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EDITORIAL

Special issue on ischaemic heart disease

Cardiovascular disease (CVD), primarily ischaemic heart disease (IHD), usually referred to coronary artery disease (CAD), is the leading cause of death in Western countries [1]. We have already had the opportunity, in previous issues, to address ischaemic heart disease [2,3] and in this issue of Acta Cardiologica we wanted to discuss the most recent publications on this subject.

Atherosclerotic CAD is a multi-factorial entity influenced by both genetic and environmental factors. MicroRNAs (miRNAs) have been found to have an essential role in cardiovascular diseases [4]. In their study, Coban et al. analysed two miRNAs in angiographically confirmed CAD ($n = 50$) and non-CAD ($n = 43$) with insignificant coronary stenosis. They found that miR-26a-5p expression was altered in CAD while miR-19a-3p expression was different in diabetes. Both miRNAs were closely related to risk factors of CAD and, therefore, could be considered as therapeutic targets for CAD treatment [5].

Myocardial bridging (MB) is a common congenital cardiovascular anomaly in which a segment of an epicardial coronary artery takes a tunnelled course under a bridge of the myocardium [6]. In their study, Narayanan et al. showed that in patients who presented with advanced clinical atherosclerosis, there was no difference in IVUS-derived qualitative and quantitative plaque characteristics in patients who had MB vs. those who did not have the abnormality [7]. Coronary computed tomography angiography (CCTA) is a non-invasive reference tool to accurately assess coronary artery anomalies. Intraoperative transesophageal echocardiogram could be of help to identify the presence of coronary anomalies (early bifurcation or separate origin of the anterior descending and circumflex artery) associated with bicuspid aortic valve (BAV) (Figures 1 and 2) [8].

Recent guidelines recommend the use of a short sensitive cardiac troponin (hs-cTn) algorithm in patients presenting with chest pain at the emergency department (ED) [9]. In their study, Van Assche et al. evaluated in 100 patients the safety and effectiveness of the new 0-1h hs-cTn I protocol in comparison with the standard 0-3h cTn I protocol for the diagnosis of acute myocardial infarction (AMI). They showed that the abbreviated protocol was effective and safe for the exclusion of AMI at the ED (Figure 3) [10].

Vandebosch et al. described a case of severe coronary artery spasm caused by a 5HT1-agonist as a result of newly diagnosed Graves' disease and myocarditis, likely triggered by mRNA-1273 SARS-CoV-2 booster vaccine [11,12]. Contrast-induced nephropathy (CIN) is a disorder that adversely affects the prognosis of ST-elevation myocardial infarction (STEMI). Özveren et al. reported in a large cohort of STEMI patients ($n = 3057$) that the logarithm of haemoglobin and albumin product (LHAP) was the most important predictor of CIN [13]. Systemic inflammation-response index (SIRI) is among the novel biomarkers, the calculation of which is based on parameters of complete blood count. It combines two formulas, namely neutrophil-to-lymphocyte ratio (NLR) and monocyte-to-lymphocyte ratio (MLR) into a single one. In a series of 676 patients, Yildiz et al. reported that SIRI had greater diagnostic power than NLR and MLR for the identification of high-risk patients for the occurrence of CIN

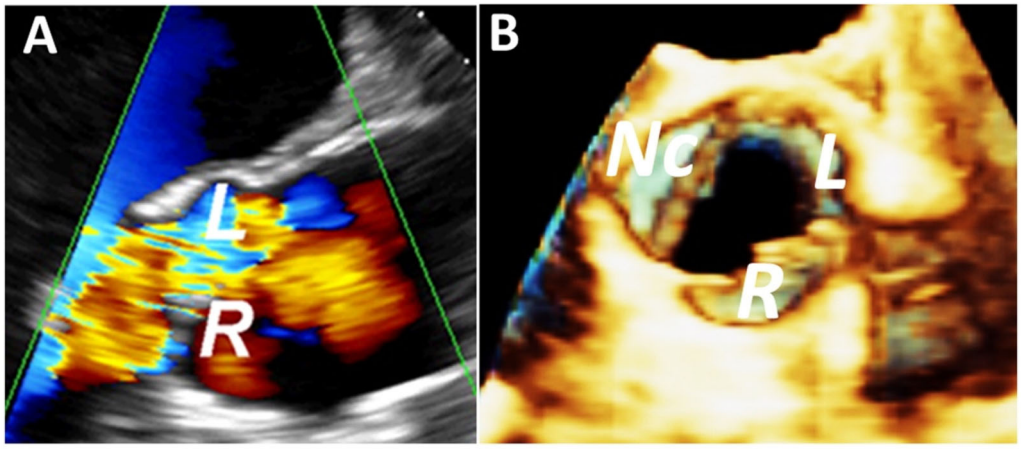

Figure 1. (A) A 120° Long-axis view (2D TEE) shows a broad jet of aortic regurgitation. (B) 3D volume of the aortic root (from reference [8]).

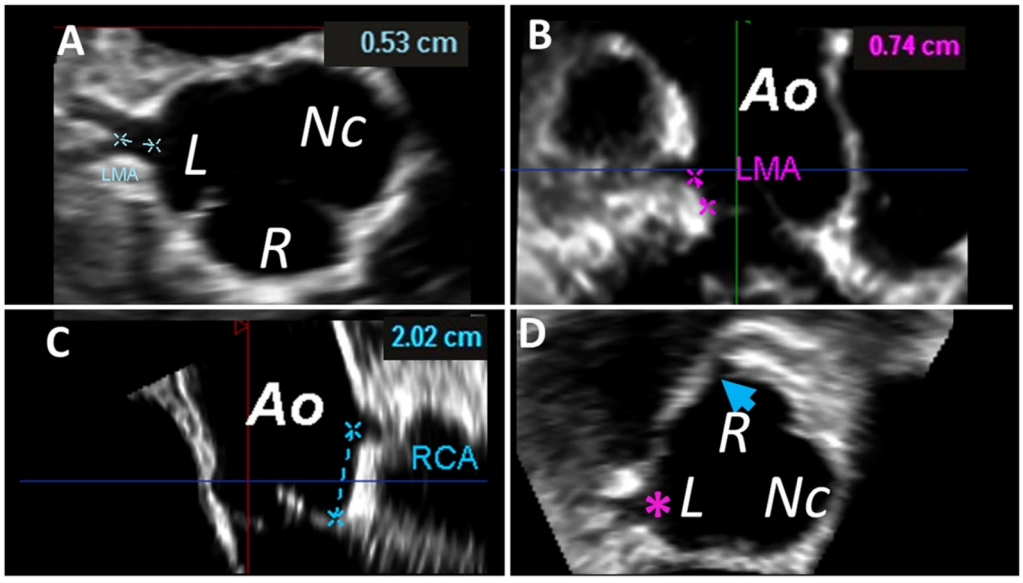

Figure 2. (A) The axial view shows the origin of the left coronary artery; the dotted line indicates the length of the left coronary artery to its bifurcation. (B) Coronal view shows the origin and the height take-off of the left coronary artery (dotted line). (C) Sagittal view shows the origin and the height take-off of the right coronary artery (dotted line). (D) Origin and deployment of the right (arrow) and left coronary arteries (asterisk). Abbreviations: Ao: aorta; L: left sinus of Valsalva; LMA: left main artery; Nc: non-coronary sinus of Valsalva; R: right sinus of Valsalva; RCA: right coronary artery (from reference [8]).

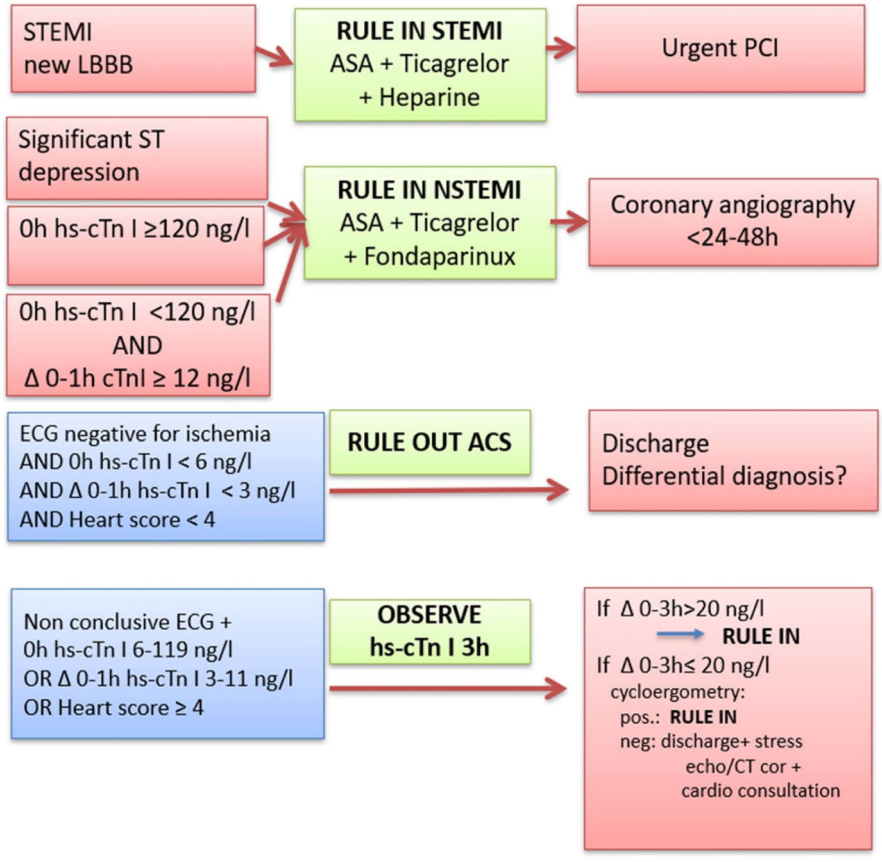

Figure 3. Chest pain flowchart. STEMI indicates ST-elevation myocardial infarction; LBBB left bundle branch block; ASA acetylsalicylic acid; PCI percutaneous coronary intervention; h hour; hs-cTnI high sensitive cardiac troponin I; NSTEMI non-ST elevation myocardial infarction; ACS acute coronary syndrome and CT computed tomography (from reference 10].

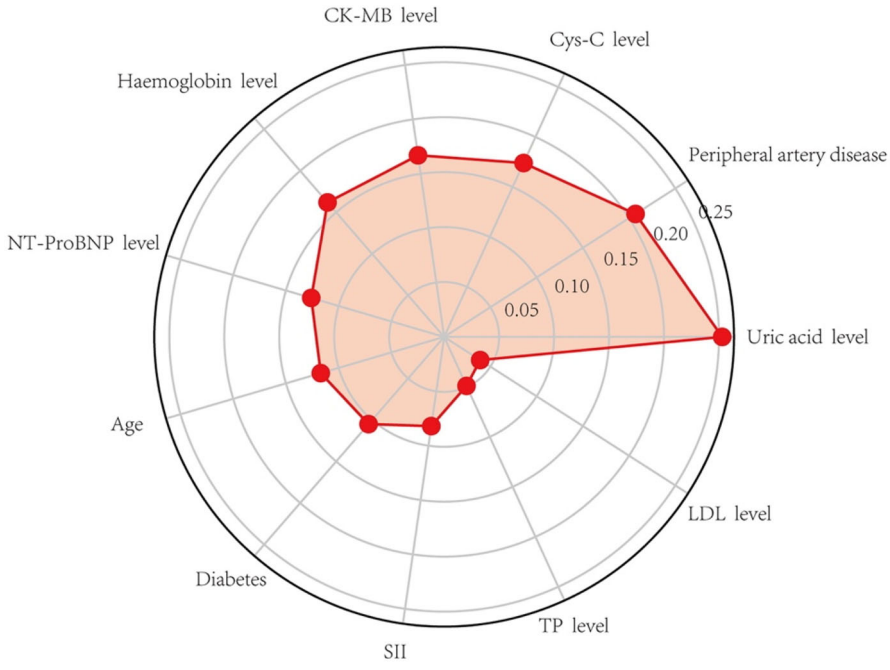

Figure 4. SHAP radar plot of 11 features in the Support vector machine model. This radar plot shows the importance of each variable in the final prediction model (from reference [15]).

[14]. Ma et al. developed and validated machine learning algorithms to predict CIN using 11 preoperative clinical features (uric acid, peripheral vascular disease, cystatin C, creatine kinase-MB, haemoglobin, N-terminal pro-brain natriuretic peptide, age, diabetes, systemic immune-inflammatory index, total protein, and low-density lipoprotein) (Figure 4) [15]. Intriguingly, Del Rio-Pertuz et al. showed that CHA_2DS_2-VASc risk score was associated with an increased risk for CIN in patients undergoing percutaneous coronary intervention (PCI) [16]. However, larger studies are needed to clarify this association and to identify strategies to reduce CIN.

In their study, Choe et al. examined the effect of nicorandil treatment for 6 months on infarct size, cardiac function as determined by cardiac magnetic resonance imaging (CMR), and clinical outcomes in STEMI patients undergoing primary PCI. The authors found that nicorandil treatment did not improve these parameters in STEMI patients [17]. These results were well discussed in an editorial accompanying this study [18]. No flow can complicate several PCI procedures, more commonly noted in STEMI and saphenous vein graft interventions. D-dimer, a simple and widely available surrogate marker of thrombosis, has been studied with conflicting results for its prognostic value in STEMI. D-dimer has also been observed by various researchers as a predictive marker for no-reflow following PCI. In their meta-analysis, Jain et al. compiled the results of 8 studies involving a total of 4,321 STEMI patients undergoing PCI, which highlighted the potential predictive role of D-dimer levels in the no-reflow phenomenon following PCI in the setting of AMI [19]. In this issue of Acta Cardiologica, several focus images highlighting interesting cases have also been reported [20–22].

Disclosure statement

No potential conflict of interest was reported by the author(s).

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