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A Call to Action: Hypertensive Crises, Non-ST-Elevation Myocardial Infarction (NSTEMI), and Heart Failure in the Early Twenties

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Abstract

A 21-year-old obese male with multiple hypertensive crises was diagnosed with non-ST-elevation myocardial infarction (NSTEMI), leading to heart failure due to uncontrolled hypertension and medication noncompliance. The patient's morbid obesity likely contributed to undiagnosed chronic hypertension, increasing the risk of atherosclerosis and cardiovascular diseases. Morbid obesity leads to increased interleukin-6 levels, promoting plaque accumulation and rupture. Obesity also triggers a pro-inflammatory and prothrombotic state, characterized by elevated levels of serum high-sensitivity C-reactive protein (hs-CRP), plasminogen activator inhibitor 1 (PAI-1), and other cytokines. This inflammatory state contributes to atherosclerosis development and renders plaques more prone to rupture. Additionally, obesity has been shown to increase the size of coronary thrombosis once the plaque ruptures. Treating obesity is crucial for the patient's well-being and reduces the burden on healthcare systems and society. Establishing a strong physician-patient relationship is essential for motivating lifestyle modifications, which are often the primary treatment approach for obesity and its complications.

Categories: Cardiology, Internal Medicine, Nutrition
Keywords: inflammation and heart failure, hypertensive crisis, heart failure, non-st elevation myocardial infarction (nstemi), extreme obesity

Introduction

A hypertensive crisis is a sudden increase in blood pressure, typically with systolic blood pressure (SBP) >180 mmHg or diastolic blood pressure (DBP) >120 mmHg. Medication noncompliance and obesity are risk factors for this condition [1]. Hypertensive emergency and hypertensive urgency can result in severe complications including stroke, myocardial infarction, and heart failure, even in young adults [2].

Case Presentation

A 21-year-old obese (BMI of 49.5kg/m^2) and active male smoker without a significant medical or family history of premature coronary artery disease (CAD) presented to the emergency department with orbital cellulitis and high blood pressure (225/135). The initial electrocardiogram revealed signs of left ventricular hypertrophy (LVH) and troponin levels were elevated (0.041ng/ml) due to demand ischemia. Echocardiography showed grade II diastolic dysfunction with ejection fraction (EF) >60%, and thyroid stimulating hormone (TSH) and urinalysis were within normal limits. The patient was admitted for his chief complaint and successfully managed for hypertensive emergency. The patient was subsequently discharged on lisinopril and hydralazine with a scheduled outpatient visit.

Over the next three years, the patient had multiple uncontrolled blood pressure visits due to medication noncompliance and was admitted numerous times to the hospital with a chief complaint of chest pain. His BMI throughout this time remained in the range of morbid obesity. On a subsequent encounter, he presented to the emergency department with chest pain, dyspnea, nausea, vomiting, and visual changes. The patient denied any substance abuse. His lipid profile was normal with mild elevation in low-density lipoprotein (LDL) at 109 mg/dl. His glycated hemoglobin (HbA1c) was in the prediabetes range at 5.8%. The hypercoagulable profile was unremarkable. The initial electrocardiogram showed T wave abnormalities in lateral leads (Figure *1*), and troponin levels were elevated at 17 ng/ml and 23 ng/ml, respectively.

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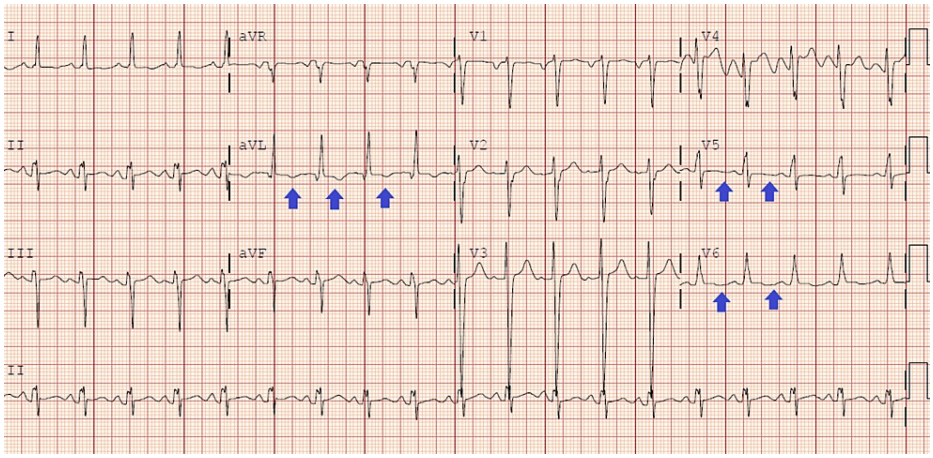


FIGURE 1: Electrocardiography showing T wave abnormalities in lateral leads (blue arrows)

The patient was diagnosed with non-ST segment elevation myocardial infarction (NSTEMI) and echocardiography findings showed combined systolic and diastolic dysfunction (EF=34%) with an apical mural thrombus (Figure *2*). Cardiac catheterization revealed an extensive clot in the posterior descending artery (PDA) and a thrombectomy was then performed (Figure *3*). The patient was started on dual antiplatelet therapy and apixaban. Screenings for secondary causes of hypertension including the aldosterone/renin ratio and urine metanephrines levels were unremarkable.

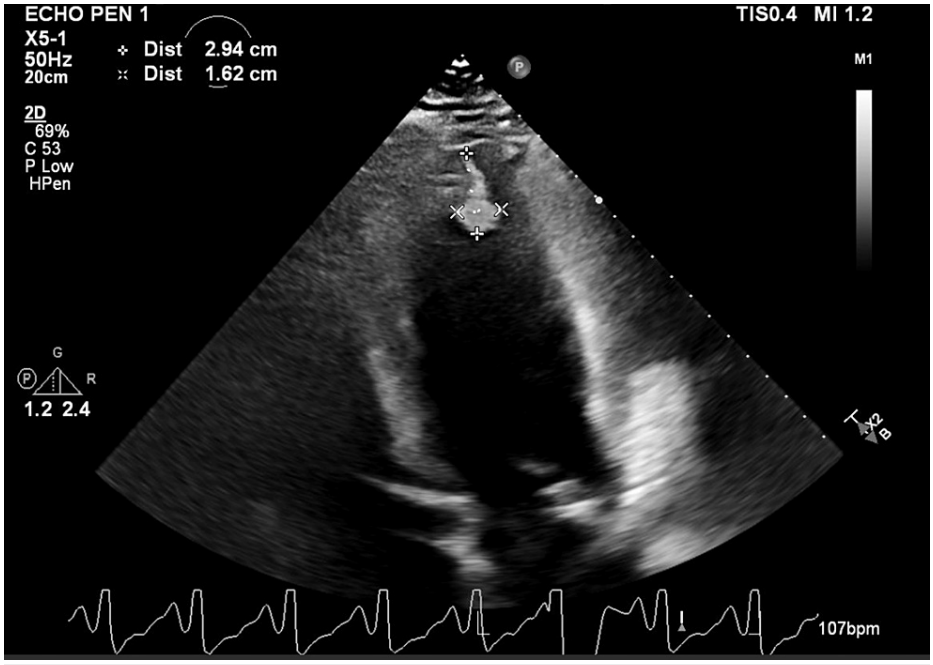


FIGURE 2: Echocardiography showing presence of apical mural thrombus

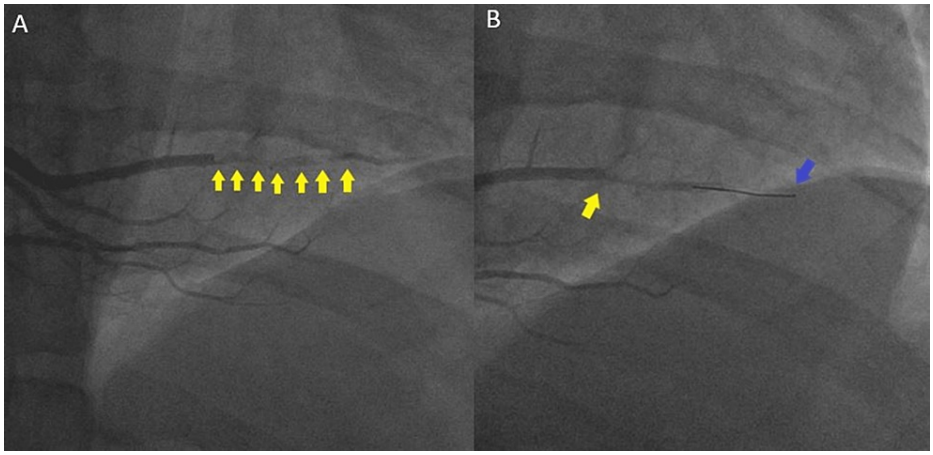


FIGURE 3: Cardiac catheterization. (A) PDA clot (yellow arrows); (B) PDA clot (yellow arrow) and coronary guidewire (blue arrow)

PDA: posterior descending artery

The patient was discharged in stable condition, on multiple anti-hypertensive medications, and has well-controlled blood pressure. At present, he reports compliance with his guideline-directed medical therapy and has been regularly seen by his cardiologist. His EF recovered in three months to >55%.

Discussion

Our case highlights the devastating complications that uncontrolled hypertension can cause in young adults. This man in his early twenties had undiagnosed chronic hypertension, which may have resulted from his morbid obesity. Research shows that young adults are more likely to have undiagnosed hypertension, even with regular primary care visits [3]. Unfortunately, his disease progressed and he experienced hypertensive crises, NSTEMI, and mixed systolic-diastolic heart failure due to medication noncompliance. Even in younger patients, increased interleukin-6 caused by morbid obesity has been linked to plaque accumulation and rupture [4]. His obesity and hypertensive shearing forces may explain the PDA clot. This case underscores the need for prevention and close monitoring of obesity, as well as patient education on medication adherence.

Obesity is an important risk factor for atherosclerosis. There is a 10% increase in the risk for atherosclerosis and coronary heart disease for each one-point increase in BMI above normal weight [5]. In obesity, there is a decrease in anti-inflammatory adipokines (adiponectin and omentum-1) and an increase in proinflammatory adipokines (retinol-binding protein 4, leptin, interleukin-6, monocyte chemoattractant protein-1, resistin), free fatty acids, and inflammatory cells. These factors lead to vascular inflammation and cell dysfunction, which contribute to the progression of atherosclerosis [6-8].

Increases in pro-inflammatory adipokines and fatty acids, resistance to insulin, and impairment in baroreceptor function trigger an increase in sympathetic nervous activation. Activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system is also promoted by increased leptin and leptin receptor signaling which is found in obesity [9,10]. Leptin also causes increased secretion of neprilysin from adipocytes and the kidneys, which causes a deficiency of endogenous natriuretic peptides in obese individuals with resultant additional sodium retention and plasma volume expansion. Increased concentrations of aldosterone, neprilysin, and leptin together with increased sympathetic nerve activity lead to hypertension, plasma volume expansion, sodium retention, vascular endothelial dysfunction, and ultimately atherosclerosis [11].

Obesity is associated with a pro-inflammatory and prothrombotic state, characterized by elevated levels of serum high-sensitivity C-reactive protein (hs-CRP), plasminogen activator inhibitor 1 (PAI-1), and numerous other cytokines [12-17]. This pro-inflammatory state contributes to the development of atherosclerosis and makes plaques more susceptible to rupture [18,19]. Furthermore, it is proposed that the prothrombotic state associated with obesity may increase the size of coronary thrombosis once the plaque has ruptured. As a result, obesity increases the risk of major adverse cardiovascular events (MACE) beyond what is expected based on traditional risk factors [20].

Evidently, obesity poses an important risk factor for the development of a wide array of diseases, especially

cardiovascular. Thus, treating obesity is crucial not only for the patient's well-being but also to reduce the socioeconomic burden on the healthcare system and society. A strong physician-patient relationship is crucial for establishing and maintaining a patient's motivation for lifestyle modifications, which are often the first line of treatment for obesity and its complications. Reducing sugar and saturated fatty acid intake can lead to a decrease in inflammation and endothelial cell dysfunction mediated by increased activation of pro-inflammatory cytokines. Consuming a Mediterranean diet, which includes increased consumption of fruits, whole grains, and vegetables, improves the quality of life for obese patients. In addition, it reduces triglyceride and total cholesterol levels and increases high-density lipoprotein (HDL) levels, thereby decreasing the risk for adverse cardiovascular events and often eliminating the need for lipid-lowering therapy [21].

Physical exercise is an important aspect of a successful obesity treatment plan in addition to dietary modifications. Moderate-intensity aerobic exercise, recommended for at least 150 minutes weekly, can yield satisfactory results for patients. Strength/resistance training can also improve weight loss and physical fitness by increasing lean body mass [21-23]. Moreover, exercise regimens combined with dietary changes can help improve other conditions such as diabetes and hypertension, which often coexist with obesity [24,25]. A thorough review of the patient’s medication list may contribute to favorable results since many drugs are associated with an increase in weight and may have appropriate alternatives that are not associated with such effects [26].

Pharmacological treatment can be considered for patients who don't achieve satisfactory results with lifestyle modifications alone. There are several classes of weight loss medications such as glucagon-like peptide (GLP) analogs, adrenergic drugs, and pancreatic lipase inhibitors, each with its unique adverse effect profile. The choice of medication should be based on the patient's preferences and the physician's consideration of the patient's distinctive characteristics.

In the event that the aforementioned treatment methods have proven to be ineffective, despite the person’s motivation for change, surgery may offer additional therapeutic modalities for such patients. Specifically, bariatric procedures such as a sleeve gastrectomy and gastric bypass surgery have proven to be efficacious in lowering visceral fat mass by around 40%, as well as improving insulin sensitivity and glucose metabolism, along with reductions in HbA1c levels in patients with type 2 diabetes [27-29]. These operations are associated with a relatively low risk of complications, the most common being anastomotic leak and fistula formation with the associated peritonitis, as well as more rare complications such as deep venous thromboses and thromboses in the portal vein system. As far as metabolic and nutritional complications are concerned, acid-base and electrolyte disturbances, along with certain nutritional deficiencies can be expected, with the most severe being hypoglycemia and osteoporosis [30-32].

Conclusions

Hypertensive crises and premature onset of the first NSTEMI are possible in young adults without known secondary causes. Excessive obesity is a significant risk factor for these conditions, highlighting the importance of prioritizing prevention and close monitoring of obesity in young patients. Additionally, medication compliance is crucial in managing hypertension and reducing the risk of cardiovascular events. Healthcare professionals should reinforce the importance of medication adherence to young patients to ensure optimal control of hypertension and minimize the risk of hypertensive crises and cardiovascular complications. Early detection and management of hypertension, along with lifestyle modifications and medication adherence, are critical for preventing these conditions and improving long-term cardiovascular outcomes in young adults.

Additional Information

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Saguner AM, Dür S, Perrig M, et al.: Risk factors promoting hypertensive crises: evidence from a longitudinal study. Am J Hypertens. 2010, 23:775-80. 10.1038/ajh.2010.71
2. Van Why SK, Pan CG: Primary causes of hypertensive crisis. Crit Care Clin. 2022, 38:375-91. 10.1016/j.ccc.2021.11.016
3. Johnson HM, Thorpe CT, Bartels CM, et al.: Undiagnosed hypertension among young adults with regular primary care use. J Hypertens. 2014, 32:65-74. 10.1097/HJH.0000000000000008
4. Madala MC, Franklin BA, Chen AY, et al.: Obesity and age of first non-ST-segment elevation myocardial

infarction. J Am Coll Cardiol. 2008, 52:979-85. 10.1016/j.jacc.2008.04.067
5. Hu F: Obesity and cardiovascular disease. Obesity Epidemiology. Hu F (ed): Oxford University Press, Inc., New York, NY; 2008. 174-95. 10.1093/acprof:oso/9780195312911.003.0009
6. Henning RJ: Obesity and obesity-induced inflammatory disease contribute to atherosclerosis: a review of the pathophysiology and treatment of obesity. Am J Cardiovasc Dis. 2021, 11:504-29.
7. Kawai T, Autieri MV, Scalia R: Adipose tissue inflammation and metabolic dysfunction in obesity. Am J Physiol Cell Physiol. 2021, 320:C375-91. 10.1152/ajpcell.00379.2020
8. Mouton AJ, Li X, Hall ME, Hall JE: Obesity, hypertension, and cardiac dysfunction: novel roles of immunometabolism in macrophage activation and inflammation. Circ Res. 2020, 126:789-806. 10.1161/CIRCRESAHA.119.312321
9. Weil BR, Westby CM, Van Guilder GP, Greiner JJ, Stauffer BL, DeSouza CA: Enhanced endothelin-1 system activity with overweight and obesity. Am J Physiol Heart Circ Physiol. 2011, 301:H689-95. 10.1152/ajpheart.00206.2011
10. Folli F, Kahn CR, Hansen H, Bouchie JL, Feener EP: Angiotensin II inhibits insulin signaling in aortic smooth muscle cells at multiple levels. A potential role for serine phosphorylation in insulin/angiotensin II crosstalk. J Clin Invest. 1997, 100:2158-69. 10.1172/JCI119752
11. Packer M: Leptin-aldosterone-neprilysin axis: identification of its distinctive role in the pathogenesis of the three phenotypes of heart failure in people with obesity. Circulation. 2018, 137:1614-31. 10.1161/CIRCULATIONAHA.117.032474
12. Chambers JC, Eda S, Bassett P, et al.: C-reactive protein, insulin resistance, central obesity, and coronary heart disease risk in Indian Asians from the United Kingdom compared with European whites. Circulation. 2001, 104:145-50. 10.1161/01.cir.104.2.145
13. Festa A, D'Agostino R Jr, Williams K, Karter AJ, Mayer-Davis EJ, Tracy RP, Haffner SM: The relation of body fat mass and distribution to markers of chronic inflammation. Int J Obes Relat Metab Disord. 2001, 25:1407-15. 10.1038/sj.ijo.0801792
14. Tchernof A, Nolan A, Sites CK, Ades PA, Poehlman ET: Weight loss reduces C-reactive protein levels in obese postmenopausal women. Circulation. 2002, 105:564-9. 10.1161/hc0502.103331
15. Sakkinen PA, Wahl P, Cushman M, Lewis MR, Tracy RP: Clustering of procoagulation, inflammation, and fibrinolysis variables with metabolic factors in insulin resistance syndrome. Am J Epidemiol. 2000, 152:897-907. 10.1093/aje/152.10.897
16. Mavri A, Alessi MC, Bastelica D, et al.: Subcutaneous abdominal, but not femoral fat expression of plasminogen activator inhibitor-1 (PAI-1) is related to plasma PAI-1 levels and insulin resistance and decreases after weight loss. Diabetologia. 2001, 44:2025-31. 10.1007/s001250100007
17. Mertens I, Van der Planken M, Corthouts B, Wauters M, Peiffer F, De Leeuw I, Van Gaal L: Visceral fat is a determinant of PAI-1 activity in diabetic and non-diabetic overweight and obese women. Horm Metab Res. 2001, 33:602-7. 10.1055/s-2001-17907
18. Morrow DA, Ridker PM: C-reactive protein, inflammation, and coronary risk. Med Clin North Am. 2000, 84:149-61. 10.1016/s0025-7125(05)70211-x
19. Blake GJ, Ridker PM: Novel clinical markers of vascular wall inflammation. Circ Res. 2001, 89:763-71. 10.1161/hh2101.099270
20. Grundy SM: Obesity, metabolic syndrome, and coronary atherosclerosis. Circulation. 2002, 105:2696-8. 10.1161/01.cir.0000020650.86137.84
21. Celik O, Yildiz BO: Obesity and physical exercise. Minerva Endocrinol (Torino). 2021, 46:131-44. 10.23736/S2724-6507.20.03361-1
22. Lopez P, Taaffe DR, Galvão DA, et al.: Resistance training effectiveness on body composition and body weight outcomes in individuals with overweight and obesity across the lifespan: a systematic review and meta-analysis. Obes Rev. 2022, 23:e13428. 10.1111/obr.13428
23. Petridou A, Siopi A, Mougios V: Exercise in the management of obesity. Metabolism. 2019, 92:163-9. 10.1016/j.metabol.2018.10.009
24. Kirwan JP, Sacks J, Nieuwoudt S: The essential role of exercise in the management of type 2 diabetes. Cleve Clin J Med. 2017, 84:S15-21. 10.3949/ccjm.84.s1.03
25. Sharman JE, La Gerche A, Coombes JS: Exercise and cardiovascular risk in patients with hypertension. Am J Hypertens. 2015, 28:147-58. 10.1093/ajh/hpu191
26. Tchang BG, Saunders KH, Igel LI: Best practices in the management of overweight and obesity. Med Clin North Am. 2021, 105:149-74. 10.1016/j.mcna.2020.08.018
27. Cogollo VJ, Rivera CE, Valera RJ, et al.: Improvement of glucose metabolism following rapid weight loss after bariatric surgery and its impact on reduction of visceral abdominal fat versus free fat muscle. Surg Obes Relat Dis. 2021, 17:933-8. 10.1016/j.soard.2021.01.031
28. Dadson P, Landini L, Helmiö M, et al.: Effect of bariatric surgery on adipose tissue glucose metabolism in different depots in patients with or without type 2 diabetes. Diabetes Care. 2016, 39:292-9. 10.2337/dc15-1447
29. Fink J, Seifert G, Blüher M, Fichtner-Feigl S, Marjanovic G: Obesity surgery. Dtsch Arztebl Int. 2022, 119:70-80. 10.3238/arztebl.m2021.0359
30. Schulman AR, Thompson CC: Complications of bariatric surgery: what you can expect to see in your GI practice. Am J Gastroenterol. 2017, 112:1640-55. 10.1038/ajg.2017.241
31. Jammah AA: Endocrine and metabolic complications after bariatric surgery. Saudi J Gastroenterol. 2015, 21:269-77. 10.4103/1319-3767.164183
32. Kassir R, Debs T, Blanc P, Gugenheim J, Ben Amor I, Boutet C, Tiffet O: Complications of bariatric surgery: presentation and emergency management. Int J Surg. 2016, 27:77-81. 10.1016/j.ijsu.2016.01.067