Case Report

Coronary artery calcification on chest computed tomography scan – Anaesthetic implications

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ABSTRACT

The significance of coronary artery calcification noted on noncontrast chest computed tomography (CT) scan is unknown. We report a case where coronary artery calcification on chest CT scan incidentally detected in an asymptomatic patient who was found to have significant coronary artery disease which resulted in perioperative morbidity.

Key words: Anaesthesia, chest CT scan, coronary artery calcification

INTRODUCTION

Diagnosis of coronary artery disease (CAD) during preoperative checkup is important to stratify the risk and to optimise patients so as to minimise perioperative cardiac complications. Detection of coronary artery calcification (CAC) by electrocardiogram (ECG)-gated computed tomography (CT) scan and its scoring is a widely available, consistent and reproducible method of assessment of risk for major cardiovascular outcomes, especially in asymptomatic patients.[1] However, the significance of incidentally detected coronary artery calcification on noncontrast chest CT scan in patients undergoing noncardiac surgery remains unknown.

CASE REPORT

A 58-year-old male patient, with a mass in the left lung upper lobe [Figure 1], was posted for thoracoscopic mediastinal lymph node sampling and left upper lobectomy. The patient was on tablets telmisartan and amlodipine for hypertension for the past 10 years. Preanaesthetic evaluation and routine investigations, including two-dimensional echocardiography, were unremarkable. The patient had a good effort tolerance. Under general anaesthesia, mediastinoscopy was performed uneventfully and decision to proceed with lobectomy was taken. A single-lumen endotracheal tube was replaced with a 37-F left-sided double-lumen tube (DLT) and its position was confirmed by bronchoscopy. Depth of anaesthesia was maintained with sevoflurane in oxygen and nitrous oxide mixture (70:30) along with intermittent boluses of intravenous fentanyl. After insertion of the thoracoscope, there was tachycardia (heart rate 100/min) with hypertension (160/100 mmHg). Infusion of intravenous dexmedetomidine (1 μg/kg over 20 min) was started, with the intention of controlling the sympathetic response. Ten minutes later, there was hypotension [blood pressure (BP) 94/40 mmHg] with mean arterial pressure (MAP) <60 mmHg. The infusion of dexmedetomidine was immediately stopped. Surgical retraction of mediastinal structures was interrupted. Bolus of normal saline (200 mL within 5 min) and two doses of mephentermine (total 6 mg) were administered. BP recovered momentarily

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but dropped again. Noradrenaline infusion was started through a wide bore cannula on the left upper limb and continued intraoperatively (0.09–0.15 mcg/kg/min) to maintain MAP between 70 and 75 mmHg. Further intraoperative course was uneventful. After excision of the left upper lobe, ST segment depression of 3.5 mm was noted on the monitor, in leads II and III. However, with an increase in the dose of noradrenaline, MAP increased to more than 90 mmHg and ST depression resolved within 5 minutes. Intercostal block, administered by the surgeon using 0.25% bupivacaine 3 ml each at three intercostal spaces, was supplemented with intravenous injection of 1 g paracetamol. Transthoracic echocardiography, done after turning the patient supine, did not show any regional wall motion abnormality. At the end of anaesthesia, BP increased further (≈140/86 mmHg) and noradrenaline infusion was tapered off. Following uneventful recovery, the DLT was removed and the patient was shifted to the recovery room (RR) on spontaneous breathing with heart rate of 120/min and BP of 130/80 mmHg without any vasopressors. He did not complain of pain at the surgical site. In RR, a 12-lead ECG was repeated, which showed no ST-T changes. Half an hour later, he again developed hypotension (90/60 mmHg) with ST depression in lead II. Noradrenaline infusion was restarted; but within seconds, the patient developed pulseless ventricular tachycardia. With five cycles of chest compressions, bag mask ventilation and defibrillation (200 J two shocks), spontaneous circulation was recovered within 3 min. ECG showed sinus rhythm with right bundle branch block. The trachea was intubated, central venous access was secured through left internal jugular vein, tablet aspirin 325 mg was administered through nasogastric tube and the rate of noradrenaline infusion was increased to maintain MAP >90 mm Hg. Transthoracic echocardiography showed global hypokinesia of the left ventricle with ejection fraction ≈10%–15%. In view of acute coronary syndrome with haemodynamic instability, the patient was immediately moved to an adjacent cardiac catheterisation laboratory. Coronary angiography revealed total occlusion of mid portion of right coronary artery, 80% stenosis of proximal portions of left anterior descending artery (LAD) and circumflex artery (LCX). Collaterals showed adequate flow around stenotic segments. Haemodynamic stability was achieved with intra-aortic balloon pump and titration of inotropes. After 5 days, the patient was discharged with ejection fraction of 35% on medications for heart failure. After 25 days from the primary event, he underwent coronary artery bypass grafting uneventfully. Retrospectively, evaluation of chest CT scan showed CAC in LAD and LCX vessels [Figure 2].

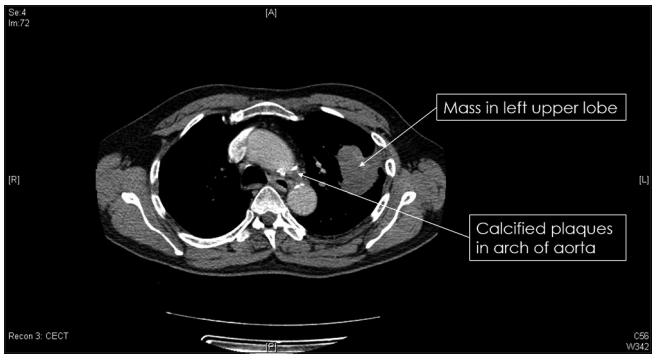

Figure 1: Chest CT scan showing left upper lobe mass

DISCUSSION

In this case, during preoperative evaluation, we failed to detect significant CAD despite following American College of Cardiology/American Heart Association (ACC/AHA) 2014 guidelines for perioperative cardiovascular evaluation for noncardiac surgery.[2] Lack of history of cardiac events, normal echocardiography findings and good effort tolerance masked the underlying CAD. In hindsight, CAC noted in chest CT scan turned out to be the only finding suggestive of CAD. Hence, the questions that arise are – does CAC detected in chest CT scan represent CAD? Should perioperative management of asymptomatic patient vary if CAC is noted on chest CT scan?

Various societies of cardiology across the world have accepted CAC scoring as a useful method in quantification of risk of CAD.[3-5] In ACC/

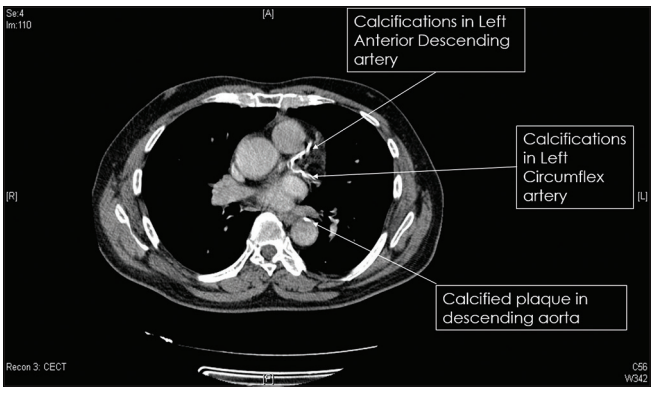

Figure 2: Chest CT scan showing coronary artery calcifications in left anterior descending and left circumflex artery. Also seen are the calcified plaques in the descending aorta

AHA 2010 guidelines, quantification of CAC on ECG-gated CT scan was considered as reasonable in asymptomatic individuals at intermediate risk and in all diabetic patients more than 40 years of age.[5] In a community-based case-controlled study, Hughes-Austin *et al*. found that Spearman's correlation of CAC scores between ECG-gated CT scan and noncontrast chest CT scan was 0.93. Similar to those with ECG-gated CT scan, each standard deviation higher calcium score on chest CT scan was associated with 50% higher odds of mortality (odds ratio 1.5; 1.2–1.9).[6] In another cross-sectional study, a significant correlation was found between CAC scores on noncontrast chest CT scans of young individuals and Framingham risk scores for cardiovascular disease.[7] These findings suggest that presence of CAC on noncontrast chest CT scan carries clinical and prognostic significance.

CAC occurs as healing process of inflamed atheromatous plaque in coronary arteries. As per a study by Mizukoshi *et al.*, spotty calcium deposits are more commonly associated with acute myocardial infarction (AMI) and unstable angina than large calcium deposits in the coronaries. Also, plaque rupture frequency correlated positively with spotty calcium deposits and inversely with large calcium deposits in coronaries.[8] Shemesh *et al.* showed that extensive calcium characterises the coronary arteries of patients with chronic stable angina, and AMI most often occurs in mildly calcified or noncalcified arteries.[9] Thus, CAC, though a marker of atherosclerosis, may be associated with stable plaques and lower risk of AMI. However, with extensive calcification of tunica media, coronary arteries loose elastic fibres and become stiff. This interferes with the ability of autoregulation.[10] Flow across the coronaries becomes dependent on diastolic pressure, as coronary perfusion occurs during diastole. Patient with calcified coronaries is likely to suffer myocardial ischemia following a drop in diastolic BP, even of mild severity. This hypothesis is further supported by the findings of Rahman *et al*.[11] In 6811 participants from the Multi-Ethnic Study of Atherosclerosis, Cox model was used to examine whether diastolic BP category is associated with coronary heart disease events, stroke and mortality. Analyses were conducted in the sample overall and after stratification by coronary artery calcium score. In multivariable-adjusted analyses, compared with diastolic BP of 80–89 mmHg, persons with diastolic BP <60 mmHg had increased risk of coronary heart disease events [hazard ratio 1.69 (95% confidence interval 1.02–2.79)] and all-cause mortality [hazard ratio 1.48 (95% confidence interval 1.10–2.00)]. After stratification, associations of diastolic BP <60 mmHg with events were present only in participants with coronary artery calcium >0. Diastolic BP <60 mmHg was not associated with events when coronary artery calcium was zero.[11] In our patient, diastolic BP of the patient remained below 65 mmHg intraoperatively, whereas his diastolic BP before induction of anaesthesia was above 80 mmHg. This could have affected the coronary perfusion across severely calcified coronaries.

Despite recommendations from various societies of radiology, it is common to find that radiologists do not report calcification of coronaries in chest CT scans.[12] Htwe *et al*. categorised the extent of calcification as absent, mild (deposits up to one-third of length of artery), moderate (deposits in one-third to two-third of length of artery) and severe (deposits at more than two-third of length of artery) based on visual screening of CT scans of 631 asymptomatic individuals. These visual scores were compared with Agatston scores calculated after reconstructions of the CT scans. The visual score categories showed excellent agreement (weighted kappa of 0.83; 95% confidence interval: 0.79–0.88) with Agatston score categories.[13] As per this method, CAC in our patient falls in severe category. Thus, visual eyeballing of chest CT scan can identify presence and extent of CAC. This is particularly important for anaesthesiologists as, with minimal additional training and practice, one can get clue about the coronary atherosclerosis, especially in an asymptomatic patient. Based on the risk of the surgery, anaesthesiologists can decide about additional investigation like exercise testing, which would stratify the cardiovascular risk.

CONCLUSION

Learning points in this case are that CAC noted in nontargeted CT scans may suggest CAD. Whether presence of CAC on noncontrast CT scan warrants any change in perioperative management is unclear due to lack of evidence.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be

reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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