with medical therapy and referred for coronary artery bypass grafting.

Discussion

KD is a febrile disorder of unknown cause with multiple systemic manifestations, affecting primarily infants and young children.[4] First described in Japan by Kawasaki in 1967 as acute mucocutaneous lymph node syndrome, it has been reported with increasing frequency around the world. Involvement of the heart with acute myocarditis and coronary angiitis occurs in 25 to 50% of the patients during the acute phase, accounting for most of the mortality. However, 50% of the aneurysms regress spontaneously over a one- to two-year period, and therefore adult ischaemic heart disease secondary to KD is infrequent and occurs mostly in young adults.[4]

Obtaining a history of childhood Kawasaki disease is quite difficult because the diagnosis of acute KD is based on clinical criteria only, without specific laboratory testing, and therefore requires a high index of suspicion. In young adults, diagnosis is based on typical features in two-dimensional echocardiography and coronary arteriography. The former consists of local wall motion abnormalities as a result of prior MIs and ectasia or frank aneurysms of the proximal coronary arteries.[5,6]

Echocardiography is particularly helpful in the paediatric population, both for initial diagnosis and for long-term follow up.[5,6] Coronary arteriography typically reveals multivessel aneurysmal disease alternating with segmental stenoses, coronary ectasia, calcifications, rich collateral circulation, and varying degrees of left ventricular dysfunction as a sequela of multiple MIs or myocarditis, or both.[4,6]

Conclusion

We believe that our patient had KD rather than atherosclerotic CAE, for the following reasons. (1) He had extensive triple-vessel disease at a very early age. He was a non-smoker without any other risk factors for atherosclerotic coronary artery disease (CAD). This would favour KD because patients with CAE have the typical risk profile of atherosclerotic CAD.[1-3,6] (2) Our patient had ectasia involving all three vessels, which is rare in atherosclerotic CAE, but typical of KD.[1-4] In our patient, echocardiography failed to reveal features of KD because the very proximal left main and right coronary arteries were spared. This is not uncommon in KD.[4,6] However, coronary arteriography clearly demonstrated the typical findings of KD.

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Letter to the Editor

Dear Sir

The review article by Owira and Ojewole[1] cites, among others, the influence of grapefruit (juice) on amiodarone pharmacokinetics and pharmacodynamics. Some apparent inconsistencies are worthy of note.

Amiodarone is not a 'prodrug' only. It has inherent pharmacodynamic effects. Its major N-dealkylation metabolite, N-desethylamiodarone (N-DEA) appears to possess even greater pharmacodynamic effects, notably with regard to cardiac electrophysiology.

The statement that concomitant ingestion of grapefruit juice 'led to clinical prolongation of QT intervals and torsades de pointes' is misleading and not substantiated by the articles cited.[2,3] On the contrary, the grapefruit-initiated inhibition of N-DEA formation resulted in decreased cardiac electrophysiological effects. It is not clear whether 'accumulation' of amiodarone resulting from inhibition of conversion to N-DEA has clinical implications, but reduction in N-DEA concentrations may compromise the anti-arrhythmic action of amiodarone.

A further observation concerns the incorrect title of the article by Libersa *et al*.,[3] which creates the impression that amiodarone metabolism is dramatically induced by grapefruit juice. The converse is true.

The authors of reference number 2 above (number 14 in the article) are incorrectly written. It should be Tsutomu U, instead of Urano T, and Ryuichi H, instead of Hasegawa R.

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