Case Report

Familial Xanthelasma with Dyslipidemia: Just Another Family Trait?

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Abstract

Some external features serve as a warning sign for accelerated atherosclerosis. Their early recognition may help in early detection and primary prevention/preemptive treatment of coronary artery disease (CAD). A 35-year-old nonsmoker, nonalcoholic, nonhypertensive, nondiabetic male presented with chest pain and was diagnosed to have acute ST elevation inferior wall myocardial infarction. His father had died of CAD at 40 years of age. The patient had bilateral extensive xanthelasma and gynoid obesity. His mother and younger brother also had evidence of bilateral xanthelasma; both turned out to be dyslipidemic – the younger brother qualifying for therapeutic intervention for dyslipidemia at 26 years of age. This case highlights the importance of familial xanthelasma as a cutaneous marker for underlying dyslipidemia and accelerated atherosclerosis in the young.

Keywords: Dyslipidemia, familial xanthelasma, premature coronary artery disease

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Introduction

Some cutaneous manifestations serve as a warning sign for high likelihood of early onset coronary artery disease (CAD) in an individual. These include xanthomas, xanthelasma, arcus juvenilis, and premature graying and balding in smokers; markers of insulin resistance like skin tags, acanthosis nigricans, and central obesity; hyperpigmented hands in betel quid sellers, cutaneous signs of gout, rheumatoid arthritis, psoriasis.[1,2]

We report here the case of a young male with familial xanthelasma and gynoid obesity who presented with acute ST elevation inferior wall myocardial infarction. He was treated with thrombolysis, anti-ischemic, and hypolipidemic agents. Interestingly other family members also had xanthelasma. We present the case to highlight the familial xanthelasma with dyslipidemia which predisposed our patient to acquire early onset CAD.

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Case Report

A 35-year-old male presented to the medical emergency with complaints of retrosternal discomfort, uneasiness, and sweating for 1 hour prior to presentation. The patient had a similar episode 2 days back for which he was observed for a few hours and sent back home after serial electrocardiograms came out to be normal. There was no past history of CAD, diabetes mellitus, hypertension or stroke. The patient was a nonsmoker and there was no history of use of smokeless tobacco either. There was no history of alcohol intake or drug abuse. His father had died of "heart attack" at around 40 years of age. The patient had developed light-colored patches around the eyes over the past 10 years; the same were also present in his mother and were also developing in his younger brother [Figures 1a-c].

On examination, the patient was conscious, oriented, and alert with a blood pressure of 112/76 mmHg. The pulse rate was 74/min with all peripheral pulses being palpable and no radio-radial or radio-femoral delay. The patient's BMI was 32 kg/m^2 with a waist to hip ratio of 100 cm/101 cm. Bilateral xanthelasma nearly encircling the eyes were a prominent physical finding. The systemic examination was unremarkable. ECG showed Q waves and "T" wave inversions in leads II,

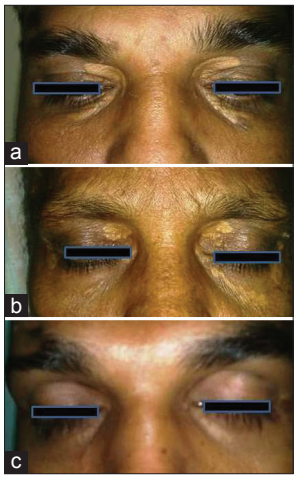

Figure 1: (a) Patient with prominent bilateral xanthelasma, (b) mother with prominent xanthelasma at medial and lateral canthi, and (c) brother with xanthelasma at medial and lateral canthi.

III, and aVF and tall "T" waves in lead V2, 3. The patient was thrombolyzed with streptokinase on the basis of ECG findings and the discomfort subsided following thrombolysis. Post-STK ECG showed Q-waves with T-inversion in the inferior leads.

The CRP was elevated (>0.8 mg/l). His fasting blood sugar was 97 mg/dl and 2 hour postprandial blood sugar was 104 mg/dl. His kidney, liver, and thyroid function tests were normal. Echocardiography revealed inferior wall severe hypokinesia with moderate apicolateral hypokinesia. Ejection fraction was 40% (normal: 55–75%) with grade II diastolic dysfunction. The patient's carotid intima media thickness was 0.056 on the right side and 0.058 cm on the left side with no plaque (normal: 0.100).

The presence of xanthelasma in family members prompted us to get the lipid profile for the mother and younger brother also. The lipid profile of the three is shown in Table 1. As it turned out, all three were dyslipidemic with even the 26-year-old, nonobese younger brother qualifying for therapeutic intervention for dyslipidemia. The Dutch Lipid network criteria score for the index case was 5 in the absence of genetic analysis and was consistent with possible heterozygous familial hypercholesterolemia. As per the Simon Broome criteria also the patient is having possible heterozygous familial hypercholesterolemia.[3]

Table 1: Lipid profile of the index case, mother, and brother

	Index case	Mother	Brother	Normal
Total cholesterol (mg/dl)	217	183	223	<200
LDL-C (mg/dl)	168	108	161	100–129
HDL-C (mg/dl)	24	43	40	40-60 (males) 50-60 (females)
VLDL	25	32	22	<40
Triglyceride (mg/dl)	124	164	111	50-150

Discussion

Xanthelasma palpebrarum (XP) refers to the planar xanthomas that occur over eyelids. These are the most common cutaneous xanthomas, seen as yellow plaques over one or both lids. They are frequently symmetrical with the inner canthi involved more commonly than the outer ones and the upper eyelids more commonly than the lower. The incidence of xanthelasma in the general population has been variously reported between 0.3% and 1.5%.[4] The age of onset ranges from 15 to 73 years with a peak in the 4th and 5th decades. Histologically XP lesions consist of foamy histiocytes laden with fat, predominantly in the upper reticular dermis. In addition to being a cosmetic problem, XP draws great attention because of its controversial relationship with hyperlipidemia, atherosclerosis, ischemic heart disease, diabetes, and hypothyroidism.

Different studies have shown a varying incidence of dyslipidemia in individuals with xanthelasma – ranging from as low as 9.1% to as high as 67.9%.[4-6] Of these, a family history of XP can be expected in around 10% of patients.[7] The Lipids Research Clinics Program Prevalence Study showed both xanthelasma and corneal arcus to be associated with increased levels of serum cholesterol and LDL-cholesterol, especially in young males. Also, the odds ratio for ischemic heart disease was found to be higher in these individuals.[8]

The possible risk for atherosclerosis in normolipidemic patients with xanthelasma is more obscure because of the relative paucity of epidemiologic data and the conflicting results in some of the lipid studies.[9,10] The cause in these patients could possibly be attributed to abnormal apolipoprotein levels or phenotype. Thus XP should be considered as a marker for accelerated atherosclerosis even independent of cholesterol levels. Interestingly our patient seemed to fit into probable heterozygous hypercholesterolemia.[3]

In conclude, one must be wary of the tendency to dismiss familial xanthelasma as "just another family trait" and look for underlying dyslipidemia and evidence of asymptomatic coronary artery disease in these individuals.

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