

Isolated acute renal infarction in an obese patient

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Abstract

Background: Uncontrolled cardiovascular (CV) risk factors is been related to a higher incidence of atherosclerosis. Obesity itself could predispose to significant cardiac disease including arterial atheromatous leading to acute events.

Case: A 46 years old obese patient who presented with left abdominal pain radiating to the left quadrant and left testicle. No significant abnormality was observed in the laboratories, but imaging showed left renal lower pole may be due to renal infarct (Figure 1), and CT-Angiography (CTA) of the abdomen showed infarction of the anterior two-thirds of the lower pole of the left kidney (Figure 2) secondary to occlusion of the supplying small inferior segmental artery and minimal atherosclerosis. Transthoracic Echocardiogram (TTE) showed a density in the LVOT may be artifact vs. calcification (Figure 3). A transesophageal echocardiogram (TEE) showed Grade III atherosclerotic changes of descending aorta (Figure 4), aortic arch, and a mobile plaque seen in the thoracic aorta.

Conclusion: Cardiovascular risk factors have been associated with multiple vascular complications. Obesity as a single cardiovascular risk factor is associated with advanced arterial disease; our case is an example of an unstable atheromatous lesion causing thrombosis and ischemia in the kidney in a patient without another risk factor for thrombosis.

Keywords: Atheroma, Renal Infarct, Ischemia.

Introduction

Uncontrolled cardiovascular (CV) risk factors are related to a higher incidence of atherosclerosis [1].

In the United States, more than 80 million patients have cardiovascular disease and the role of the CV risk is evident in the literature [2,3]. It is important to understand the high risk of uncontrolled modifiable risk factors like obesity lead that may lead to CV disease or worsening of the existent condition [4].

Obesity is classified as one of the major CV risk factors due to its proven increase in atherosclerosis and heart disease [5].

The arterial disease is caused by atherosclerosis, which could be stable or unstable [6]. An unstable atherosclerotic plaque would be a high risk for major ischemic events, including myocardial infarction due to its rupture, but its consequences would depend based on its location [7].

Obesity itself could predispose to significant cardiac structural

abnormalities, vasculopathy, and cardiac events [8]; but its progression is usually associated with other metabolic conditions like diabetes or lipid disorders [9]. Our patient is an exceptional case of a young patient with an ischemic event due to a unstable atherosclerotic plaque with obesity as a single CV risk factor.

Case

A 46 years old obese previously healthy Hispanic male, admitted due to acute progressive left abdominal pain radiated to the left quadrant and left testicle. No comorbidity was known and denied active tobacco or recent alcohol use or recreational drugs. During his admission, the CBC, CMP, CK was normal, A1c 6.1, LDH 232unit/L, HDL 48, LDL 156, Trig 173, total cholesterol 239, hypercoagulative tests were negative (Lupus anticoagulant, antiphospholipid, factor V, homocysteine, protein C & S, antithrombin III), SARS Ab negative, SARS PCR negative, urine toxicology was negative.

CT abdomen showed left renal lower pole may be due to renal infarct (Figure 1), laceration or nephritis; the patient was placed

on enoxaparin and CTA abdomen was done and showed infarction of the anterior two-thirds of the lower pole of the left kidney (Figure 2) secondary to occlusion of the supplying small inferior segmental artery and minimal atherosclerosis. TTE showed HpEF pattern with density in the LVOT may be artifact vs calcification (Figure 3). A TEE was done to evaluate for cardioembolic source and showed Grade III atherosclerotic changes of descending aorta (Figure 4), aortic arch and a mobile plaque seen in the thoracic aorta. The scrotal US showed left varicocele and right microlithiasis. The CTA and MRI were done to localize the atheromatous plaque, but they did not show any plaque. The patient was discharged home with high intensity statin, aspirin and anticoagulation.



Figure 1: Non-contrast CT abdomen, Red arrow indicates the left renal lower pole renal infarct.

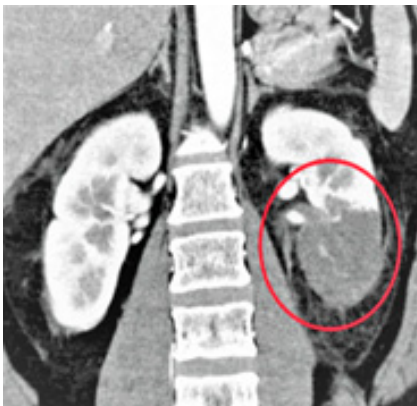


Figure 2: CTA Abdomen, Red circle shows the occlusion of the supplying small inferior segmental artery and minimal atherosclerosis causing the infarction of the anterior two-thirds of the lower pole of the left kidney.

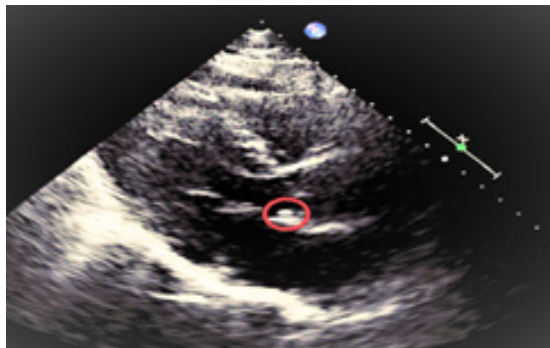


Figure 3: Transthoracic echocardiogram, Red circle shows the density in the LVOT (artifact vs calcification).

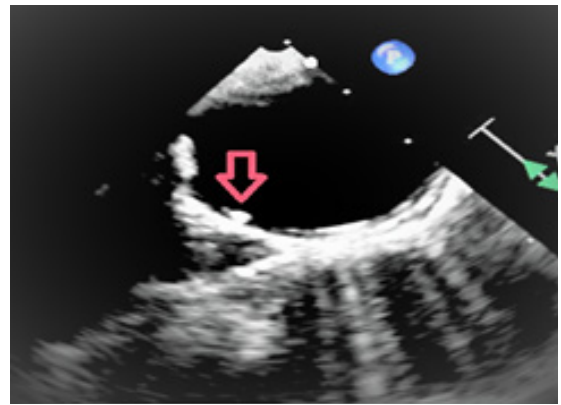


Figure 4: Transesophageal echocardiogram, Red arrow shows a grade III atherosclerotic changes of descending aorta.

Discussion

Aortic atheromatous plaque is defined as an irregular thickening of the intima, usually ≥ 2 mm, could be stable or unstable. Unstable is a non-homogeneous, spongiform plaque with mobility and ulceration, whereas the stable plaque is a homogeneous, non-mobile, calcified, and is not ulcerated [10]. Aortic atheromas usually occur in various areas of the aorta, while in ascending aorta and arch, it is extremely rare; it is most commonly found below the level of the kidney [11].

The progression of aortic atheromas is still not well understood. Unfortunately, an embolic event happens before the diagnosis of the atheroma, and this could affect the prognosis [10]. A proximal large aortic plaques is a main risk factor of stroke, it could lead up to 4 fold increased risk of stroke [12]. The Risk factors for aortic atheroma include advancing age, history of hypertension (HTN), hypercholesterolemia, increased body mass index (BMI), diabetes, and smoking [13]. TEE is the preferred modality for diagnosing a thoracic aortic atheroma, allowing a detailed view of the aorta and also permits quantification of atheromatous plaque in terms of thickness and stability [14], but the preferred studies would be angiography to identify and describe the plaque in better details [15,16].

Although multiple studies have investigated aortic atheromas, the mechanism for thrombosis is still not completely clear, but the ischemic event could be explained as the result of an unstable atheromatous plaque breaking [17]. A hypercoagulable state also needs to be ruled out when an unexplained ischemic event is present; it may be caused by the deficiency in the elements of the coagulation-anticoagulation system [18,19], due to autoimmune disease [20], hormonal therapy [21], immune reaction induced by heparin [22], or other mechanism affecting this system including malignancies [23,24].

Embolic events in the presence of atheromas in the aorta could be spontaneous [25,26], or provoked due to invasive interventions (Guidewire or catheter) [27], intra-aortic devices [28], and during cardiac and vascular surgery due to clamping or manipulations of the vessels [29]. The presence of mobile or protruding, with >4 mm in thickness plaques significantly increase the risk for an embolic

event [30].

The American Heart Association and the American Stroke Association (AHA/ASA) recommend antiplatelet and statin therapy in these patients. However, the role of surgery in these patients is not well defined. Besides endovascular therapy has been studied, destabilizing atheroma with catheter manipulation is well-founded [31]. Surgical approach would be limited to patients who are having cardiac surgery; however, it does not blunt the risk of embolic event during the surgery [32]. The AHA/ASA does not routinely recommend the routine surgical endarterectomy of aortic arch plaque for secondary stroke prevention [33].

Our case had a normal hypercoagulable or systemic disorder markers that would cause thrombosis. TEE was done to evaluate for cardio-embolic source and revealed the presence of an unstable atherosclerotic plaque. However, this plaque was not visualized on CTA and MRI, probably as it was too small. No other potential embolic sources were detected.

Conclusion

Cardiovascular risk factors have been associated with multiple vascular complications, besides chronic conditions like hypertension, dyslipidemia, diabetes, and kidney disease, which have been widely studied for their effect on the cardiovascular system. Obesity is also classified as a significant cardiovascular risk factor, and its incidence has been increasing, itself as a single cardiovascular risk factor is associated with the advanced arterial disease; our case is an example of thrombosis related to acute ischemia in the kidney in a patient without another risk factor for thrombosis.

References

1. Hajar R (2017) Risk Factors for Coronary Artery Disease: Historical Perspectives. *Heart views : the official J of the Gulf Heart Association* 18:109-114.
2. <https://www.heart.org/idc/groups/ahamah-public/>.
3. <https://www.texasheart.org/heart-health/heart-information-center/topics/heart-disease-risk-factors/>.
4. Sarah D de Ferranti (2019) Cardiovascular Risk Reduction in High-Risk Pediatric Patients: A Scientific Statement From the American Heart Association. *Circulation* 139:603-634.
5. Yoo HJ, Choi KM (2014) Adipokines as a novel link between obesity and atherosclerosis. *World J* 5:357-363.
6. Aloke V (2010) Finn, Masataka Nakano: Concept of Vulnerable/Unstable Plaque. *Arterioscler Thromb Vasc Biol* 30:1282-1292.
7. Yung-Chih Chen (2016) Atherosclerotic Plaque Rupture. Identifying the Straw That Breaks the Camel's Back. *Arterioscler Thromb Vasc Biol* 36:63-72.
8. Carbone S, Canada JM (2019) Obesity paradox in cardiovascular disease: where do we stand?. *Vasc Health Risk Manag* 15:89-100.
9. Tune JD, Goodwill AG (2017) Cardiovascular consequences of metabolic syndrome. *Transl Res* 183:57-70.
10. Alvarez C, Aslam HM (2018) A Large Grade 5 Mobile Aortic Arch Atheromatous Plaque: Cause of Cerebrovascular Accident. *Case rep medicine* 5134309.
11. Kojima K, Kimura S, Hayasaka K (2019) Aortic Plaque Distribution, and Association between Aortic Plaque and Atherosclerotic Risk Factors: An Aortic Angioscopy Study. *J Atheroscler Thromb* 26:997-1006.
12. Paul A Tunick, Barry P (1994) High risk for vascular events in patients with protruding aortic atheromas: A prospective study. *J American College of Cardiology* 23:1085-1090.
13. Pierre Amarenco, Ariel Cohen (1994) Atherosclerotic Disease of the Aortic Arch and the Risk of Ischemic Stroke List of authors. *N Engl J Med* 331:1474-1479.
14. Jansen Klomp WW (2016) Imaging Techniques for Diagnosis of Thoracic Aortic Atherosclerosis. *Int J Vascular Med* 4726094.
15. Kolossváry M, Szilveszter B () Plaque imaging with CT-a comprehensive review on coronary CT angiography based risk assessment. *Cardiovasc Diagn Ther* 7:489-506.
16. Kramer CM, Anderson JD (2007) MRI of atherosclerosis: diagnosis and monitoring therapy. *Expert review* 5:69-80.
17. Luis H Arroyo, Richard T (1999) Lee. Mechanisms of plaque rupture: mechanical and biologic interactions. *Luis H Arroyo, Richard T Lee. Cardiovascular Research* 41(2):369-375.
18. Hooda A, Khandelwal PD, Saxena P (2009) Protein S deficiency: Recurrent ischemic stroke in young. *Ann Indian Acad Neurol* 12:183-184.
19. Folsom AR, Ohira T (2009) Low protein C and incidence of ischemic stroke and coronary heart disease: the Atherosclerosis Risk in Communities (ARIC) Study. *J Thromb Haemost* 7:1774-1778.
20. Sun LL, Tang WX (2019) Clinical Manifestations and Mechanisms of Autoimmune Disease-Related Multiple Cerebral Infarcts. *Cell transplant* 28:1045-1052.
21. Henderson VW, Lobo RA (2012) Hormone therapy and the risk of stroke: perspectives 10 years after the Women's Health Initiative trials. *Climacteric* 15:229-234.
22. Ahmed M, Habis S (2019) Mesenteric Ischemia Caused by Heparin-induced Thrombocytopenia: A Case Report. *Cureus* 11:3900-3910.
23. Dardiotis E, Aloizou AM (2019) Cancer-associated stroke: Pathophysiology, detection and management (Review). *Int J Oncol* 54:779-796.
24. Suero-Abreu GA, Cheng JZ (2017) Then RKM Multiple recurrent ischaemic strokes in a patient with cancer: is there a role for the initiation of anticoagulation therapy for secondary stroke prevention? *Case. Reports* 2017:2016-218105.
25. Lyaker MR, Tulman DB (2020) Arterial embolism. *Int J Critical Illness Injury Science* 3:77-87.
26. Itzhak Kronzon, Paul A Tunick (2006) Aortic Atherosclerotic Disease and stroke. *Circulation* 114:63-75.
27. Colt HG, Begg RJ () Cholesterol emboli after cardiac catheterization. Eight cases and a review of the literature. *Medicine* 67:389-400.
28. Parissis H, Soo A, Al-Alao B (2011) Intra aortic balloon pump: literature review of risk factors related to complications of the intraaortic balloon pump. *J Cardiothoracic Surgery* 6:147-110.
29. El Zayat H, Puskas JD (2012) Avoiding the clamp during off-pump coronary artery bypass reduces cerebral embolic events: results of a prospective randomized trial. *Interact Cardiovasc Thorac Surg* 14:12-16.
30. Ariel Cohen, Christophe Tzourio, Bernard Bertrand, et al.: Aortic Plaque Morphology and Vascular Events. A Follow-up Study in Patients With Ischemic Stroke. *Circulation*. 1997, 96:3838-3841.
31. Yadav JS, Wholey MH, Kuntz RE (2004) Stenting and

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- Angioplasty with Protection in Patients at High Risk for Endarterectomy Investigators. Protected carotid-artery stenting versus endarterectomy in high-risk patients. *N Engl J Med* 351:1493-1501.
32. Powers WJ, Clarke WR, Grubb RL (2011) COSS Investigators: Extracranial-intracranial bypass surgery for stroke prevention in hemodynamic cerebral ischemia: the Carotid Occlusion Surgery Study randomized trial. *JAMA* 306:1983-1992.
33. Dawn O Kleindorfer (2021) Guideline for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack: A Guideline From the American Heart Association/American Stroke Association. *Stroke* 52:364-467.

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