/IJAR01/2451  
 DOI URL: <http://dx.doi.org/10.21474/IJAR01/2451>



### RESEARCH ARTICLE

#### A CASE OF MESENTERIC VASCULITIS.

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#### Manuscript Info

##### Manuscript History

Received: 20 October 2016  
 Final Accepted: 22 November 2016  
 Published: December 2016

##### Key words:-

Vasculitis, Polyarteritis nodosa,  
 Localized vasculitis of the  
 gastrointestinal tract (LVGT)

#### Abstract

The mechanisms of vasculitis are poorly understood, but involve immune-mediated destruction of vessel walls. Depending on the syndrome, there is significant variability in the size and types of vessels involved, as well as the nature of the inflammatory infiltrate. Localized gastrointestinal vasculitis is a rare condition. may present as unexplained abdominal pain with or without unexplained lower gastrointestinal bleeding. In this report we describe a new case of localized vasculitis of the gastrointestinal tract involving the celiac trunk. The most important step in evaluating patient with suspected LVGT to determine whether the disease involved single organ or an initial manifestation of a more severe systemic vasculitis. That's why a comprehensive evaluation must be conducted because it's associated with significant morbidity and mortality.

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#### Introduction:-

Vasculitis is an inflammatory disorder of blood vessels, can affect vessels of any type in any organ. Gastrointestinal (GI) manifestations of vasculitis are considered rare and the presentation is often nonspecific ranging from gastrointestinal dysfunction such as motility disorders to ischemia that may result in ulceration and perforation. GI involvement is frequent in polyarteritis nodosa (PAN), Henoch-Schönlein purpura and also often noted in microscopic polyangiitis and Takayasu arteritis. The diagnosis and classification of vasculitis depends on a combination of clinical, serological, radiological and histological findings.

#### Case Description:-

A 52 years old Saudi male with seizure disorder and dyslipidemia presented to ER complaining of abdominal pain for three days. He was in his usual state of health till 3 days prior to this presentation when he developed sudden, intermittent, colicky, upper abdominal pain unrelated to food intake, 5/10 in severity, with no aggravating/relieving factors or associated symptoms like fever, weight loss, nausea, vomiting or change in bowel habits. No recent travel or use of NSAIDs. He had no similar attacks in the past. He has been seizure free on medication for several years. His only took rosuvastatin 10 mg qd and carbamazepine 200 mg bid on regular basis. He didn't smoke, drink alcohol or use illicit drugs.

#### On Examination:-

Temperature: 36.6°C, BP: 118/70, pulse: 80 beats/min and respiratory rate: 18/min. Head and ENT exam showed no mucosal ulcers, pallor, or jaundice. Neck exam showed no jugular venous distension, lymphadenopathy, or bruits. Chest examination was clear to auscultation. His cardiovascular examination revealed normal S1 and S2, with no

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murmurs. Abdominal examination showed mild epigastric, right and left upper quadrant tenderness without rebound tenderness, guarding or rigidity. There was no organomegaly and bowel sounds were normal. Rectal exam was negative for occult blood. Neurological and musculoskeletal examinations were normal. Skin exam showed no rash, erythema or discoloration. Extremities exam showed no clubbing, cyanosis, or edema with normal pulses.

**Lab works:-**

WBC: 5500/L, Hemoglobin: 14 g/dl, Platelet count: 236,000/L, ESR: 11, CRP: 27.3 mg/L. Normal amylase, lipase, electrolytes, liver and kidney function tests and negative hepatitis profile

Autoimmune work up showed negative ANCA, ANA, RF, cryoglobulins, anti-cardiolipin antibodies, lupus anticoagulants, beta-2 glycoprotein I antibodies with normal C3 and C4

Hypercoagulability work up showed normal PT, PTT, protein C, protein S and anti-thrombin III with negative factor V Leiden and thrombin mutations

The patient was admitted to general surgical ward and was started on IV fluids and analgesics. His liquid diet was well tolerated and eventually advanced to regular diet as abdominal pain subsided. Abdominal CT with contrast showed marked thickening of the wall of the celiac artery and its branches with patent lumen and a splenic infarction but normal bowels and no involvement of other vessels. (Figure 1)

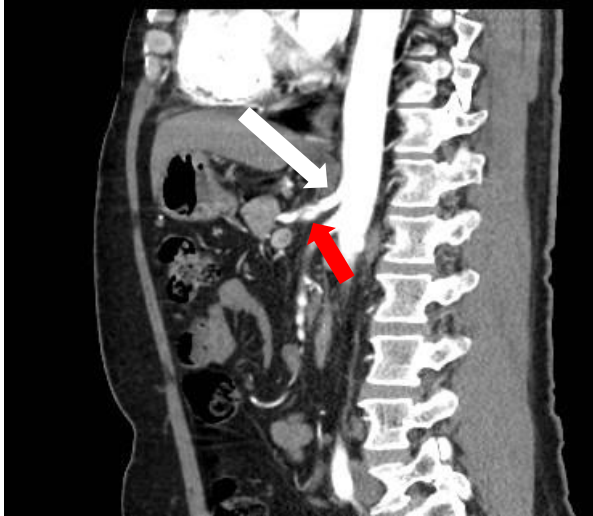


**Figure 1:-** Contrast-Enhanced abdominal CT

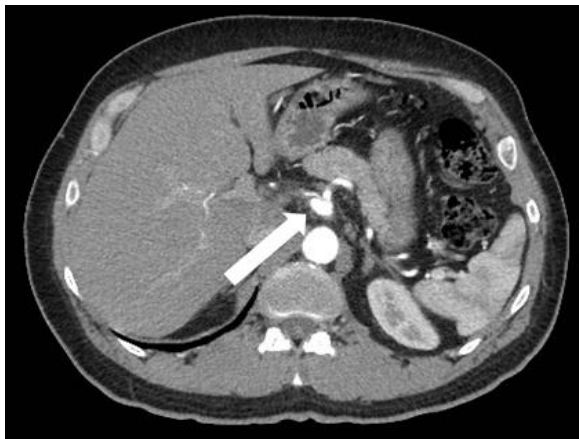
Vascular surgery and rheumatology services were consulted to entertain the diagnosis of isolated vasculitis of the celiac trunk and its branches complicated with splenic infarction. Then the patient was started on prednisone 60 mg qd in addition to calcium and vitamin D.

His CRP dropped from 27.3 mg/L to 7.8 mg/L after 3 days of prednisone therapy with complete resolution of abdominal pain. The patient was discharged home to follow up in the rheumatology clinic 2 weeks later.

During his follow up visit, he was still completely asymptomatic with normal CRP. After 3 months of prednisone therapy, a follow up abdominal CT with contrast (Figure 2 & 3) showed improvement of the narrowing at the origin of the celiac trunk and development of a small aneurysm at the origin of the hepatic artery. Prednisone was tapered down to 30 mg. DXA scan showed T-score of -2.5 at the lumbar spine and IV Zoledronic acid 5 mg was given to be repeatedly yearly.



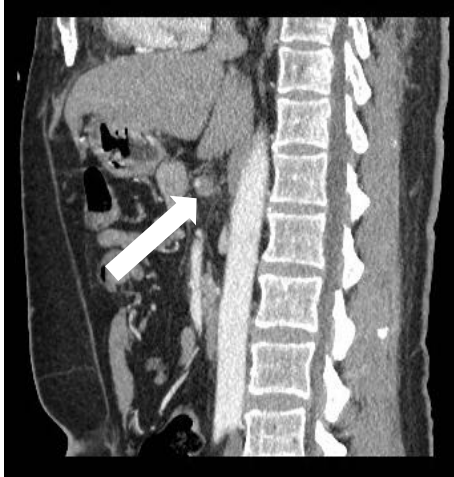
**Figure 2:-** Contrast-Enhanced CT Abdomen: Improvement of the narrowing at the origin of the celiac trunk (*white arrow*) and a small aneurysm at the origin of the hepatic artery (*red arrow*).



**Figure 3:-** Contrast-Enhanced CT Abdomen showed an aneurysm arising at the root of the hepatic artery (*white arrow*).

Then the patient remained asymptomatic with prednisone taper and his lab results including ESR, CRP, and liver function tests were within normal limits.

CT angiogram of the abdomen was done 3 months later and showed a little worsening of the hepatic artery narrowing with a thrombus formation in the aneurysm at the origin of the hepatic artery (Figure 4&5). Aspirin 81 mg qd and azathioprine 100 mg qd were started and prednisone was tapered off over 5 weeks.



**Figure 4:-** CT Angiogram of the abdomen.



**Figure 5:-** CT angiogram of the abdomen shows a newly developed thrombus in the aneurysm at the root of the hepatic artery (arrows).

### **Discussion:-**

Vasculitis involving the gastrointestinal (GI) tract often occurs as part of a systemic inflammatory process and is a well-recognized manifestation of small- and medium-sized vessel vasculitides [1,2]. In particular, GI involvement frequently occurs in polyarteritis nodosa (PAN), ANCA-associated vasculitis, Henoch–Schönlein purpura, and Takayasu arteritis [1,2,3]. When present, GI complications adversely affect prognosis and are an indicator of disease severity [2]. Vasculitis of the GI tract may occur in isolation as a form of single-organ vasculitis (SOV) referred to as localized vasculitis of the GI tract (LVGT). [4,5]. SOV tends to have better prognosis and excision of the vasculitic lesion can be curative, although SOV can also progress to a systemic illness [2,6,7].

There are limited data in the literature regarding localized vasculitis of the GI tract (LVGT) and this entity is not well understood. The clinical features of LVGT have primarily been described in individual case reports and small case series. GI manifestations of systemic vasculitis present with abdominal pain, nausea/vomiting, diarrhea, or symptoms of GI bleeding, and therefore, may be indistinguishable from clinical features of LVGT [2,7].

In one case series, most patients had multiple manifestations among which abdominal pain was the most frequent, present in almost all patients (94.4%). The pain was usually intense with no preferential site [7]. Other manifestations included abdominal angina (44%), nausea/vomiting (66.7%), diarrhea (8%), hematochezia/ melena (16.7%), weight loss (72.2%) and fever (22.2%)[7]. The laboratory features found in patients with LVGT are nonspecific. Inflammatory markers may be elevated, although half of the patients in that series had an ESR <30 mm/h [7]. Autoantibody profiles were negative in all patients; conversely, patients with systemic vasculitis or

connective tissue diseases with GI involvement frequently have positive antibodies, including ANA and ANCA. [1,2,7]

**Conclusion:-**

Although uncommon, LVGT can be associated with significant morbidity and mortality. Patients with LVGT typically present with abdominal pain. Diagnosis requires radiographic imaging studies or histopathological examination of surgical specimens.

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