CLINICAL IMAGE

doi:10.1093/omcr/omu066

‘Tendon xanthomas’ as an indicator of premature coronary artery disease

Beeresha Puttegowda, Amjad Ali*, B. Ramesh and Cholenahally N. Manjunath

Sri Jayadeva Institute of Cardiovascular Science and Research, Bangalore, Karnataka, India
*Correspondence address. Tel: +91-080-22977267; Fax: +91-80-26534477; E-mail: drmz2007@gmail.com

Familial hypercholesterolemia (FH) is an autosomal dominant disorder that causes severe elevations in total cholesterol and low-density lipoprotein cholesterol [1]. A ‘definite’ diagnosis of FH is made if a patient has elevated cholesterol levels and tendinous xanthomata, or if the patient has an identified mutation in the *LDLR* gene or the apolipoprotein B-100 gene [2]. Here, we report the case of 40-year-old man who presented to the emergency department complaining of cardiac sounding chest pain associated with radiation to the left arm and sweating. Inspection revealed multiple painless swellings around elbows, ankles and toes, suggestive of tendon xanthomas and xanthelasma around eyes (Figs 1 and 2). He was diagnosed with acute coronary syndrome and proceeded to angiography, which revealed a totally occluded left anterior descending artery and right coronary artery. A lipid profile showed total cholesterol of 453 mg/dl (normal range $<$200 mg/dl), LDL 391 mg/dl (normal range $<$100 mg/dl) and triglycerides 79 mg/dl (normal range $<$150 mg/dl), highly suggestive of heterozygote FH. He underwent coronary artery bypass grafting successfully.

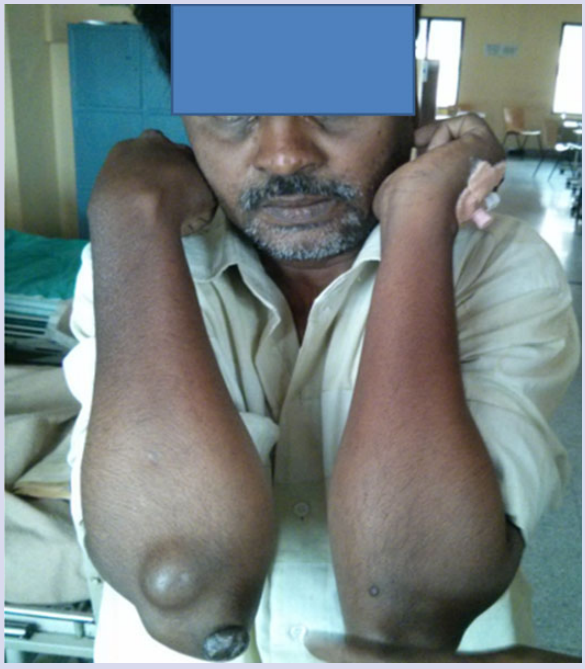

Figure 1: Xanthelasma around eyes and tendon xanthoma around right elbow.

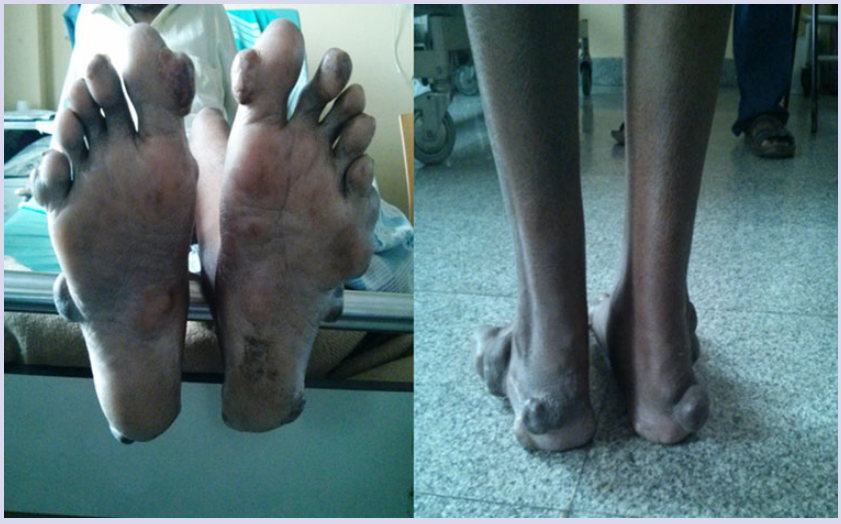

Figure 2: Tendon xanthomas around feet.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

1. Marks D, Thorogood M, Neil HA, Humphries SE. A review on the diagnosis, natural history, and treatment of familial hypercholesterolemia. *Atherosclerosis* 2003;**168**:1–14.
2. Risk of fatal coronary heart disease in familial hypercholesterolemia. Scientific Steering Committee on behalf of the Simon Broome Register Group. *BMJ* 1991;**303**:893–6.