Acute Myocardial Infarction in a Patient with Isolated Dextrocardia

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Isolated dextrocardia without situs inversus is found in less than 1% of the general population. In patients with this condition, the heart is presented as a mirror image of itself with its apex pointing to the right. Pulmonary, thoracic, and abdominal organs are not reversed. Dextrocardia occurs at birth but its diagnosis may be postponed to adulthood. We report a case of acute myocardial infarction (MI) in a patient with dextrocardia.

PATIENT AND METHODS

A 69 year old woman presented to the emergency department complaining of severe prolonged retrosternal chest pain. Electrocardiogram (ECG) showed minimal ST-elevation with peaked T waves in chest leads V1-V2 and inverted T waves in the inferolateral leads (II, III, AVL, V5-V6). There was also marked right axis deviation implicated by inverted QRS complexes in lead AVL in addition to low voltage in precordial leads V4 through V6 [Figure 1A]. Posterior ECG showed ST-elevation in V4R-V6R and inverted T waves in V7-V9 [Figure 1B]. High cardiac troponin levels provided further confirmation of acute coronary syndrome, and a chest X-ray suggested the diagnosis of dextrocardia.

Cardiac catheterization was performed and demonstrated total occlusion of the proximal right coronary artery (RCA) [Figure 1C]. Revascularization was successful using a drug eluting stent (DES) [Figure 1D]. Two days later the patient was readmitted to the hospital with pulmonary edema. Echocardiography showed global akinesia of the left ventricle with severely reduced ejection fraction. Cardiac computed tomography to establish the proper anatomy was preformed [Figure 1E]. The examination showed normal aortic and abdominal organs anatomy; thus, confirming the diagnosis of isolated dextrocardia.

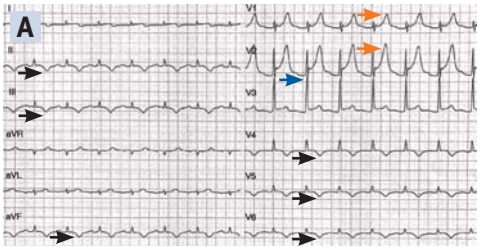


Figure 1A. Electrocardiogram demonstrating right axis deviation together with ST segment elevation (blue arrow) with peaked T waves in chest leads V1-V2 (orange arrow) and inverted T waves in the inferolateral leads (black arrow)

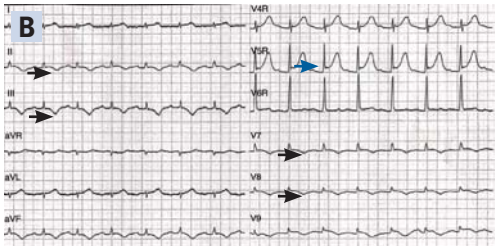


Figure 1B. Posterior electrocardiogram demonstrating ST elevation in V4R-V5R (blue arrow) and inverted T waves in leads II, III , AVF, V7-V9 (black arrow)

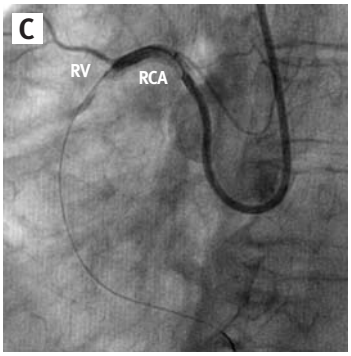


Figure 1C. Cardiac catheterization in an anterior projection demonstrating total occlusion of the proximal RCA which is pointing towards the right

RCA = proximal right coronary artery, RV = right ventricular branch

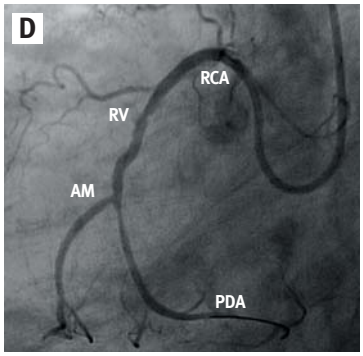
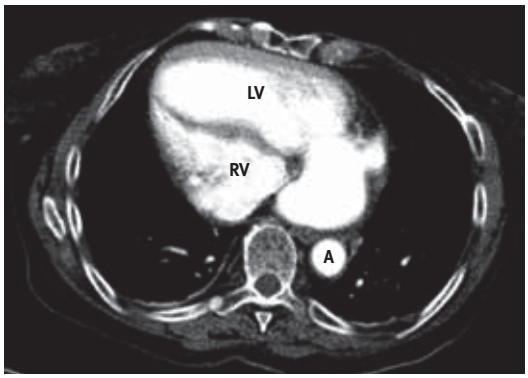


Figure 1D. Cardiac catheterization in an anterior projection demonstrating TIMI 3 flow after revascularization with DES

TIMI = thrombolysis in myocardial infarction, DES = drug eluting stent, RCA = proximal right coronary artery, RV = right ventricular branch, AM = acute marginal, PDA = posterior descending artery

Figure 1E. Cardiac computed tomography showing the apex of the heart pointing to the right with normal aortic anatomy



LV = left ventricle, RV = right ventricle, A = descending aorta

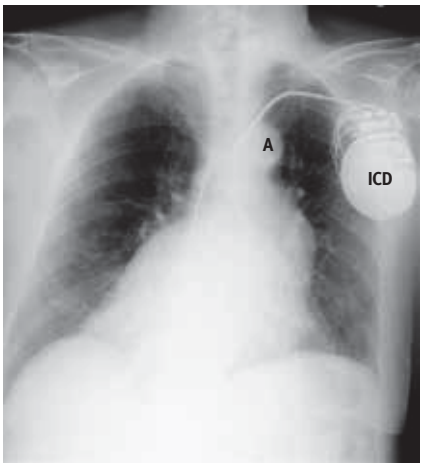


Figure 1F. Chest X-ray demonstrating the heart in dextrocardia position along with the aorta in normal position and the implanted ICD

ICD = implanted cardiac device, A = aorta

An implanted cardiac device was introduced uneventfully [Figure 1F].

DISCUSSION

The clinical diagnosis and electrocardiographic localization of MIs in patients with dextrocardia remain a great challenge. There are some reports in the literature that emphasize that the extent of MI in such patients may be underdiagnosed [1,2].

Permanent pacemaker implantation in patients with dextrocardia may be challenging because of the peculiar anatomy. Use of a technique using angiography to delineate chamber anatomy and relationship can assist the operator during such difficult procedures. The survival after a successful pacemaker implantation in patients with dextrocardia is favorable [3].

Acknowledgments

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Capsule

Defining total-body AIDS-virus burden with implications for curative strategies

In the quest for a functional cure or the eradication of HIV infection, it is necessary to know the sizes of the reservoirs from which infection rebounds after treatment interruption. **Estes** and co-authors quantified SIV and HIV tissue burdens in tissues of infected nonhuman primates and lymphoid tissue (LT) biopsies from infected humans. Before antiretroviral therapy (ART), LTs contained › 98% of the SIV RNA+ and DNA+ cells. With ART, the numbers of virus (v) RNA+ cells substantially decreased but remained detectable, and their persistence was associated with relatively lower drug concentrations in LT than in peripheral blood. Prolonged ART also decreased the levels of SIV- and HIV-DNA+ cells, but the estimated size of the residual tissue burden of 10^8vDNA+ cells potentially containing replication-competent proviruses, along with evidence of continuing virus production in LT despite ART, indicated two important sources for rebound following treatment interruption. The large sizes of these tissue reservoirs underscore challenges in developing 'HIV cure' strategies targeting multiple sources of virus production.

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