Clinical Images and Case Reports Journal

Case Report | Vol 5 Iss 3

Hypertensive Urgency in the Setting of Markedly Asymmetric Systolic Blood Pressures

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Received: March 07, 2023; **Accepted:** March 20, 2023; **Published:** April 05, 2023

Abstract

Background: Hypertension is common with potentially severe complications of end-organ damage if not recognized, characterized, and managed early. Defining the cause of hypertension, primary versus secondary, is critical to preventing complications and is much needed to manage its treatment course accurately. Some secondary causes of hypertension can be reversible if recognized early.

Case Presentation: A 72 y.o male with a history of hypertension and hyperlipidemia presents to the ED with progressive chest pain and left arm weakness with asymmetric upper extremities. A detailed clinical vascular examination and a systematic diagnostic approach are carried out to reveal severe left subclavian artery stenosis. The interventional radiology team performed an endovascular correction of the stenosis with improvement in the patient's symptoms and hypertension.

Conclusions: In this case, a correctable cause of secondary hypertension in the elderly is depicted; it can be overlooked and treated as essential hypertension if not clinically astute.

Keywords: Hypertension; Essential hypertension; Secondary hypertension; Subclavian stenosis

Case Presentation

A 72-year-old Indian male with a history of hypertension and hyperlipidemia presented to the emergency department with intermittent mid-chest pain radiating bilaterally across the chest, epigastric pain, tingling, and weakness in his left arm, especially with his left arm prolonged use. His blood pressure was 260/90 in his right arm and 130/70 in his left arm.

On examination, his pulse was 75; his temperature was 97.9 °F (36.6 °C); his respiratory rate was 13, and his O_2 saturation was 98%. His BMI was 27.79 kg/m^2. His left radial and brachial pulse were non-palpable compared to 2^+ right radial pulses. Physical exam was negative for carotid brachial bruits, ulcers, gangrenous skin changes, or splinter hemorrhages of the digits. The heart exam also demonstrated a regular rate and rhythm without gallops or murmurs.

His respiratory exam showed clear breath sounds without wheezes or rales. The abdomen was soft and non-tender without bruits or palpable thrills. He did not exhibit any neurological motor or sensory deficits. The remainder of the review of systems was negative.

We treated the patient for hypertensive urgency in the ICU, targeting the right arm's high blood pressure (BP). Our initial goal of therapy was to reduce blood pressure by about 20-25% within the first 8 hours. We administered IV labetalol, which resulted in an improvement in blood pressure, and chest and epigastric pain. We then transitioned to an oral regimen of Labetalol, Hydralazine, and Norvasc and added Aspirin and a high-intensity statin. There were no signs of heart failure or stroke on clinical reassessment.

The EKG showed T wave abnormalities in the lateral leads with poor R wave progression. Q waves in the anterior leads, and the lack of ST elevations and serial negative troponin. CXR revealed no pulmonary vascular congestion. Echocardiography showed a preserved left ventricular ejection fraction and no valvular abnormality.

CT angiogram of the chest scan with contrast (figure 1) revealed calcification in the aortic arch that extended into the proximal great vessels, with some peripheral calcification extending into the descending thoracic aorta. Most notably, the scan revealed occlusion of the left subclavian artery near the point of origin, with reconstitution 2.8cm distal to the origin. The scans did not show an intimal dissection flap, double lumen, aortic dilation, and hematoma, and/or contrast leaks. An abdominal portion revealed extensive peripheral atheromatous disease with calcification in the celiac axis, superior mesenteric artery, and right and left common iliac arteries.

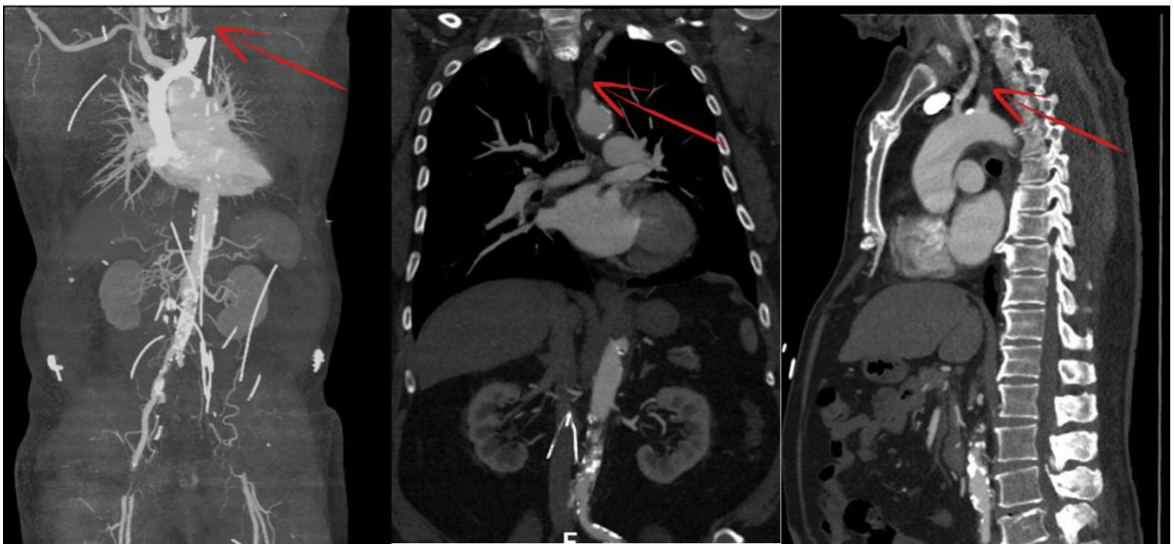

Figure 1: Coronal (left/middle) and sagittal (right) views on the CT angiogram revealed severe left subclavian stenosis (red arrows).

On hospital day 2, the patient's BP improved to 150-160/'80s, remaining asymptomatic. However, we could still not detect palpable left radial and brachial pulses and a 2^+ diminished right radial pulse. The lower extremities are warm and well-perfused bilaterally with normal, palpable popliteal, posterior tibial, and dorsalis pedis pulses-no evidence of ischemic changes in the upper or lower extremities bilaterally.

On hospital day 3, the patient was scheduled for an angiogram with an endovascular intervention for subclavian stenosis of the left arm and evaluation of the mesenteric axis.

Approach To Diagnosis

Marked differences in brachial blood pressures indicate several possible disease states, including obstructive processes that impinge on vessels (i.e., malignancy), peripheral vascular disease (PVD), subclavian steal syndrome (SSS), and acute aortic syndromes such as aortic dissection. In hypertensive urgency, as seen with our patient, one must consider alternative contributors to high blood pressure, such as primary and secondary hypertension (renal artery stenosis). Furthermore, one must watch for acute complications resulting from high blood pressure, such as myocardial infarction (MI), heart failure, and stroke. Thorough neurologic, cardiac, pulmonary, and vascular examinations at the bedside are imperative.

In our patient, the marked asymmetry of brachial BPs, in conjunction with a history of hyperlipidemia, hypertension, and symptoms of left upper extremity claudication, was highly suggestive of upper extremity PVD [1]. The patient denied headaches, dizziness, or syncope, thus making SSS less likely. He also denied any prior history of transient ischemic attack or stroke, had no apparent neurological motor or sensory deficits, and no gangrene or digital ulceration on the exam, thus decreasing the likelihood of thromboembolism or acute limb ischemia respectively. However, the presence of chest and left arm pain in the patient's history of hypertension and age was worrisome for an acute aortic syndrome such as aortic dissection or MI.

Aortic dissection should be considered in patients with brachial BP differentials of >20mmHg, and may also present with loss of peripheral pulses, as observed in our patient. Additional physical exam findings may include widened pulse pressure, aortic insufficiency, a diastolic murmur, muffled heart sounds (cardiac tamponade), syncope, altered mental status, and/or Horner syndrome [2]. However, aortic dissection classically presents with acute onset of severe, "tearing" chest pain. CT chest would note an intimal dissection flap, double lumen, aortic dilation, hematoma, and/or contrast leaks.

Subclavian artery stenosis is a less-common manifestation of PVD in about 2-7% of the general population [3]. Patients are often asymptomatic due to collateral circulation and insidious disease onset [4], making the disease process easy to miss until clinically severe. Symptomatic patients typically present with upper extremity claudication and/or vertebrobasilar insufficiency [5]. Upper extremity claudication is the most common presenting symptom and shows in about 57% of patients before endovascular treatment [6].

The standard screening procedure for subclavian stenosis involves bilateral brachial artery blood pressure measurements with a clinically significant difference of >15mmHg. Statistically, significant associations and risk factors include age, systolic blood pressure, smoking history, and HDL levels [3].

Discussion

Current guidelines suggest treating asymptomatic subclavian stenosis patients with antiplatelet agents, high-dose statins, and antihypertensives [7]. Management of subclavian artery stenosis varies based on clinical findings and patient presentation [8]. It is common for patients to present asymptomatically, with stenosis found incidentally. This can be explained by collateral circulation with the vertebrobasilar arteries. The presence or absence of symptoms is a crucial determinant of whether or not a patient should receive surgical intervention. Endovascular stenting remains the first-line intervention for symptomatic subclavian stenosis [9].

Conclusions

Atherosclerosis is the most common cause of subclavian stenosis [8], and subclavian stenosis is associated with increased cardiovascular disease (CVD) mortality and overall mortality [10]. Workup for underlying atherosclerosis should be performed, and long-term management of patients should align with secondary CVD preventative measures, including management of hypertension, dyslipidemia, glycemic control, smoking cessation, and antithrombotic therapy. Monitoring for continued cardiovascular disease risk progression through maintenance and further interventional management is essential. Systematic methods of diagnosing secondary causes of hypertension are crucial. In this case, a correctable cause of secondary hypertension in the elderly is depicted; it can be overlooked and treated as essential hypertension if not clinically astute. Once detected and treated successfully, it can avoid end-organ complications.

List of Abbreviations

CVD: Cardiovascular disease; **CT:** Computed tomography; **PVD:** Peripheral vascular disease; **BP:** Blood pressure; **SSS:** Subclavian steal syndrome; **MI:** Myocardial infarction; **ICU:** Intensive care unit; **ED:** Emergency department.

Authors' Contributions

Camille Briskin, MD: Writing, Editing, and Finalizing; **Steven Reichard, MD:** Expert radiologist images review, Opinion; **Bijo Chacko, MD:** Senior editor review; **Benson A. Babu, MD MBA:** Senior editor, Writing, Finalizing, and Supervision.

REFERENCES

1. Caesar-Peterson S, Bishop MA, Qaja E. Subclavian Artery Stenosis. [Updated 2021 Mar 3]. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing. 2021.
2. Levy D, Goyal A, Grigorova Y, et al. Aortic Dissection. [Updated 2021 Jun 17]. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing. 2021.
3. Shadman R, Criqui MH, Bundens WP, et al. Subclavian artery stenosis: prevalence, risk factors, and association with cardiovascular diseases. J Am Coll Cardiol. 2004; 44: 618-623.
4. Rodriguez-Lopez JA, Werner A, Martinez R, et al. Stenting for the atherosclerotic occlusive disease of the subclavian artery. Ann Vasc Surg. 1999; 13: 254-260.
5. Saha T, Naqvi SY, Ayah OA, et al. Subclavian Artery Disease: Diagnosis and Therapy. Am J Med. 2017; 130: 409-416.

6. Brott TG, Halperin JL, Abbara S, et al. ASA/ACCF/AHA/AANN/AANS/ACR/ASNR/CNS/SAIP/SCAI /SIR/SNIS/ SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American Stroke Association, American Association of Neuroscience Nurses, American Association of Neurological Surgeons, American College of Radiology, American Society of Neuroradiology, Congress of Neurological Surgeons, Society of Atherosclerosis Imaging and Prevention, Society for Car- diovascular Angiography and Interventions, Society of Interventional Radiology, Society of NeuroInterventional Surgery, Society for Vascular Medicine, and Society for Vascular Surgery. Developed in collaboration with the American Academy of Neurology and Society of Cardiovascular Computed Tomography. Catheter Cardiovasc Interv. 2013; 81: E76-E123.
7. Ochoa VM, Yeghiazarians Y. Subclavian artery stenosis: a review for the vascular medicine practitioner. Vasc Med. 2011; 16: 29-34.
8. Spittell, PC. (n.d.). Subclavian Steal Syndrome.
9. Mousa AY, AbuRahma AF, Bozzay J, et al. Anatomic and clinical predictors of reintervention after subclavian artery stenting. J. Vasc. Surg. 2015; 15: S0741-S5214.
10. Aboyans V, Criqui MH, McDermott MM, et al. The vital prognosis of subclavian stenosis. J Am Coll Cardiol. 2007; 49: 1540-1545.