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Mimic of Acute Appendicitis: Acute Intestinal Ischemia Secondary to Mesenteric Venous Thrombosis: A Case Report Laser in Genito-urinary Syndrome of Menopause; A Review of Literature

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1. Abstract

Intestinal ischemia is a diagnostic dilemma often mimicking other conditions. A late diagnosis of such clinical entity may lead to delay in necessary treatment. This paper presents a case of a 34 years old male drug user from a tertiary hospital in Manila who presented with right lower quadrant pain. Patient was brought to the operating room with intraoperative findings of ischemic bowels, hence resection was done and post operatively started on heparinization. Primary consideration of an intestinal ischemia secondary to mesenteric venous thrombosis. The paper emphasizes the importance of a clinical suspicion of a patient with a history of drug use. Prompt intervention with surgery and early anticoagulation should be performed without any delays.

2. Introduction

Intestinal ischemia is a life-threatening condition that affects the small or large intestine. The etiologies of mesenteric ischemia are arterial occlusion, venous occlusion, and arterial vasospasm. Consequences of mesenteric ischemia include bowel ischemia and necrosis, sepsis, and death. Ischemia involving the small intestine is called mesenteric ischemia. Ischemia involving the large intestine is called colonic ischemia. Acute mesenteric ischemia refers to a sudden onset of compromised blood flow, possibly from an embolic process or a thrombotic process, most commonly affecting the superior mesenteric artery. It can be occlusive or non-occlusive. Venous thrombosis involves obstruction of the intestinal outflow tract. Non-occlusive mesenteric ischemia is a consequence of a low flow state due to a low cardiac output, intravascular volume, or vasopressor use [1].

2. Objectives

This case report aims to present a case of a Mimic of acute appendicitis: acute intestinal ischemia secondary to mesenteric venous thrombosis encountered at Dr. Jose R. Reyes Memorial Medical Center and to report the history, physical examination, diagnosis and management of this rare clinical entity.

3. Case Report

3.1. A. History

The patient is a 34 years old male coming in for a 2-day history of right lower quadrant pain. Two days prior, the patient experienced right lower quadrant pain after ingesting food, with no radiation to other quadrants. He described the pain as vague, with a severity graded as 2/10. The symptoms were associated with multiple episodes of nausea and vomiting (clear, occasionally purely composed of saliva), anorexia, however no fever, diarrhea, or blood-streaked stools. The patient denies changes in his bowel movement. The patient did not seek consultation nor self-medicate during symptom onset. Symptoms of abdominal pain, vomiting, and anorexia continued intermittently throughout the interim, with the same severity and localization. The patient denies similar symptoms of abdominal pain before the present occurrence. He denies food fear or

weight loss. He divulged that he used methamphetamine two days before the onset of abdominal pain. Few hours before the consult, the patient experienced worsening of the severity of the abdominal pain, with a severity of 8/10, still with right lower quadrant localization, which occurred 2 hours after ingesting a meal. He experienced multiple vomiting episodes. He denies diarrhea, hematemesis, fever. The patient then sought consult at our institution.

3.2. Past Medical History

The patient has no known co-morbidities, no diabetes, no hypertension, no asthma, no history of cancer. He has not undergone any surgical procedures, has no previous hospitalization, and is not currently taking any medications.

3.3. Personal and Social History

The patient is currently unemployed but was previously working as a contractual construction worker. The patient smokes 2-5 sticks of cigarettes per day for twenty years. He is an occasional alcoholic beverage drinker. The patient has a three-year history of illicit drug use (methamphetamine), amounting to three usages per week. His last intake was two days before the onset of symptoms. He denies polygamous relationships, only has sexual relations with his wife. He denies any history of intravenous drug use. Family history: no history of cardiac disease, diabetes, stroke, hypertension; he denies any history of thrombosis/bleeding in the family.

3.4. Physical Examination

At the emergency room, the patient was awake, alert, not in distress, with non-sunken eyeballs, moist lips. Vital signs were as follows: GCS 15, 100/70, heart rate of 76, respiratory rate of 20, temperature of 36.9, and oxygen saturation on room air of 99%. The patient has non-distended neck veins, with symmetric chest expansion, clear breath sounds, distinct heart sounds, regular heart rate, and regular rhythm. Abdominal examination showed a flabby, non-distended abdomen, with direct tenderness and rebound tenderness on the right lower quadrant associated with guarding. He had no hernial bulge on the inguinal area. The clinical working impression at this point was acute appendicitis. The operative plan was appendectomy using a Rocky-Davis incision. The symptoms remain unchanged prior to bringing the patient to the operating room (still with right lower quadrant tenderness), and there was no recurrence of vomiting. Grossly normal appendix and serosanguinous fluid were seen upon doing a Rocky Davis incision (Figure 1). Small intestinal segments were gangrenous. The surgeons proceeded with an exploratory laparotomy for better visualization of the visceral organs. Serous to sanguinous fluid was drained from the peritoneal cavity. Two separate segments of gangrenous jejunal segment were noted: [1] 10cm of length of jejunum, 20cm from the ligament of Treitz; and [2] 20 cm of length of jejunum, 50cm from the ligament of Treitz, and 200cm from ileocecal valve (Figure 2). The small bowel mesentery was thick with intact arterial pulsation. The bowels were resuscitated using a warmed crystalloid solution. No noted improvement in the bowel's color was noted. Segmental jejunal resection with end-to-end anastomosis, incidental appendectomy, and placement of Jackson-Pratt drain was performed. The working impression is acute mesenteric ischemia, possibly from a mesenteric venous thrombosis. Representative slides showed thrombosis on the vascular structures, mostly on veins and venues. Arterioles shows present of blood cells. Lymphocytic infiltrates are seen surrounding the vasculature as well as the surrounding connective tissues. The surrounding connective tissues are edematous and congested. The mesentery likewise shows presence of inflammatory cells. This can be compared to non-necrotic segments, which shows largely empty arteries, venules, and capillaries. It has scant inflammatory infiltrates.

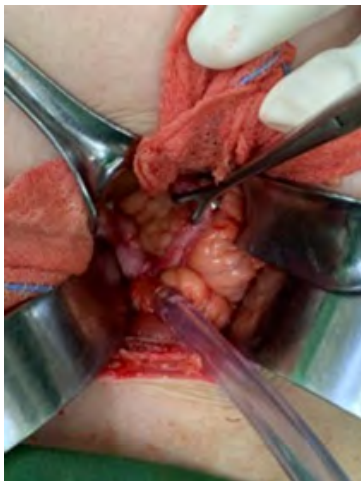

Figure 1: A grossly normal appendix was seen upon doing a Rocky-Davis incision. Serousto sanguineous fluid was observed.

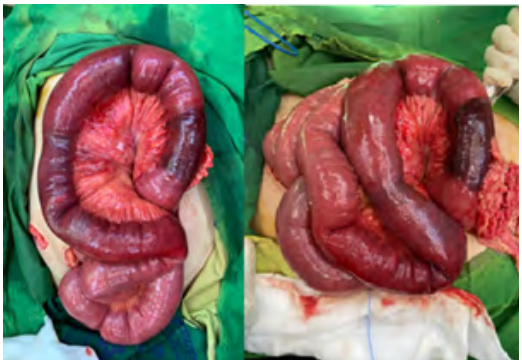

Figure 2: Two separate segments of gangrenous jejunal segment were noted: (1) 10cm of length of jejunum, 20cm from the ligament of Treitz; and (2) 20 cm of length of jejunum,50cm from the ligament of Treitz, and 200cm from ileocecal valve (Figure 2). The small bowel mesentery was thick with intact arterial pulsation.

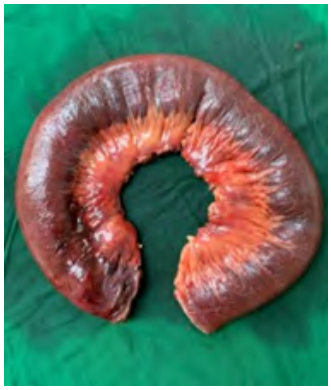

Figure 3: Resected jejunal segment.

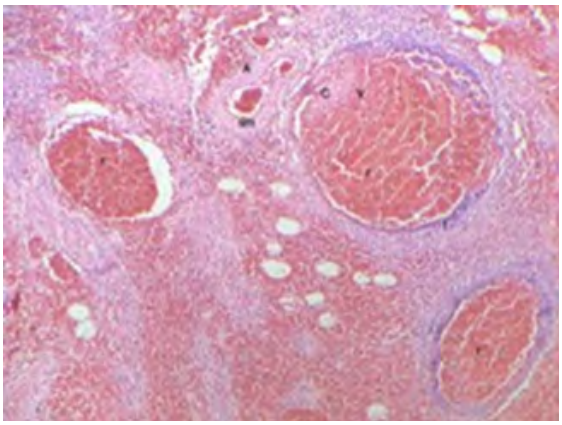

Figure 4: Presence of red blood cells (r) are seen in the venules (v). Thrombosis or clots (c) are likewise seen. The representative cut shows an arteriole (A) adjacent to thevenules filled with red blood cells.

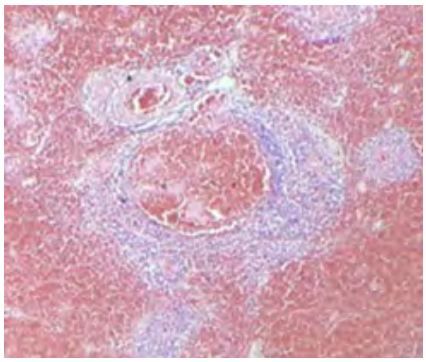

Figure 5: Clot formation (c) and red blood cells (r) are seen inside the venule (v) withsurrounding inflammatory infiltrates (l).

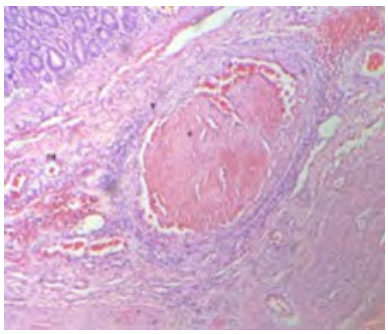

Figure 6: Clot formation (c) inside a venule. Capillaries (ca) are seen in the representativeslide.

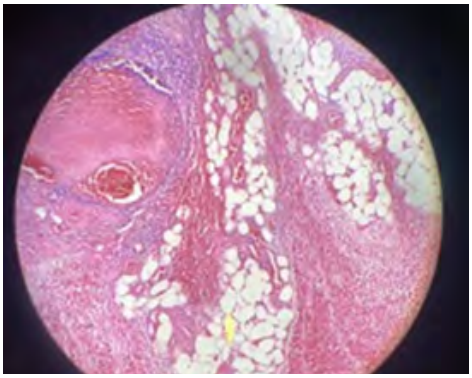

Figure 7: The mesentery shows inflammatory infiltrates (yellow arrow).

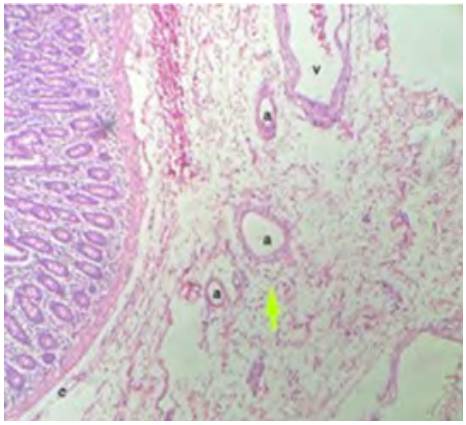

Figure 8: Representative slide on the non-necrotic segments of the jejunum. Arterioles, venues, and capillaries are largely empty, devoid of clots. Minimal inflammatory infiltratesare seen. Intestinal glands are unremarkable.

	Results
Hemoglobin	HB 15.9, Hct 46, WBC 22.3, Neut 81, Plt 208
Electrolytes	Na 136.1, K 4.3, Creatinine 76
PT/PTT	PT 14, PTT 32.5, 88%, INR 1.08
Urinalysis	pH 5.5, Sp Gravity 1.030, Trace glucose, Protein 1+, RBC 4,WBC1
Chest X-ray	Clear, no infiltrates/pneumonia

	Peak systolic velocity (cm/s)	Diameter (cm)
Aorta	73.7	1.31
Right externaliliac artery	113.0	0.53
Left internal iliacartery	117.3	0.61
Right externaliliac vein		0.69
Left external iliacvein		0.73

3.5. B. Course

Post-operatively, the patient was extubated and brought to the surgical ICU for close monitoring. A nasogastric tube was maintained for decompression and eventual feeding. He was co-managed with Behavioral Medicine for drug detoxification, and was given topiramate and bisperidone. The patient was also co-managed with the Vascular service. Given that the current working impression is mesenteric ischemia secondary to venous thrombosis, the patient was maintained on a heparin drip. Bleeding parameters were monitored while he was on the drip. The patient was initially started on total parenteral nutrition while he had nothing per orem. Cefuroxime and metronidazole was started. The patient was monitored closely for any signs of hemodynamic instability. Deterioration may suggest a re-occlusion of mesenteric vessels and the development of ischemia of the viable bowels. On the second day of SICU admission, the patient had flatus and had normoactive bowel sounds. The feeding tube was removed. The patient tolerated a general liquid diet. Heparin infusion was continued and was later bridged with warfarin. On the third day of SICU admission, the patient tolerated a soft diet. There was no hemodynamic instability. The patient had flatus and bowel movements with soft stools. Peritoneal drain output at this time was 130 cc serous. Because the patient was immediately started on anticoagulation treatment, testing for protein C, protein S, and antithrombin III deficiency was not performed. Furthermore, limited resources precluded the surgeons from requesting the said tests. They deemed it is more prudent to start the patient on anticoagulation immediately rather than wait for confirmation of these deficiencies. Delaying anticoagulation increases the risk of recurrence of thrombotic occlusion of mesenteric vessels. Echocardiography of the patient revealed a 58% ejection fraction, no regurgitation; no concentric left ventricular remodeling; with adequate contractility and systolic function; normal left atrium (<34 mL left atrial volume index); normal right ventricle with normal contractility and systolic function; normal pulmonary artery pressure. There was an absence any plaque formation in the carotid duplex scan that may be at high risk for rupture. Carotid and vertebral vasculature were of normal morphology. Normal anterograde blood flow was seen in both the carotid arteries and the vertebral arteries.

24-hour holter monitoring showed the absence of any rhythm and rate abnormalities. Aortic duplex scan showed normal morphology of the abdominal aorta, bilateral internal iliac artery, and inferior vena cava. No plaque formation, stenosis, nor occlusion were seen. Peak systolic velocity was not increased, suggesting absence of any stenosis or obstruction of blood flow. The following peak seak systolic velocity and diameters were observed: The patient was eventually discharged. The patient followed up twice with the surgeons afterward. There was no recurrence of abdominal pain. He was adequately feeding and had regular bowel movements. Warfarin dose was adjusted accordingly. Upon giving consent, he will be admitted into a drug rehabilitation facility. Long term plans also involves work up for thrombotic risk factors such as Protein C, ProteinS, or anti-thrombin.

III. This may be done once the patient stops taking anti-coagulation. Rheumatologic co- management may also be warranted.

4. Discussion

4.1. Introduction

The case presents a diagnostic dilemma. The clinical picture of the patient is characteristic of an acute appendicitis, however intraoperative findings revealed a case of an intestinal ischemia. Acute appendicitis may be diagnosed based on clinical findings, especially in outright cases of a few days history of right lower quadrant pain. Imaging is not necessary to diagnose acute appendicitis. The similar clinical presentation of the patient's condition to acute appendicitis precluded the authors to undergo further imaging, hence the true cause of the abdominal pain was not elucidated pre-operatively. Furthermore, the patient has no relevant personal or familial history of thrombotic risk factors that may lead to acute intestinal ischemia. A similar case report by Gaspary et al. in 2011 revealed a

case of a 21 year old female with an outright presentation of a right lower quadrant pain (appendicitis mimic) but imaging revealed a case of mesenteric venous thrombosis. However since the patient had a history of a gonadal vein thrombosis, the authors deemed it necessary to perform a CT scan for the patient. Intestinal ischemia is a life-threatening condition that affects the small or large intestine. The etiologies of mesenteric ischemia are arterial occlusion, venous occlusion, and arterial vasospasm. Consequences of mesenteric ischemia include bowel ischemia and necrosis, sepsis, and death. Ischemia involving the small intestine is called mesenteric ischemia. Ischemia involving the large intestine is called colonic ischemia. Acute mesenteric ischemia refers to a sudden onset of compromised blood flow, possibly from an embolic process or a thrombotic process, most commonly affecting the superior mesenteric artery. It can be occlusive or non-occlusive. Venous thrombosis involves obstruction of the intestinal outflow tract. Non-occlusive mesenteric ischemia is a consequence of a low flow state due to a low cardiac output, intravascular volume, or vasopressor use [11].

4.2. Pathophysiology

Ischemia is a consequence of either complete arterial obstruction, venous obstruction, or even combined arterial and venous obstruction (strangulation). Non-occlusive intestinal ischemia can be a consequence of shock states or drug intake. Severe vasospasm from these states overwhelms the intestinal vascular bed (mediated by angiotensin II and vasopressin). The clinical picture of mesenteric arterial embolism is characterized by a fast progression of ischemia and hemodynamic deterioration. It can progress over a few hours. The clinical picture of mesenteric venous thrombosis worsens over several days [12]. Chronic mesenteric ischemia, which is also called abdominal angina, is rare. Patients usually present with a history of postprandial abdominal pain. Occlusion is present in visceral arteries, but collateral circulation can prevent the progression into full-thickness ischemia. The occlusion leads to a lack of increased blood flow at stimulation, leading to severe postprandial pain. Patients suffering from chronic mesenteric ischemia usually complain of food fear and weight loss. Anorexia is not influenced, however [5]. A duplex scan may reveal increased peak systolic velocity and stenosis. Mesenteric ischemia etiologies may also be divided into: (1) a primary vascular cause,(2) extrinsic compression cause (e.g. volvulus, hernia, median arcuate ligament syndrome, retroperitoneal fibrosis), and (3) injurious or traumatic cause. The distinction between these different etiologies has implications in terms of the ideal diagnostic procedure. Primary vascular etiologies require angiography, vascular surgery, and anticoagulation. A secondary vascular cause can be diagnosed using an abdominal CT scan.

4.3. Epidemiology

The frequency of acute mesenteric ischemia is less than 1% of all acute laparotomies [12]. Arterial embolism and thrombosis account for 50% of and 15% of all acute mesenteric vasculopathy, respectively. Nonocclusive intestinal vasculopathy accounts for 20-30% of all cases [5]. Venous thrombosis accounts for 5-10% of cases of intestinal vasculopathy. It is most commonly encountered in the middle- aged and older adult patints, with equal gender distribution [6]. It In a comprehensive study from 1911 through 1984, only 372 cases of venous thrombosis were reported [1]. While only 53 acute cases and 19 chronic cases were reported during a 22-year study in the Mayo Clinic (Kaleya et al., 1989). Only 22 acute or chronic cases were reported at Montefiore Medical Center during a 20-year study [16]. The overall incidence with transmural intestinal infarction was estimated to be 1.8 per 100,000 person-years in Sweden between 1970 and 1982 [17]. In a study with a high autopsy rate location of 87%, it was found that 0.1% examined postmortem had MVT. 77% of these had transmural infarction which was believed to be the cause of death [2].

4.4. Clinical Features of Mesenteric Ischemia

As mentioned earlier, symptoms of mesenteric arterial obstruction progresses within a few hours. The clinical picture of venous thromboembolism progresses at a much slower pace [12]. Patient with symptom duration of less than four weeks are considered to have an acute mesenteric venous thrombosis, while those who an incidental finding of mesenteric ischemia on abdominal imaging are considered as chronic. Most patients have acute mesenteric venous thrombosis [13]. Irrespective of the etiology, there are stages to mesenteric ischemia: hyperactive stage, paralytic stage, fluid balance disarrangement, and shock. The hyperactive stage occurs after the immediate occlusion of mesenteric vessels. Patients usually complain of severe pain, diarrhea, vomiting, and hyperperistalsis [12]. The paralytic stage is characterized by symptoms of diminished, vague, and continuous abdominal pain. Fluid balance disturbances occur due to leakage of fluid, proteins, and electrolytes. In the final stage, patients suffer from clinical deterioration and progress into a shock state [12]. In most cases of mesenteric venous thrombosis, diagnosis is late due to the vague clinical picture [12]. It usually produces subtle and nonspecific clinical findings early, and develops outright abdominal symptoms when ischemia has progressed [5]. In a case series done by [9], patients with mesenteric ischemia from venous thrombosis complained of an average duration of pain of 8 days, intermittent in character and bearable in severity. The degree of mesenteric ischemia is dependent on the degree of vascular obstruction and if there is a presence of collateral flow. The area of ischemia of the intestine has a linear, one-to-one correspondence to the location and extent of occlusion. Upon opening, patients with mesenteric venous thrombosis usually have a limited segment of intestinal ischemia. According to [9,10]. Cases of mesenteric venous thrombosis have serosanguinous peritoneal fluid. Dark discoloration and edema of the intestinal walls are observed. The mesentery is thick with arterial pulsation.

Transition zones between normal or infarcted bowel are usually gradual [20]. Examined histopathologic features of patients presenting with mesenteric venous thrombosis. Venous systems may or may not be engorged, but all present with thrombosis. The arteries were largely devoid of thrombosis. There could be inflammatory cells surrounding the vasculature, usually lymphocytes or plasma cells, but some leukocytes of eosinophils may be present. Periarterial infiltrates are not seen. There could be necrotizing vasculitis of the muscular coats of the veins. Similar histopathologic findings are seen in the patient.

4.5. Risk Factors for Venous Thrombosis

Less than 50% of cases of ischemia secondary to venous thromboembolism have an identifiable cause. Some may be due to a hypercoagulable syndrome, which includes: protein C deficiency, Protein C and proteins S deficiency, factor V Leiden mutation, antithrombin III deficiency, and anti-cardiolipin antibodies. However, these patients present with previous episodes of thrombosis, such as recurrent deep venous thrombosis. In Clavien's case series, 5 out of 8 patients with primary venous mesenteric ischemia had prior episodes of deep venous thrombosis [9]. The typical manifestation of protein C or S deficiencies are leg thrombosis, cerebral, renal, axillary, portal vein, and pulmonary embolism. Most of the patients that live disease-free only develop symptoms during and after trauma, surgery, or pregnancy [23]. Vasculopathies may also cause mesenteric ischemia through the involvement of the mesenteric arteries. Buerger's disease typically occurs in young male smokers with occlusion of small to medium-sized distal arteries and veins of the upper and lower extremities [19]. Due to the smoking history of the patient, it is prudent to consider Buerger's disease. Small bowel involvement may be present in Buerger's disease. Most would present with significant peripheral vascular involvement before developing mesenteric involvement. Mesenteric ischemia as an initial presentation of Buerger's is extremely rare, with only five such cases published in the scientific literature [22]. Smoking more than 21 years was found to increase the peak systolic velocity in superior mesenteric arteries based on Doppler scan due to stenosis or plaque occlusion [23]. Other causes that are associated with systemic manifestation include anticardiolipin antibodies (peripheral thrombophlebitis, stroke, retinal artery occlusion, pulmonary thromboembolism, myocardial infarction, etc.), systemic lupus erythematosus, Polyarteritis Nodosa (necrotizing arteritis affecting many organ systems), Takayasu's disease (stenosis or dilatation of aortic arch and its major branches, as well as the mid-abdominal aorta), Behcet's disease (uveitis, aphthous stomatitis, genital ulcers, although vascular involvement is unusual), and Crohn's disease.

4.6. Methamphetamine use and Mesenteric Ischemia

Certain drugs are associated with mesenteric ischemia. These include digitalis, ergotamine, contraceptive, diuretics, cocaine, and pseudoephedrine. Pertinent to the history of the patient is the chronic use of methamphetamine. Methamphetamine use is associated with thrombosis of different sized vessels. Scientific literature only has data on its thrombotic effects on coronary arteries, cerebral vessels, and aorta. Mesenteric venous thrombosis was not reported. Methamphetamine use may be associated with hypertension, tachycardia, and vasoconstriction. It may lead to severe shearing forces, resulting in intimal injury, venous thrombosis, and subsequent ischemia [27].

5. Diagnostics

Early diagnosis of mesenteric venous thrombosis is achieved with a high degree of suspicion. A plain abdominal X-ray is unspecific. Gas in the bowel wall and mesenteric veins may be present in a plain abdominal radiograph in the later stages. Multidetector CT scan with IV contrast may also aid in the diagnosis by showing abnormalities in the bowel wall, the mesentery, and the vessels. It has a sensitivity in diagnosing mesenteric ischemia of 90% [28]. It is the most important, most available, and accurate diagnostic tool. However most clinicians do not suspect mesenteric venous thrombosis when they request for a CT scan [26]. Thrombosis in the portomesentlric system is usually seen with extension to the portal and splenic veins. Angiography may also aid in diagnosing mesenteric ischemia, although it can lead to delays in treatment. The reported sensitivity and specificity of CT angiography are 96% and 94%, respectively. A finding of pneumatosis intestinalis, venous gas, superior mesenteric artery occlusion, celiac and inferior mesenteric artery occlusion with distal SMA disease, or arterial embolism was 100% specific but only 73% sensitive. A finding of bowel wall thickening in addition to focal lack of bowel wall enhancement, solid organ infarction, or venous thrombosis was 50% sensitive and 94% specific [26]. Angiography may offer an additional therapeutic benefit through a local infusion of papaverine through the catheter. Lesions seen in angiography without functional disease are not indicative of reconstructive surgery [12]. D-dimer was elevated in a subset of patients in a case series by Acosta et al. however larger studies are needed to determine its actual value in clinical practice [3]. Thrombotic risk factors (Protein C deficiency, Protein S deficiency, or anti-thrombin III deficiency) can be investigated for after the operation. It was not determined post- operatively for this case because of three reasons: (1) the patient was started on anticoagulation, (2) the hospital has no resources for immediate testing for these deficiencies, and (3) the lack of identification of a personal or familial history of recurrent thrombosis, or a history of inciting factors that could trigger thrombosis (such as trauma or surgery) [23]. In a study by [24], post-operative levels of protein C were decreased and were maximal on the first day, lasting for seven days. Immediate administration of heparin may alter the levels of Protein S or C functionality, leading to a false-negative result for deficiency [26], in their study, suggested that Protein C and Protein S can be acquired after a thrombotic event, however should be

deferred if the patient is started immediately on anticoagulation. Repeated testing by the authors was done for confirmation three months after initiating treatment and at least fourteen days after cessation of the oral anticoagulants. Echocardiography was done to detect any cardiac wall abnormalities or valvular problems that may have caused an embolic process. 24-hour Holter was done to detect any paroxysmal atrial fibrillation since it is associated with an increased incidence of embolic processes. A duplex scan can be utilized as a screening tool for mesenteric ischemia and can be combined with a test meal. Abnormal findings, if present, may predict the recurrence of mesenteric ischemia. Duplex scan has the benefit of showing blood flow velocity and characteristics in the splanchnic or carotid vessels. Vascular anatomical status, vascular functional status, and vascular damage can be elucidated by this study. Peak systolic velocity can be increased by smoking or drug use, with slight elevation if PSV is 141-180 cm/s; moderate elevation if the velocity is 181- 220 cm/s, and severe elevation if velocity is > 220 cm/s [23]. A peak systolic velocity of more than 275 cm/s in the SMA identifies a >70% SMA stenosis with a sensitivity of 92%, a positive predictive value of 80%, and a negative predictive value of 99%. If negative for any increase in peak systolic velocity or stenosis, clinically important mesenteric artery stenosis is ruled out [22]. Full anticoagulation with unfractionated heparin should be started once diagnosis of a mesenteric venous thrombosis is made. This can be then converted to a Vitamin K antagonist such as warfarin. Lifelong treatment is recommended because of high recurrence rate of up to 36% without anticoagulation. However for patients with reversible causes, a 6 month duration of anticoagulation can be done. Indications for surgery for mesenteric venous thrombosis includes peritonisi, gastrointestinal bleeding, small bowel perforation, and intestinal stricture. If there is extensive bowel necrosis, thrombectomy with bowel resection, heparinization, and second-look surgery may be done. Alternatively, stapling off infarcted segments with skin-only closure of the abdominal wall can be done, and delayed reconstruction during second look surgery can be performed. If only a short segment is involved, bowel resection can then be performed. A second look surgery may be performed within 12-24 hours. The decision to perform second look surgery should be made at the first operation. There should be an early judgment of the questionability of the viability of the anastomosed bowels [12]. Regardless of the decision made in the first surgery, hemodynamic deterioration should persuade surgeons to bring the patient to the operating suite again. The value of second look surgery is that it reduces unnecessary bowel resection at the time of primary exploration, permitting salvage of segments with doubtful viability. The value of post operative imaging (CT scan) is unclear. However repeat scan after 6 months of anticoagulation therapy may be helpful in some patients where lifelong treatment is not considered. The indication for endovascular management of mesenteric venous thrombosis is unclear. Some centers advocate its use for patient in whom medical management has failed. Examples of endo vascular management include the following: percutaneous transjugular intrahepatic portosystemic shunting (TIPS) with mechanical aspiration thrombectomy; direct thrombolysis; percutaneous transhepatic mechanical thrombectomy; percutaneous transhepatic thrombolysis; thrombolysis via the SMA; thrombolysis via an operatively placed mesenteric vein catheter. Conservative management (anticoagulation without bowel resection) is indicated for patients who were diagnosed preoperatively and started on anticoagulation immediately. These patients should not have any episodes of clinical deterioration during observation. 30-day survival in a contemporary series was 70%. Prognosis is highly associated with underlying diseases, with cancer patients having the poorest survival [24].

6. Prognosis

Patients with mesenteric venous thrombosis usually have a protracted course as compared to patients with arterial occlusion as the cause of the intestinal ischemia. Mesenteric venous thrombosis has a mortality of 20-38%, which is decreased by early diagnosis and anti-coagulation. Mortality is increased for an acute presentation and presence of bowel ischemia [11].

7. Conclusion

The case highlights the diagnostic dilemma of an acute mesenteric ischemia in a patient with no obvious predisposition. Historical difficulty of early diagnosis of an acute intestinal ischemia has lead to increase mortality and morbidity. If there is doubt regarding the cause of abdominal pain, regardless if it presents as a mimic of another condition such as acute appendicitis, should warrant early investigations using imaging modalities. A high clinical suspicion can help with early identification of intestinal ischemia. Upon diagnosis, early anti-coagulation and appropriate surgical procedures should be performed without any delays. Relevant clinical history derived from the patient can help identify any risk factors. Drug use (methamphetamine) has rarely been discussed as cause of intestinal ischemia. This case highlights the importance of increased suspicion of the clinical entity if a history of drug abuse is elicited. Future studies that will discuss how exactly methamphetamine use can lead to a thrombotic risk factor leading to intestinal ischemia is warranted.

References

1. Abdu RAy, Zakhour BJ, Dallis DJ. Mesenteric venous thrombosis. Surgery. 1987; 101: 383-388.
2. Acosta S. Mesenteric venous thrombosis with transmural intestinal infarction: a population-based study. J Vasc Surg. 2005; 41: 59-63.
3. Acosta S. Epidemiology, risk and prognostic factors in mesenteric venous thrombosis. Br J Surg. 2008; 95: 1245-1251.
4. Blamey SL, Lowe GD, Bertina RM, Kluft C, Sue-Ling HM, Davies JA. Protein C antigen levels in major abdominal surgery: relationships to deep vein thrombosis, malignancy and treatment with stanozolol. Thromb Haemost. 1985; 54(3): 622-5.
5. Cappell MS. INTESTINAL (MESENTERIC) VASCULOPATHY I. 1998.
6. Gastroenterology Clinics of North America. 2020; 27(4): 783-825.
7. Akira Furukawa, Shuzo Kanasaki, Naoaki Kono, Makoto Wakamiya, Toyohiko Tanaka. American Journal of Roentgenology. 2009; 192: 408-416.
8. American Journal of Roentgenology. 2009; 192: 408-416
9. Clavien PA. Venous mesenteric infarction: A particular entity. British Journal of Surgery. 1998; 75(3): 252-255.
10. Cronenwett JL, Johnston KW. Rutherford's vascular surgery (9th ed.). Philadelphia, PA: Saunders/Elsevier. 2010.
11. Gaspary MJ, Auten J, Durkovich D, Gable P. Superior mesenteric vein thrombosis mimicking acute appendicitis. The western journal of emergency medicine. 2011; 12(2): 262-265.
12. Haglund U, Bergqvist D. Intestinal ischemia - the basics. Langenbeck's Archives of Surgery. 1999; 384(3): 233-238.
13. Jagannath Darshan, Emily Shaw, Bryan Green, Patrick J. Intestinal ischaemia and mesenteric necrosis in a heavy smoker, QJM: An International Journal of Medicine. 2013; 183-186.
14. Jagannath Darshan, Emily Shaw, Bryan Green. Intestinal ischaemia and mesenteric necrosis in a heavy smoker. 2013.
15. Kaleya RN, Boley SJ. Mesenteric venous thrombosis. In Najarian JS, Delaney JP (eds): Progress in Gastrointestinal Surgery. Chicago, Year Book Medical Publishers. 1989; 417-425.
16. Kazmers A. Intestinal ischemia caused by venous thrombosis. In Rutherford RB (ed): Vascular Surgery, ed 4. Philadelphia, WB Saunders. 1995; 1312-1320.
17. Kirkpatrick IDC, Kroeker MA. Biphasic CT with Mesenteric CT Angiography in the Evaluation of Acute Mesenteric Ischemia: Initial Experience. Radiology. 2003; 229(1): 91-98.
18. Mesenteric CT. Angiography in the Evaluation of Acute Mesenteric Ischemia: Initial Experience. Radiology. 2023; 229(1): 91-98.
19. Krupski WC, Selzman CH, Whitehill TA. UNUSUAL CAUSES OF MESENTERIC ISCHEMIA. Surgical Clinics of North America. 1997; 77(2): 471-502.
20. Jagannath Darshan, Emily Shaw, Bryan Green, Patrick J. Gadola, Intestinal ischaemia and mesenteric necrosis in a heavy smoker, QJM: An International Journal of Medicine. 2013; 106: 183-186.
21. Mitchell EL, Moneta GL. Mesenteric duplex scanning. Perspect Vasc Surg Endovasc Ther. 2006; 18(2): 175-83.
22. Rabl H, Fruhwirth H. Die klinische Bedeutung von Protein-C- und -S-Mangelzuständen für das chirurgische Patientenkollektiv [The clinical importance of protein C and S deficiency for surgical patients]. Langenbecks Arch Chir. 1992; 377(2): 75-80.
23. Rhee RY, Gloviczki P, Mendonca CT, Petterson TM, Serry RD, Sarr MG. Mesenteric venous thrombosis: still a lethal disease in the 1990s. J Vasc Surg. 1994; 20(5): 688-97.
24. Mesenteric venous thrombosis: still a lethal disease in the Disease in the 1990s. J Vasc Surg. 1994; 20(5): 688-97.
25. Rodger MA, Carrier M, Gervais M. Normal Functional Protein S Activity Does Not Exclude Protein S Deficiency. Pathophysiology of Haemostasis and Thrombosis. 2003; 33(4): 202-205.
26. Zou, Xiaojing, Huang, Haiyan MNa. Methamphetamine consumption and life-threatening abdominal complications, Medicine. 2018; 97: 18-647.
27. Medicine: May. 2018; 97: 18 - 0647