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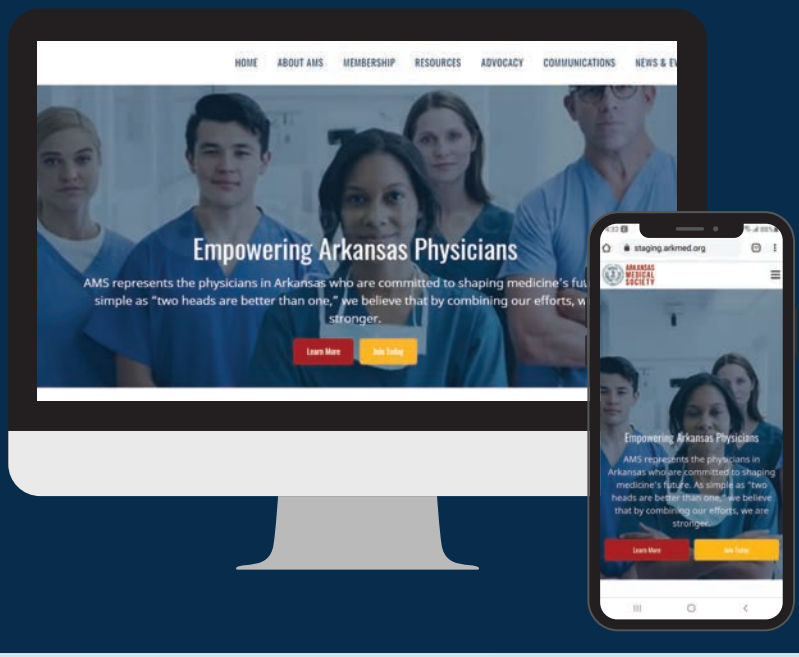

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by CHRISTA SUPER, OMS III[1]; MUHAMMAD WAQAS, MD[2]

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Case Report: A Patient with Lambda Light Chain Cardiac Amyloidosis

Abstract

Cardiac amyloidosis is a disease that occurs when a protein byproduct termed amyloid builds up within the heart. This build-up changes the heart's morphological structure and overall function. There are two subtypes of amyloid that have frequent cardiac involvement: light chain amyloidosis (AL) and transthyretin amyloidosis (ATTR). Of the patients diagnosed with cardiac amyloidosis, about 40% have AL amyloidosis, and 60% have ATTR. It is critical to diagnose which subtype a patient has, since both require specific treatment ranging from medications to stem-cell transplantation.[1]

AL pathophysiology is from the production of immunoglobulin light chain, most commonly from clonal plasma cell disorders like multiple myeloma.[2] ATTR occurs when a protein (transthyretin) from the liver becomes unstable and starts to misfold. These misfolded proteins form aggregates that build up and can deposit in the heart and other organs. Cardiac involvement is common and is the main prognosis factor for the two diseases.[3]

AL amyloidosis is a rapidly progressing and life-threatening disease. Over the past several years, the prognosis has improved due to advances in light chain suppressive therapies.

Case Presentation

A 69-year-old Caucasian female was presented as a hospital transfer for severe shortness of breath with exertion for the past year. The patient works as a respiratory therapist, and her symptoms impeded on her work and daily activities. Aside from her shortness of breath, the patient had no other complaints. The patient recalled contracting a viral illness back in the fall/winter of 2019, and soon after this illness, her health rapidly deteriorated.

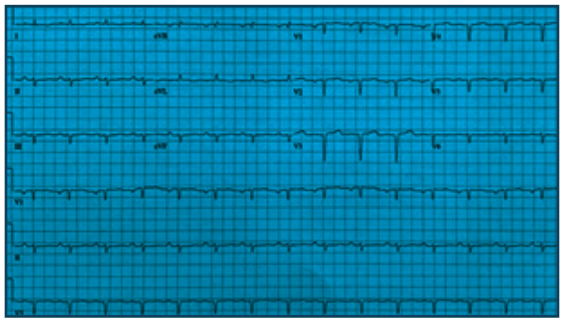

Figure 1. ECG

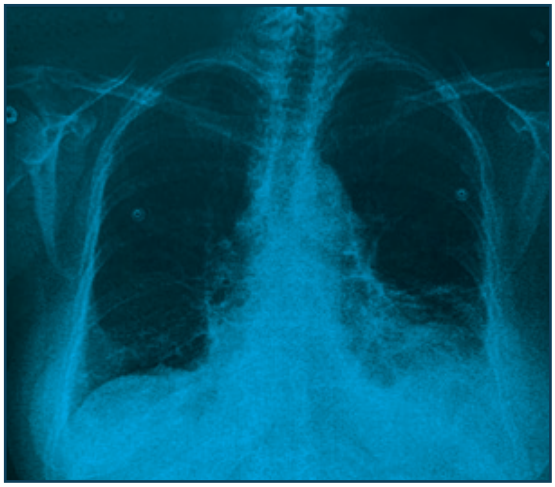

Figure 2. Chest X-Ray

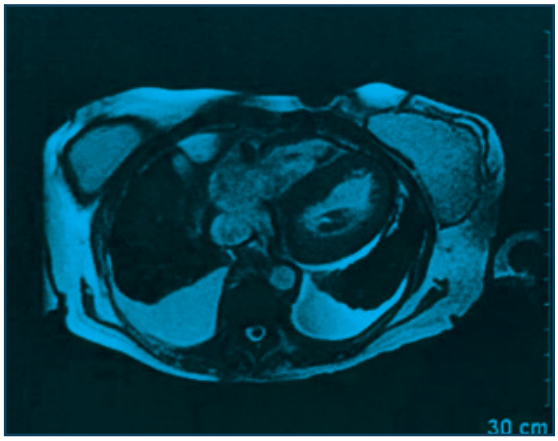

Figure 3. Cardiac MRI

After her viral illness, she was diagnosed with atrial fibrillation (AFib). Shortly after developing AFib, she suffered a cerebrovascular stroke.

At the hospital, the patient was diagnosed with diastolic heart failure, with preserved ejection fraction (HFpEF) at 45-50%. She was classified as being New York Functional Class III. Echocardiogram showed an enlarged heart with severe, concentric LV hypertrophy. Echo also revealed severe right-ventricular dysfunction. ECG showed normal sinus rhythm, low voltage QRS, and left atrial enlargement (Figure 1). Right-heart catheterization showed elevated biventricular filling pressures and mild pulmonary hypertension associated with WHO Group 2. Patient also had a reduced Fick cardiac output and index. Her V/Q scan ruled out pulmonary thromboembolism. Chest x-ray showed bilateral lower lobe densities, a small pleural effusion, and congestive heart failure (CHF) atelectasis (Figure 2). The patient also underwent a cardiac MRI that showed unremarkable pulmonary parenchyma, moderate pleural effusions, and global left ventricle hypertrophy, with a septal wall measuring 2.1 cm (Figure 3). MRI measurements reported: LVEDV 108 ml, LVESV 51 ml, EF 51%, LV mass 212 grams, and SV 57 ml.

Many tests were ordered based on the results of a low voltage ECG, significant left ventricular hypertrophy, and the patient's recent diagnosis of HFpEF. These tests were ordered to determine possible causes of her heart failure, focusing on cardiac amyloidosis. The tests included serum electrophoreses (SPEP), urine electrophoresis (UPEP), serum-free light chain (SFLC), and 99mTechnetium-Pyrophosphate (PYP). These tests helped to distinguish between AL amyloidosis and ATTR amyloidosis. SPEP and UPEP re-

vealed no M spike. The PYP scan showed the myocardium to contralateral lung ratio was quantified at 1.06, meaning that it was not likely to be ATTR amyloidosis. SFLC showed an elevated free lambda light chain, directing the diagnosis towards primary cardiac amyloidosis due to deposition of light chains.

More diagnostic tests were done to confirm the diagnosis. A fat pad biopsy and bone marrow aspirate were then collected. Fat pad biopsy was negative, but bone marrow aspirate showed a 10-15% increase in plasma cells. From here, the gold standard (cardiac biopsy) was performed to confirm the diagnosis. Cardiac biopsy showed mild myocyte hypertrophy, scant interstitial fibrosis, and interstitial and arteriolar deposition of congo red positive material. Lambda light chain deposits were seen along with amyloid features on electron microscopy. These findings were consistent with AL lambda-type amyloidosis.

Heart failure treatment was started to improve the patient's symptoms and overall outcome. The patient was started on spironolactone, bumetanide, and metoprolol.

Discussion

This patient presented with a classic case of lambda light chain amyloidosis. Her chief complaint of shortness of breath in the absence of chest pain and a negative V/Q scan was suggestive of another underlying condition. The patient's abnormal echo with a severely hypertrophied left ventricle was indicative of infiltrative disease. Cardiac magnetic resonance suggested cardiac amyloidosis features that included a diffuse decrease in T1 and T2 signal intensity and diffuse left ventricular filling, with a ventricular septal wall measuring 2.1 cm. Confirmatory evidence was still needed to determine what type of amyloidosis she had. A positive SFLC informed us that she did have an infiltrative disease. PYP and bone marrow biopsy were completed. PYP was negative, which indicated that it was not likely to be ATTR, but an endomyocardial biopsy was done to rule out ATTR completely. Bone marrow biopsy was positive for clonal expansion of plasma cells. Endomyocardial biopsy showed amyloid aggregates that were of lambda light chain origin. These results reinforce her diagnosis of AL amyloidosis.[4]

AL amyloidosis usually occurs in males over the age of 50 and typically involves one or more organs. It is vital to diagnose AL amyloidosis early because untreated patients with heart involvement have the most rapid disease progression. Diuretics are the primary drugs of choice for the patient's symptoms. Patients must follow up with a cardiologist for the management of their disease. Patients with AL amyloidosis in addition to cardiac involvement may be more sensitive than others to some cardiac drugs. For example, digoxin has cardiotoxic effects in amyloidosis patients. Also, calcium channel blockers and beta-blockers can be harmful to the patient, as they may lower blood pressure dramatically.

Specific goal-oriented therapy aims to halt the production of abnormal light chains by plasma cells. This can be achieved by a variety of chemotherapy drugs and other agents. With research underway, the goal of treatment of AL amyloidosis is moving toward patient-tailored therapy that might combine anti-amyloid treatments with anti-plasma cell chemotherapy.[5]

ATTR is a protein misfolding disorder where transthyretin, a protein made in the liver, deposits in the nerves and other organ tissues. There are two forms of ATTR amyloidosis: hereditary and wild-type. The hereditary condition of ATTR has a mutation of the TTR protein that can be passed on from one generation to the next. Wild-type does not involve abnormal DNA and cannot be passed down. Instead, as you get older, your TTR protein is prone to becoming unstable and misfolding. This form of amyloidosis presents the same as AL amyloidosis. However, treating ATTR is very different. ATTR silencers like patisiran and inotersen act on the liver to decrease the production of TTR. ATTR stabilizers stabilize the TTR protein and prevent it from folding like tafamidis and diflunsial. Fibril disruptors like doxycycline and green tea extract have been found to help break up and clear ATTR fibrils.[6]

By gaining information about the different cardiac amyloidosis types, physicians can better recognize the clinical signs and catch the disease early. This will help physicians develop multidimensional treatment plans and provide better patient education to those currently suffering from either AL amyloidosis or ATTR.

References

1. Hearteditor. Case Study in Cardiac Amyloidosis: Daunting but Ever More Treatable. Consult QD.
2. Witteles, R. and Liedtke, M., 2021. AL Amyloidosis for the Cardiologist and Oncologist: Epidemiology, Diagnosis, and Management. [online] JACC: CardioOncology.
3. Yamamoto H, Yokochi T. Transthyretin cardiac amyloidosis: an update on diagnosis and treatment. ESC Heart Fail. 2019Dec;6(6):1128-1139. doi: 10.1002/ehf2.12518. Epub 2019 Sep 25. PMID: 31553132; PMCID: PMC6989279.
4. Sofie B, Natallia L, Bernhard G, Rahel S, Frank R, J. FA. Cardiac amyloidosis. Cardiovascular Medicine. https://cardiovascmed.ch/article/doi/cvm.2018.00592.Published November 14, 2018.
5. Quarta CC, C. Cristina Quarta From the Brigham and Women's Hospital/ Harvard Vanguard Medical Associates Cardiac Amyloidosis Program (C.C.Q., Kruger JL, et al. Cardiac Amyloidosis. Circulation. https://www.ahajournals.org/doi/full/10.1161/CIRCULATIONAHA.111.069195. Published September 18, 2012.
6. ATTR Amyloidosis (Transthyretin): Types, Symptoms & Treatment. Cleveland Clinic. https://my.clevelandclinic.org/health/diseases/17855-amyloidosis-attr#:~:text=Unlike%20hereditary%20ATTR%20amyloidosis%2C%20wild,misfolds%20and%20forms%20amyloid%20fibrils.

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Additional Physician Organizations Support COVID-19 Vaccine for All Health Care Workers

On August 25, the Arkansas Medical Society Board of Trustees adopted a health policy statement encouraging physicians and health care workers to be champions of the vaccine in their communities. Since then, the following organizations have added their names to the list of supporters encouraging that all health care workers get the COVID-19 vaccine:

- The American Academy of Pediatrics, Arkansas Chapter
- The American Association of Physicians of Indian Origin (Arkansas)
- The American College of Cardiology, Arkansas Chapter
- The American College of Physicians, Arkansas Chapter
- The American College of Surgeons, Arkansas Chapter
- The Arkansas Academy of Family Physicians
- The Arkansas Dermatological Society
- The Arkansas Radiological Society
- The Arkansas Ophthalmological Society
- The Arkansas Orthopaedic Society
- The Arkansas Osteopathic Medical Association
- The Arkansas Society of Anesthesiologists
- The Arkansas Trauma Society
- The Arkansas Urologic Society

The statement says, "The physicians of Arkansas are deeply concerned about their patients and the citizens of our state as we continue to wage a battle with the COVID-19 virus and its highly contagious variants. Our ethical obligations, as well as the ethical obligations of most health care workers, teach us to always put patients first. To live up to that moral and ethical standard, it is imperative that we lead by example and take a stand in support of required vaccination for health care workers.

That ethical commitment demands that we take appropriate precautions to ensure that our fellow physicians and other health care workers are protected from the virus. In doing so, this will allow us to continue performing our patient care responsibilities while also protecting our families, our patients, and our communities. More critical, is the need to protect the more vulnerable citizens of our state, including unvaccinated children, hospital patients, residents of long-term care facilities and those who are immunocompromised.

For these reasons, the Arkansas Medical Society supports requirements for physicians and all other health care workers to become vaccinated against COVID-19.

The evidence of safety and effectiveness of the COVID vaccines is unmistakable and mounting. By August 2021, over 1 million Arkansans were fully vaccinated (over 163 million Americans), with minimal side effects. Even more striking is the fact that, according to the Arkansas Department of Health, 98% of COVID-19 hospitalizations in our state between January and July were among people who had not been vaccinated.

As physicians, we also recognize our responsibility to educate our patients and our communities on scientifically valid treatment protocols for patients testing positive for COVID-19 and provide factual and substantiated information on the prevention, diagnosis, and treatment of this deadly virus. As part of that effort, it is imperative that health care workers lead by example and get the vaccine. In doing so, not only do we preserve our long-term ability to be there for our patients, but we also set an example to the citizens of our great State of Arkansas that the vaccine is not only safe but – lifesaving."

by AMANDA EDERLE, MD

A healthy, 16-year-old boy is brought into clinic by his parents, who are concerned about a white ring that was recently noticed around a mole on his back. The parents state that over the last several weeks, white rings have developed around other preexisting moles. The parents are understandably concerned about the significance, if any, of these changes. Physical examination is significant for multiple pink-brown papules surrounded by an areola of hypopigmentation on the patient's back.

The next step in management should be:

A. Excision of all the moles
B. Biopsy of one of the moles for histologic evaluation to aid in diagnosis
C. Obtain scrapings for potassium hydroxide examination, as these changes likely represent tinea versicolor
D. Reassurance

Answer: D – Reassurance

The patient above has multiple halo nevi (Sutton's nevi). Halo nevi are melanocytic nevi that are surrounded by symmetric circular areas of hypo- or depigmentation. Halo nevi are not uncommon, occurring in about 1-5% of children and adolescents. The areola of pigment loss portends spontaneous regression of the surrounded nevus, usually a benign, common-acquired melanocytic nevus via a T-cell mediated immunologic cascade. Typically, halo nevi appear on the back or trunk, with multiple lesions being present in half of those affected. If the central nevus does not have features concerning for malignancy, such as asymmetry, dark non-uniform pigmentation, or irregular borders, biopsy is not warranted, and the provider may provide reassurance to patients. However, halo nevi are quite uncommon in middle-aged and older adults and may represent an immune response to a cutaneous or ocular melanoma, so thorough skin and eye examination in this demographic is imperative. Halo nevi tend to follow a stepwise progression: 1) development of an annular area of depigmentation; 2) the central nevus fades from brown to pink; 3) the nevus disappears completely, leaving behind a circular area of depigmentation; and 4) repigmentation gradually occurs. This process may take years, so it is important to educate patients on the length of the process. Because many patients with halo nevi have an increased number of common acquired nevi, total body skin examination is recommended.

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by SIJI THOMAS, MD; CAMERON SANDEFUR, DO; AMANDA SALE, MD; MAVEED YOUNIS, MD; MAX ARROYO, MD

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Myocardial Bridging

Abstract

Myocardial bridging(MB) refers to an anatomic variation in which major coronary arteries run in the sub epicardial adipose tissue intramurally. It usually involves left anterior descending artery. Long and multivessel myocardial bridging is uncommon. Here we present two cases of MB: a 60-year-old female, presented with chest pain, found to have MB involving four vessels and a 34-year-old male, found unresponsive on the street, with >4cm long LAD MB. Since MB can be seen in one-third of adults, a high index of suspicion is imperative to avoid treatment delay, even when presentation is subtle.

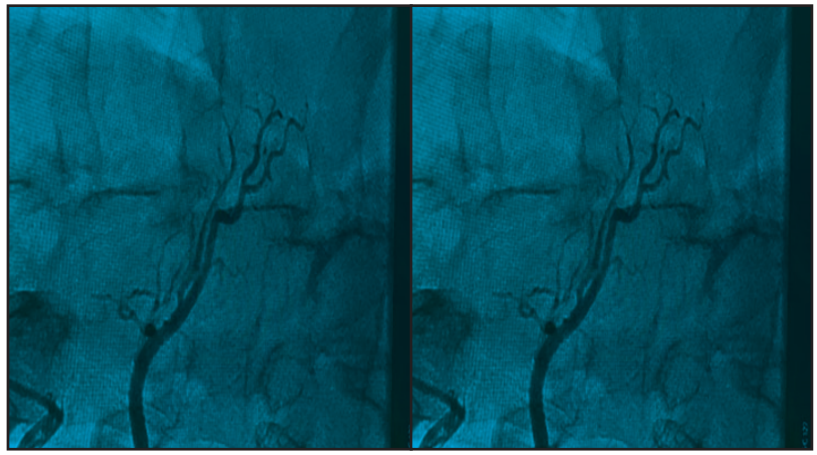
Introduction

The myocardial bridge (MB) is a normal anatomical variant under which a part of the major coronary arteries, usually running in the sub-epicardial adipose tissue, courses intramurally. It is almost exclusively situated in the mid-portion of left anterior descending coronary artery (LAD). Here we present a case series of rare presentations of myocardial bridging with regards to size, number, and clinical presentation. To the best of our knowledge based on review of literature, long LAD and multivessel myocardial bridging have not been reported.

Case Presentation 1

A 60-year-old Caucasian female presented to the ER with right-sided, achy chest pain that started about eight hours prior to presentation. It is worse with activities, has associated right-arm numbness, lasts about five minutes at a time, and no interventions seemed to help her pain. She was recently diagnosed with pericarditis and was started on colchicine, to which she was non-compliant given its side effects. Her surgical history includes cholecystectomy. Family history is significant for diabetes. No history of alcohol, smoking, or recreational drug use. She consumes caffeine occasionally. Her initial vital signs were temperature 98.3 F, pulse rate 64/min, respiratory rate of 15/min, BP 148/68 mm hg, and Sao2 100% on room air. Troponin I were 0.019 and 0.0030. 2D echo revealed EF of 60-65%, with trace pericardial effusion without evidence of tamponade. Coronary angiography revealed significant myocardial bridging of mid LAD, first obtuse marginal branch of circumflex, and two terminal branches of RPDA. Coronaries were non-obstructive. She was started on beta blockers and monitored closely.

Figure 1. Myocardial bridge involving terminal branches of RPDA.



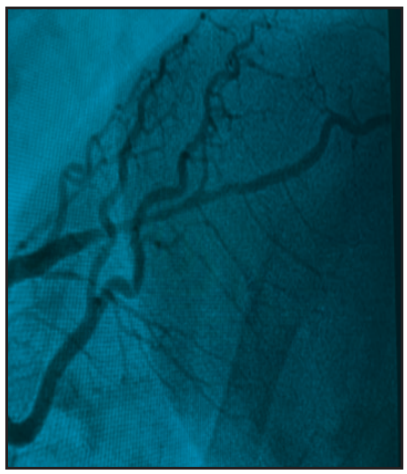


Figure 2: Long LAD myocardial bridge during contraction.

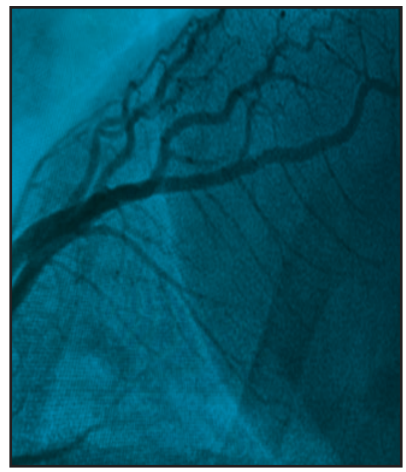


Figure 3: Post contraction. (Step down-step up phenomenon).

Case Presentation 2

A 34-year-old male found unresponsive on the road was brought to the hospital. Per the patient later, he had intermittent episodes of chest pain, with occasional dizziness and short-lived, fluttering palpitations. His past medical history included hypertension and hyperlipidemia. Family history is noncontributory. No history of smoking. Vital signs on initial presentation was BP 121/80 mm hg and HR 62/minute. Home medications included Lisinopril, pravastatin, and famotidine. Physical exam was unremarkable. CT head was negative for acute intracranial pathology. 12 lead EKG revealed NSR with inferolateral T wave changes concerning for LAD territory ischemia. CXR was unremarkable. His echo cardiogram revealed EF of 55%, LVH, and mild MR. He underwent coronary angiogram. He was found to have myocardial bridge of around 4 cm length involving mid LAD. Vessel was free of angiographic atherosclerosis. Medical management was recommended, including beta blockers, and he was discharged home with stable vital signs.

Discussion

Mohlenkamp et al., defines myocardial bridge as a muscle overlying intra-myocardial segment of an epicardial artery. Although generally a benign condition, myocardial bridging can be associated with myocardial ischemia. On average, myocardial bridges are present in one-third of adults. The rate of angiographic bridging is <5%. The prevalence also appears to be high in post-heart transplant patients and in patients with hypertrophic obstructive cardiomyopathy (HOCM). The current gold standard for diagnosing myocardial bridges is coronary angiography with the typical "milking effect" and a "step down-step up" phenomenon induced by systolic compression of the tunneled segment. Other attempted treatment modalities include the use of calcium channel blockers, stenting of tunneled segment, surgical myotomy, and CABG.[1] Corban et al. finds that myocardial bridges are commonly located over the left anterior descending artery.[2] Ishikawa and colleagues find that myocardial bridges suppress coronary atherosclerosis by altering endothelial permeability, which may be due to changes in hemodynamic force tending towards higher shear stress.[3] However, there have been no consistent associations described in the literature between atypical or angina-like chest pain and symptom severity, length or depth of tunneled segment, or degree of compression by the myocardial bridge. Schwarz et al., point out that in patients with myocardial bridges, administration of short-acting beta-blockers during atrial pacing alleviates angina symptoms and signs of ischemia.[4] Pratt and colleagues state that surgical approach is usually reserved for patients who are symptomatic and refractory to medical treatment.[5] Long-term prognosis in patients with isolated myocardial bridging is generally good. A study done by Kramer et al., in Ohio revealed the rate of incidence of myocardial bridging to be 12% in 658 normal angiograms performed. The same study found that five-year survival in 81 subjects aged 46 years was 97.5%, with neither of the two deaths related to the myocardial bridge[6]. A long-term follow-up study done by Juilliere et al., found that among 7,467 consecutive coronary angiograms performed during an eight-year period, 61 patients had a myocardial bridge of the left anterior descending coronary artery. The overall prevalence of myocardial bridges was 0.82%.[7] A descriptive cross-sectional study done by Saidi et al., attempted to better study the morphology of myocardial bridges by dissecting 109 adult hearts. Myocardial bridges were found in 40.4% of the hearts, most commonly in the left anterior descending artery (LAD). The average length of the bridges was 22.66 +11.94 mm while the depth was 1.83+ 0.98mm, with only 11% being long (34.87mm - 50mm) and 9% of them being deep (3.46mm - 5.00mm). The tunica intima was thickest proximal to and thinnest under the myocardial bridge. The same study revealed that myocardial bridges were found in 44 (40.4%) hearts (32 males, 12 females). About 84% of the bridges were found in the middle third of the left anterior descending. Other bridged vessels included the right coronary artery (6.8%) and the left circumflex, arteria ramus intermedius. The posterior interventricular and the right marginal arteries had one bridge each (2.3%).[8] As described by Yukio, et al. knowledge about myocardial bridge is relevant because coronary vasospasms can result from increased contractions as seen in physical activity or increased size and as seen in hypertrophic cardiomyopathy.[9]

Conclusion

Myocardial bridging can present with multiple variations. Patient symptomatology can also vary ranging from asymptomatic presentation, angina, syncope, and sudden death. When obvious cardiac and non-cardiac causes of chest pain do not explain patient's symptomatology, a high index of suspicion for anatomical coronary variations should be kept in mind. Subtle EKG changes and pain relief with beta blockers may help point towards structural variations. Early cardiology referral and timely coronary angiogram help prevent adverse patient outcomes.

References

1. Möhlenkamp S, Hort W, Ge J, Erbel R: Update on myocardial bridging. Circulation. 2002, 12:2616-22. doi10.1161/01.cir.0000038420.14867.7a. PMID: 12427660
2. Corban MT, Hung OY, Eshtehardi P, et al.: Myocardial bridging: contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. J Am Coll Cardiol. 2014, 63:2346-55. doi10.1016/j.jacc.2014.01.049
3. Ishikawa Y, Ishii T, Asuwa N, et al.: Absence of atherosclerosis evolution in the coronary arterial segment covered by myocardial tissue in cholesterol-fed rabbits. Virchows Archiv430. 163-171.
4. Schwarz ER, Klues HG, vom Dahl J, et al.: Functional, angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: effect of short-term intravenous beta-blocker medication. J Am Coll Cardiol. 1996, 27:1637-16. doi10.1016/0735-1097(96)00062-9. PMID: 8636548.
5. Pratt JW, Michler RE, Pala J, et al.: Minimally-invasive coronary artery bypass grafting for myocardial muscle bridging. Heart Surgery Forum. 1999, 2:250-253.
6. Kramer JR, Kitazume H, Proudfit WL, et al.: Clinical significance of isolated coronary bridges: benign and frequent condition involving the left anterior descending artery. Am Heart J. 1982, 103:283-288. oi10.1177%2F021849239700500410
7. 7. Juillière Y, Berder V, Suty-Selton C, et al.: Isolated myocardial bridges with angiographic milking of left anterior descending coronary artery: a long-term follow-up study. Am Heart J. 1995, 129:663-665. doi10.1016/0002-8703(95)90312-7.
8. 8. Saidi, H., Ongeti, W. K., & Ogeng'o, J. (2010: Morphology of human myocardial bridges and association with coronary artery disease. African health sciences. 10:242-247.
9. 9. Yukio I, Yoko K, Ehiichi K, et al.: (1) coronary events caused by myocardial bridge. Annals of vascular diseases. 2009, 2:79-94.

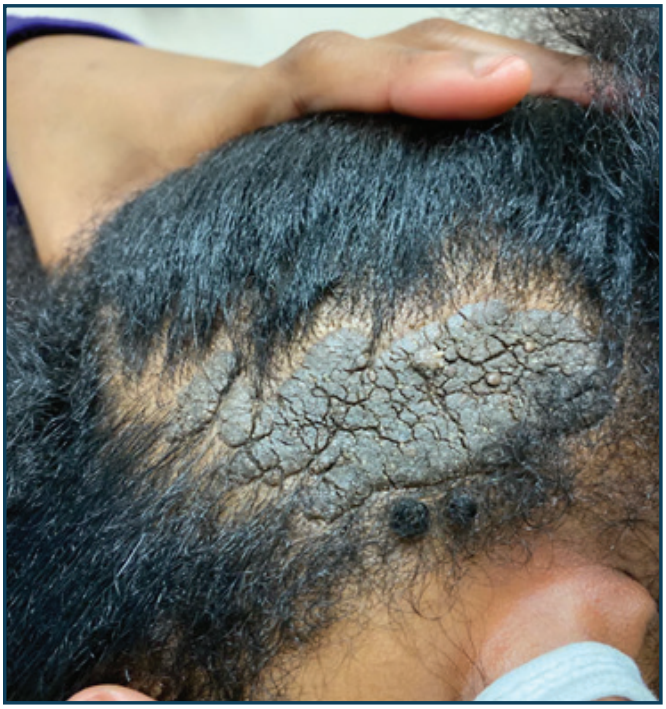
by KATIE DREHER

MD Candidate, Class of 2022, University of Arkansas for Medical Sciences, Little Rock, Ark.

A 15-year-old African American female presented with a hyperkeratotic, slightly hyperpigmented plaque of the right parietal scalp that had been present since birth. Initially flatter and smoother, it became thicker and rougher around the time of puberty, and a few discrete papules arose on the surface. It is now cosmetically concerning to the patient and is slightly painful to touch and when brushing her hair.
What is the most appropriate management?

A) Apply 0.05% clobetasol propionate ointment to the plaque until resolution
B) Obtain a punch biopsy from the edge of the plaque to confirm the diagnosis
C) Obtain a scraping for potassium hydroxide (KOH) preparation
D) Educate the patient about the low lifetime risk of cancer arising within the plaque and offer either longitudinal monitoring or excision
E) Reassure the patient about the benign nature of the lesion with no need for follow-up

Answer: D: Educate the patient about the low lifetime risk of cancer arising within the plaque and offer serial monitoring or removal.



This patient has a nevus sebaceous, a relatively uncommon benign hamartomatous birthmark of the skin. They are characterized by immature adnexal structures including hair follicles, sebaceous glands, and apocrine sweat glands, often with overlying epidermal hyperplasia. While some may occur in association with rare syndromic conditions, the majority develop in otherwise healthy infants.

Nevus sebaceous, also called nevus sebaceous of Jadassohn, are most commonly located on the scalp but may also be seen on the face, neck, or (rarely) on the trunk. They present as oval, hairless, smooth plaques at birth that are often yellow-orange in color; they may be difficult to appreciate at this stage. During childhood, they grow proportionally with the patient. At puberty, hormonal changes commonly result in lesion thickening, with development of a bumpy or warty appearance that may be cosmetically concerning to the patient.

Due to their characteristic clinical appearance, biopsy is not usually necessary for diagnostic confirmation. While the majority of sebaceous nevi remain unchanged histologically, the development of secondary neoplasms within them is possible. These proliferations are usually benign hair follicle tumors; however, rarely basal cell, sebaceous, and squamous cell carcinomas may develop.

Due to the small potential for malignant transformation, both close clinical monitoring over time and prophylactic removal are reasonable approaches to management. Development of concerning papules, nodules, or ulcerations should prompt biopsy. If the patient is concerned aesthetically, complete excision is reasonable even without consideration of the small risk of cutaneous malignancy. The patient should be counseled that a scar from this procedure is inevitable.

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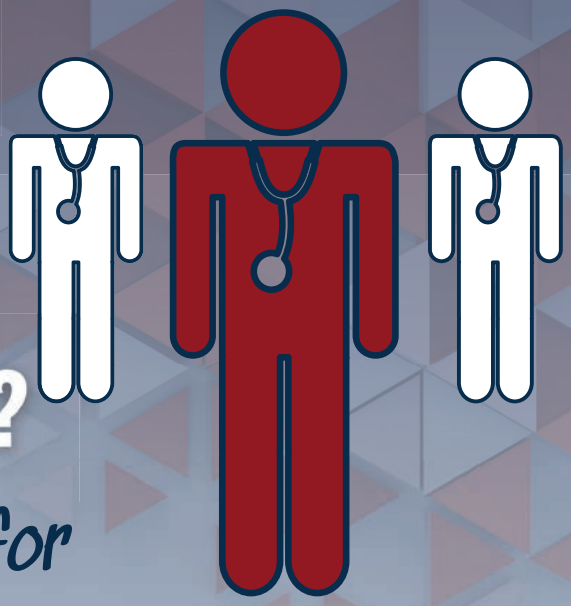

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