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Solid Abdominal Organ infarcts of Unknown Etiology: Could silent atrial fibrillation the undetected culprit?

Case Report

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Abstract

Background: Solid organ infarctions are uncommon causes of abdominal or flank pain. This could partially be due to under-diagnosis. Several causes, including emboli in a setting of atrial fibrillation (afib) or left ventricular thrombus formation, vasculitis, local vascular compression, underlying malignancies, infections, vascular dissection and substance abuse may be responsible.

Aim: Despite extensive diagnostic work up, the exact source, or the reason for the emboli remains unknown. Small studies have highlighted that afib is one possible cause for emboli, similar to the mechanism of stroke. Determining the cause, when possible, may help initiate appropriate treatment to avoid future events that can lead to a devastating stroke, myocardial infarction or organ loss. We seek to investigate the cause of the infarct in all these cases.

Design: Retrospective study

Method: Case review including diagnostic tests and literature review.

Conclusion: No clear source was identified. Intermittent afib could be one source, but due to lack of long term rhythm monitoring, no definitive conclusion could be made.

Introduction

Solid organ infarcts are uncommonly diagnosed etiologies of abdominal or flank pain, and proper diagnostic tests are often not performed [1,2]. Sometimes the diagnosis of solid organin farction is incidentally found on images obtained for other reasons. A number of common diseases such as kidney stones, musculoskeletal pain and lumbago mimic the symptoms of kidney or splenic infarct. Solid abdominal organ infarcts are the result of emboli, dissection of vessels, malignancies, hypercoagulable states, vasculitides or infections. Other causes include drugs and

vascular anomalies or compression from local masses or lymph nodes [3-12].

Solid abdominal organ infarcts will not be diagnosed unless suspected, and the proper diagnostic tool chosen [13]. Even in cases where pyelonephritis or kidney stone is suspected and the imaging is non-diagnostic, at times it is believed that the stone has passed and the pain has persisted due to irritation of the ureter during stone passage.

Huang et al. did a study in their Emergency Department (ED) looking for characteristics of patients that may tip the physician in the direction of a kidney infarct diagnosis [14]. Multiple case reports and retrospective studies have highlighted the frequency of solid organ infarcts. Two of the organs affected are the spleen and the kidneys [2,14-16]. A small number of studies have looked at case series of patients with splenic and renal infarcts and have attempted to study the possible causes. Afib is one of the causes listed as an etiologic factor [15, 17-19].

Here we report cases of young and relative young patients who presented with intractable abdominal or flank pain and had solid abdominal organ infarcts.

Case presentations

Below are cases from different hospitals and their work up is summarized in Table1.

Case 1

A 31 year old African American female presented to the ED complaining of constant, progressive epigastric/left upper quadrant (LUQ) and left flank pain for 3 weeks, not responding to acetaminophen. Review of systems (ROS) was remarkable for a fever of 105 degrees Fahrenheit, intermittent palpitations, dry cough, nausea, non-bloody vomiting, watery diarrhea and numbness in the left arm and leg.

The patient was seen at different hospitals, treated for suspected colitis. Diagnostic tests there showed positive for cytomegalovirus (CMV) and multiple splenic infarcts splenic infarcts on Computed tomography (CT).

The patient had no known drug allergies, but had confirmed sickle cell trait, depression and arthritis. She reported a past surgical history of left knee arthroscopy with lateral release (2009). Home medications included bupropion and trazodone, dose unknown. The patient denied tobacco and recreational drug use.

Vital signs at presentation were as follows: blood pressure: 136/75 mmHg; heart rate: 120 beats per minute; respiratory rate: 18 breaths per minute; oxygen saturation 99% on room air and temperature of 98.2 degrees Fahrenheit. Physical examination was remarkable for left upper quadrant (LUQ) tenderness with splenomegaly.

CT scan of the chest, abdomen and pelvis in our institution also showed multiple splenic infarctions with mediastinal lymphadenopathy and fatty infiltration of the colonic submucosa, but no pulmonary embolism.

The electrocardiogram (ECG) showed sinus tachycardia and chest x-ray (CXR) revealed mild peribronchial cuffing and arthritic changes of the spinal column.

The patient was treated with pain medication and antacids without significant improvement and was admitted for persistent pain. In addition, treatment with ganciclovir was started after consulting with the infectious disease (ID) team. The patient underwent upper and lower endoscopies for suspected CMV colitis with positive IgM titer, however, both failed to detect any remarkable macroscopic findings except chronic gastritis. The

Table 1: Demography, Laboratory tests and other studies

Categories	Case 1	Case 2	case 3	case 4
Date	1/18/2016	1/10/2018	4/26/2018	5/24/2018
Gender	F	F	M	F
Age	31	21	24	50
Pregnancy test	Negative	Negative	NP	Negative
CT abdomen and pelvis finding	multiple splenic infarcts	splenic infarcts	b/l kidney infarcts	multiple splenic infarcts
CT Chest finding	No PE	NP	No PE	NP

Duplex Ultrasound	NP	NP	No DVT, normal Aorta , IVC and renal arteries flow	upper and lower extremity no DVT
Echo (TTE)	mild mitral regurgitation, no mass, no wall motion abnormalities	three leaflet mild-moderate regurgitation, no mass, no wall motion abnormalities	tricuspid mild regurgitation, no mass, no wall motion abnormalities	PFO, no mass, no wall motion abnormalities
Ultrasound of organs	NP	Splenic infarct (portal vein splenic vein US)	Left kidney infarct	NP
ECG	NSR	NSR	Sinus bradycardia, LVH, TWI in I and avL	NSR, no Afib
comments	NA	Rheumatoid arthritis, r/o PE 2013 No PE on CT	NA	Previous DVT withPE 2004, PFO
CK (u/L) Normal range (21-232)	Normal (106)	NP	Normal (196)	NP
Antiphospholipid syndrome panel	positive	NP	Normal	NP
PTH Normal range (14-72 pg/mL)	NP	NP	NP	NP
CRP (mg/L) normal range <3	Elevated (77)	Normal	Normal	NP
ESR (mm/hr) Normal range (0-20)	Normal (18)	Normal (18)	Normal (3)	NP
Urine toxicology	Negative (7panel)	Negative (7 panel except THC)	THC positive (7 panel)	NP
Prothrombin gene analysis	NP	NP	NP	NP
Fibrinogen Normal range (200-400 mg/dL)	NP	NP	Normal (222)	NP
Antithrombin III function % Normal range 83-128	Normal (84%)	NP	NP	NP
Protein S antigen % Normal range (55-124)	Low (29)	Normal (74)	Normal (126)	NP
Protein C antigen % Normal range (80-184)	Cancelled	low (75)	Normal	NP
Protein C function % Normal range (7-140)	NP	NP	NP	NP
Coagulation panel	NP	NP	Normal	NP

D-dimer Normal range (<1 mg/L)	NP	NP	Normal (0.25)	Elevated (1.6)
Factor V Leiden	Normal	NP	Normal	NP
Homocysteine Normal range (5-15 umol/L)	NP	NP	Normal (7.8)	NP

CMV IgG Normal range <0.59 U/mL	Positive (2.20)	NP	NP	NP

Cardiolipin antibody IgG and IgM	NP	Negative	Normal	NP
Sickle Cell Disease screening test	Positive Sickle cell trait	Negative	Negative	NP
Proteinase-3 antibody (C-ANCA) Normal range <20 Units	Normal (<5)	Normal (<5)	NP	NP
AntiNuclear Antibody (ANA) titer Normal range <1:80	Negative	Elevated (1:320)	Negative	Negative
Double Stranded DNA Antibody Normal range (<29 IU/mL)	NP	Elevated (39)	NP	NP
Von Willebrand Factor	NP	NP	Negative	NP
Prothrombin Gene Analysis (Prothrombin Mutation)	Normal (<5)	NP	Normal	NP
Myeloperoxidase antibody (P-ANCA) Normal range <20 Units	Normal (<5)	Normal (9.1)	NP	NP
TSH Normal range (0.27-4.20uIU/mL)	NP	Normal (1.39)	Normal (2.86)	NP
CyclineCitrullin Peptide (CCP) antibody IgG Normal range (0-19 Units)	NP	Normal (<8)	NP	NP
CMV PCR Normal <200 copies per mL	Positive (1089)	NP	NP	NP
CMV IgM Normal range <29.9 AU/mL	Positive (240)	NP	NP	NP
CMV IgG Normal range <0.59 U/mL	Positive (2.20)	NP	NP	NP
EBV capsid IgM Normal range (<35.9 U/mL)	Positive (>160)	NP	NP	NP
EBV capsid IgG Normal range (<17.9 U/mL)	Positive (299)	NP	NP	NP
HBV	NP	NP	NP	NP
HCV	NP	NP	NP	Negative
HIV	NP	NP	NP	NP
Hexagonal array phospholipids	NP	NP	NP	Negative
Hemoglobinopathy study	NP	NP	NP	NP
H/o PE or DVT	No	No	No	Yes
Current Anticoagulant use	No	No	No	No
AKI Creatinine (mg/dL)	No	No	No	No

Beta 2 glycoprotein IgG, IgM	See anti-cardiolipin syndrome panel	See anti-cardiolipin syndrome panel	See anti-cardiolipin syndrome panel	Negative
INR	1.54	1.09	1.09	1.1
Complement factor 4 Normal range (10-45 mg/dL)	NP	Normal (14)	NP	NP
Complement factor 3 Normal range (80-180 mg/dL)	NP	Low (78)	NP	NP
ACE Normal range (14-82 U/L	NP	NP	NP	NP
Scleroderma-70 Antibody Normal range (<0.9AI)	NP	Normal (0.2)	NP	NP
Extractable Nuclear antigen antibody SM & RNP Normal range (<0.9AI)	NP	Normal (SM<0.2, RNP <0.5)	NP	NP
J0-1 antibody Normal range (<0.9AI)	NP	Normal (<0.2)	NP	NP
ACTH normal range (0-46 pg/mL)	NP	NP	Normal (20)	NP
AFP Normal range (0-10ng/dL)	NP	NP	Normal <1.3	NP
Dilute Russell Viper Venom time	NP	NP	NP	Negative

Abbreviations:

ACE= Angiotensin convertingenzyme
ACTH=Adrenocorticotropic hormone
AFB=Alpha Fetoprotein
Afib= atrial fibrillation
AKI= Acute Kidney injury
ANA= Anti-nuclear antibody
Anti-phospholipid syndrome panel (PT, INR, Anti-cardiolipin antibodies and antiBeta-2-GP1 antibodies)
b/l= bilateral
CCP= CyclineCitrullin Peptide
CMV= Cytomegaovirus
CRP=C reactive protein
DVT= deep venous thrombosis
EBV= Epstein-Barr virus
ECG= Electrocardiogram
Epstein-Barr virus panel (capsid IgM and IgG antibodies, VCA IgG, and Early antigen)
ESR= Erythrocyte sedimentation rate
HBV= hepatitis B virus
HCV= hepatitis C virus
HIV= Human immunodeficiency virus
INR= international normalized ration
NP= not performed
NSR= Normal sinus rhythm
PE= pulmonary embolism
PFO= persistent foramen ovale
PTH=Parathyroid hormone
TTE= Trans thoracic echocardiogram
TSH= Thyroid stimulating hormone
Urine toxicology [5 panel (Barbiturate, Benzodiazepine, cocaine, opiates, Methadone)and 7 panel (5 panel plus cannabinoid and Phencyclidine)]

patient's clinical status improved with ganciclovir, both in regards to pain and diarrhea, and she was subsequently discharged with pain management and outpatient follow-up appointments.

See table 1 for results of selected laboratory tests and other studies.

Case 2

A 26 year old female with a known history of rheumatoid arthritis (RA) or probable systemic lupus

erythematous (SLE) presented to the ED with burning LUQ pain with radiation to the epigastric area worsening over a 2 days prior to her ED visit. The pain was not related to food ingestion, but was exacerbated by deep breathing and laying in the right lateral decubitus position. It was alleviated by laying in the decubitus prone position. The patient reported nausea and non-bloody vomiting. She had been using adalimumab and methotrexate, but her medications were switched to certolizumab shortly before the abdominal pain started.

The rest of ROS was unremarkable with the exception of a cough for 1 month.. The patientsmoked marijuana for alleviation of her RA-induced pain, as the prescribed medications did not improve her joint pain significantly.

At presentation vital signs were as follows: blood pressure: 102/64 mmHg; heart rate: 90 beats per minute; respiratory rate: 16 breaths per minute; oxygen saturation: 99% on room air and temperature of 98.2 degrees Fahrenheit. Her physical examination was remarkable for left lower quadrant tenderness.

The ECG showed normal sinus rhythm, the CT scan of the abdomen and pelvis revealed peripheral foci of low attenuation in the spleen consistent with infarcts and fibrotic contraction of the spleen. The surgery team was consulted and recommended medical management.

The patient received antacid and pain medication, but due to persistent pain was advised that she should be admitted. She refused admission and left against medical advice (AMA). The patient has had multiple ED visits for similar pain since leaving the hospital.

Case 3

A 24 year old African American male with anxiety disorder presented to the ED for LUQ abdominal pain for approximately 4 days. The pain was non-radiating, intermittent, lasting up to 4 hours, and alternated between sharp, and dull and constant. He denied testicular pain, vomiting, diarrhea or fever. The patient reported episodes of palpitations recently. There was no trauma or fall.

The rest of ROS was unremarkable. The surgical history was remarkable for an umbilical hernia repair 14 years ago. Hehad no known drug allergies, denied substance abuse, but drank daily. The patient was given analgesics and antacids and was admitted for intractable pain.

At presentation, his vital signs were as follows: blood pressure: 126/71 mmHg; heart rate: 50 beats per minute; respiratory rate: 16 breaths per minute; oxygen saturation: 99% on room air and temperature of 97.9 degrees Fahrenheit. Physical examination was remarkable for right lower quadrant and right costovertebral angle (CVA) tenderness.

CT scan of the abdomen and pelvis demonstrated bilateral kidney infarctions, as well as pulmonary nodules and hepatomegaly. While in the hospital patient developed, T wave inversions in leads II, III and aVF, although Troponin and CK were negative, the patient was placed on telemetry to rule out arrhythmias. Cardiology consult recommended to stress testing. Echocardiogram was unremarkable and duplex of the lower extremities found no deep venous thrombosis (DVT). Vascular surgery did not have any recommendations. Meanwhile, the patient was started on enoxaparin and warfarin. The patient did not want to stay in the hospital any longer and left AMA.

Case 4

A 51 year old woman with a history of patent foramen ovale (PFO) and post-cesarean section pulmonaryembolism (PE) presentedto the hospital with LUQ pain. She had nausea without vomiting or diarrhea. She denied dizziness, recent fever or other illnesses, bloody bowel movements, dietary changes, or changes to her routine. The rest of the ROS was unremarkable. After her post-postpartum PE, the patient had taken anticoagulation for 6 months. She had a hypercoagulability work up at that time that was unremarkable, although it was unclear what tests were performed. She had had an intrauterine device placed in 2018. A trans-esophageal echo (TEE) with color flow doppler performed in June 2007 had confirmed that the patient had a PFO, an aneurysmal inter-atrial septum, and a small bidirectional shunt. She denied any known drug allergies, smoking or drinking alcohol as well as abuse of illicit drugs.

At presentation, her vital signs were as follows: blood pressure: 128/72 mmHg; heart rate: 76 beats per minute; respiratory rate: 17 breaths per minute; oxygen saturation 98% on room air and temperature of 97.9 degrees Fahrenheit.

Physical examination was remarkable for a holosystolic murmur heard throughout precordium (II/VI) and left upper quadrant/epigastric tenderness.

Her pain was treated with antacids and analgesics without significant improvement.

A CT scan of the abdomen and pelvis demonstrated multiple wedge-shaped regions of hypo-attenuation within the spleen. The patient was then admitted for intractable pain and splenic infarct.

The in-hospital echocardiogram demonstrated shunting at the atrial level consistent with a patent foramen ovale. Bilateral doppler ultrasounds of the upper and lower extremities showed no DVT. CXR showed no acute cardiopulmonary process. Her ECG showed normal sinus rhythm and no morphology suggestive of ischemia. The patient was found to have a profound iron-deficiency anemia with a ferritin of 5 ng/mL. She may have had a concomitant hemoglobinopathy and was started on iron supplementation therapy.

The patient was discharged in an improved condition to follow up with hematology and her primary care physician.

Discussion

Sources for embolic occlusion of solid organs or vasculature in the body can arise from multiple sources. Some sources include atrial fibrillation, hypercoagulable states such as malignancies or Protein C or S deficiencies, factor V Leiden, vasculitides or known underlying thromboembolic conditions that are inadequately treated and subsequently migrate to the arterial side in the presence of a persistent PFO. Other causes are local thrombus formation in the left ventricle or tumors with local vessel compression.

The most important aspect of approaching patients with flank, back or abdominal pain is to broaden the differential diagnosis and perform a systematic work-up in order to make the correct diagnosis. It is equally important to attempt to find the underlying cause for the emboli as to appropriately treat these patients to avoid future stroke and/or heart attack [1,2, 20-22]. A significant amount of attention has been paid to researching silent atrial fibrillation as a cause of cryptogenic stroke, and empiric anticoagulation of these patients may avoid a devastating future stroke [21-25]. Some of the causes for stroke are also responsible for the infarcts in the kidneys or spleen. The question is, should similar attention be given to patients who sustained these infarcts, especially the ones with multiple infarctions. Despite extensive work-up, the source for the emboli is unknown in most cases. Moreover, traditional diagnostic tests such as a Holter monitor cannot adequately detect paroxysmal afib or other causes of intermittent arrhythmias [26,27]. Recent studies suggest implantable loop recorders may be the best option to detect these paroxysmal attacks, and patients with spleen or kidney infarcts may benefit from similar testing when all other tests are non-diagnostic [27]. The kidney or splenic infarct may have a bigger impact on a patient's health at the time of the event or after the event, and furthermore, the next attack may cause a severe debilitating stroke, fatal myocardial infarction or organ failure from larger infarct. Advances in anticoagulation medications has resulted in oral anticoagulants that are effective, require no strict international normalized ratio (INR) check, and have fewer major bleeding side effects. These facts beg the question of whether or not these patients should be anticoagulated after their initial event. Finding the cause is unfortunately not always possible, despite extensive work-up. Afib is the most common cause of cardioembolism and it is the most common cause of ischemic stroke [20,28]. Similarly,silentafib may be the cause of solid organ infarcts, especially when all tests for hypercoagulability and vasculitides are non-diagnostic. Silent afib is believed to be an important cause of cryptogenic strokes and treatment with oral anticoagulants has been advocated [24,29,30]. Improved diagnostic tests such as implantable loop recorders have helped identify these silent afib episodes. Several studies have shown the existence of silent afib in patients without symptoms who have pacemakers or other implantable devices as well as in patients on follow-up after a stroke. Varying rates of silent afib were reported in these patients [27,31]. In addition, another cause of emboli is a venous source with arterial occlusion via movement through a PFO, where closure of the foramen was advocated, but 3 randomized clinical trials failed to show a benefit in doing so [22], making silent afib the the most likely cause of cryptogenic strokes and possible solid organ infarcts rather than a venous clot that crossed the PFO.

Atrial fibrillation is present in 20% of patients with ischemic stroke prior to the event [27]. Afib incidence increases with age, therefore, an older patient's afib may be the likely cause of embolic events [32], whereas in younger patients the incidence may be low, but silent afib may still occur and cause periodic emboli that may warrant anticoagulation after the first incidence of solid organ infarct, similar to that of patients with their first

cryptogenic stroke. All of our patients had nearly normal work-ups for hypercoagulable states and vasculitides. Their ECG's showed no pathology and no local mass were found in their CT scan. Two patients reported episodes of palpitations. This suggests the possibility of silent afib as a plausible cause of their emboli. One of the patients had an undifferentiated mass in their left ventricle but he never followed up. One patient had rheumatoid arthritis, while another had a previous DVT and PFO, but a normal hypercoagulabilitywork up. Assuming silent afib as a plausible cause for their infarcts, an approach similar to the management of stroke with oral anticoagulation might be beneficial for these patients.

Limitations

The patients did not follow up at regular intervals and some may have gone to other hospitals, so it was not obvious if they had any additional work-ups, including loop recordings. It was also unknown whether or not they were started on anticoagulation. In addition, the work-up for each patient varied based on the individual providers. Furthermore, their pain could well have been from gastritis or other causes. It would be difficult to prove that the pain was solely from the solid organ infarct, but given the persistency of pain despite treatment for gastritis it is reasonable to assume that the infarct was the cause of the persistent pain. It is also not obvious from chart review if the female patients were taking oral contraceptive pills or if all patients had long distance flights or travel.

Conclusion

These case reports highlight that flank pain and abdominal pain may be due to infarcts of solid organs and should be part of the initial differential diagnostic consideration, particularly in those patients whose pain does not respond to adequate pain medication. As most cases, despite extensive work up, have no obvious source of the emboli and it can be assumed that intermittent afib could be one of the possible causes. There is no clear recommendation nor guideline to start anticoagulation for these patients so far. Loop recorders may help identify intermittent afib and should be considered in these patients.

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