Original Article

Factors Associated with Delayed Cardiac Tamponade after Cardiac Surgery

Edgar Hernández Leiva, Marisol Carreño[1], Fernando Rada Bucheli, Alberto Cadena Bonfanti, Juan Pablo Umaña[1], Rodolfo José Dennis[2]

Departments of Cardiology, [2]Internal Medicine and [1]Cardiovascular Surgery, Instituto de Cardiología-Fundación Cardioinfantil, Universidad del Rosario, Bogotà DC, Colombia

Abstract

Context: Cardiac tamponade (CT) following cardiac surgery is a potentially fatal complication and the cause of surgical reintervention in 0.1%–6% of cases. There are two types of CT: acute, occurring within the first 48 h postoperatively, and subacute or delayed, which occurs more than 48 h postoperatively. The latter does not show specific clinical signs, which makes it more difficult to diagnose. The factors associated with acute CT (aCT) are related to coagulopathy or surgical bleeding, while the variables associated with subacute tamponade have not been well defined. **Aims:** The primary objective of this study was to identify the factors associated with the development of subacute CT (sCT). **Settings and Design:** This report describes a case (n = 80) and control (n = 160) study nested in a historic cohort made up of adult patients who underwent any type of urgent or elective cardiac surgery in a tertiary cardiovascular hospital. **Methods:** The occurrence of sCT was defined as the presence of a compatible clinical picture, pericardial effusion and confirmation of cardiac tamponade during the required emergency intervention at any point between 48 hours and 30 days after surgery. All factors potentially related to the development of sCT were taken into account. **Statistical Analysis Used:** For the adjusted analysis, a logistical regression was constructed with 55 variables, including pre-, intra-, and post-operative data. **Results:** The mortality of patients with sCT was 11% versus 0% in the controls. Five variables were identified as independently and significantly associated with the outcome: pre- or post-operative anticoagulation, reintervention in the first 48 h, surgery other than coronary artery bypass graft, and red blood cell transfusion. **Conclusions:** Our study identified five variables associated with sCT and established that this complication has a high mortality rate. These findings may allow the implementation of standardized follow-up measures for patients identified as higher risk, leading to either early detection or prevention.

Keywords: *Cardiac surgery, delayed cardiac tamponade, postoperative care*

Introduction

Hemodynamically relevant effusion following cardiac surgery is a potentially life-threatening condition, requiring rapid diagnosis and treatment. Cardiac tamponade (CT) is the cause of surgical reintervention in 0.1–6% of cases.[1] CT can be divided into two types, based on the time of presentation: early or acute (aCT), occurring in the first 48–72 h, and subacute or late (sCT), which can develop without clear clinical signs 2–3 or more days after cardiac surgery. Although most published works recognize these two types of postoperative CT, the temporal cutoff point varies among authors.[1-4] This classification, although arbitrary, seeks to highlight a characteristic with a significant prognostic implication, which is that sCT, thanks to its insidious presentation, can be more difficult to diagnose.[5,6] Factors associated with aCT are related mainly to coagulopathy or surgical bleeding, while sCT seems to be of multifactorial origin.[7-9] Beyond the first 48 h, variables associated with this complication are not well defined. Better information is required to identify the factors that will, in turn, allow early recognition of patient populations at risk of developing sCT and the implementation of strategies for its early detection. A significant proportion of patients undergoing coronary revascularization surgery have had a recent coronary event[10] and routinely receive antiplatelet and/or antithrombin therapy until the day of surgery. The association of these medications with the appearance of sCT has not been conclusive.

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Address for correspondence:
Dr. Edgar Hernández Leiva, Calle 163 A # 13B-60, Bogotá DC, Colombia.
E-mail: edgarhernandez@cardioinfantil.org

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appearance of sCT. The primary working hypothesis was that postoperative exposure to anticoagulant medications might increase the probability of this complication, but that other perioperative factors would also be associated with this outcome.

Methods

Patients

The study included all adult patients who underwent cardiac surgery and had a diagnosis of sCT in a high-complexity cardiac hospital between January 2008 and November 2015. In accordance with the Institutional Research Ethics Committee norms, informed consent was not required due to the retrospective design of the study.

The general inclusion criteria were defined as adult patients who underwent any type of urgent or elective cardiac surgery. The occurrence of sCT was defined as the presence of the following three criteria at any point between 48 h and 30 days after surgery:

1. A compatible clinical picture: arterial hypotension not explained by hypovolemia, arrhythmia, ventricular dysfunction, vasoplegia, or other causes
2. Pericardial effusion with echocardiographic signs of increased intrapericardial pressure
3. Confirmation of CT during the required emergency intervention. Percutaneous (pericardiocentesis) or surgical treatment (pericardial window or sternotomy and re-exploration) showing intrapericardial accumulation and/or clots, the removal of which immediately improved the patient's hemodynamics.

The reason for defining this period of time is that early CT (earlier than 48 h) is more likely related to problems concerning the surgical technique or acquired coagulopathy, while the appearance of sCT seems to be the result of a confluence of multiple factors and is appropriate for analysis in the search for related variables.

Radiologic tests were not required for sCT diagnosis since the chest X-ray may be normal.[5] Heart size is known to be normal until there is moderate effusion with a volume of at least >200 mL,[11] and echocardiography is widely held to be the gold standard in tamponade diagnosis.[12]

Once CT was diagnosed, immediate treatment was carried out, in most cases through a subxiphoid pericardial window. In stable patients without signs of thrombi on echocardiogram and with a predominantly anterior pericardial effusion, guided pericardiocentesis was the procedure of choice, leaving a pigtail catheter in the pericardial cavity for approximately 48 h. Occasionally, when ongoing active bleeding or a constrictive effect due to clots was suspected, resternotomy was performed to completely remove the clots and pericardial effusion.

Design

This case–control study is nested in the previously defined historical cohort, with a 1:2 case–control relationship. A case was defined as a patient diagnosed with sCT in the time period described. All patients received interventions at the time of diagnosis through pericardiocentesis or surgery. For each case, two controls were analyzed, chosen as the next two consecutive patients undergoing any type of cardiac surgery after the patient was identified as a case, and in whom neither aCT nor sCT was documented postoperatively. This methodology for selecting controls fulfilled the basic criteria of the study design, which is that the controls must be selected from the same population base from which the cases are taken so that if the control should develop the event being studied (sCT), he/she would have to appear on the list of cases.

We did not try to limit the type of surgery to a single subgroup (Coronary artery bypass graft [CABG], valvular, etc.) since the type of surgery itself may be an important risk factor. For this same reason, neither did we restrict the study to elective surgery or another subpopulation of patients.

All factors potentially related to the development of sCT were taken into account. A complete list of the analyzed variables is presented in Table 1, classified in three groups: preoperative, intraoperative, and postoperative. Table 2 describes the type of surgeries. Of the 127 patients undergoing CABG, 52 (41%) had off-pump procedures. We do not perform atrial volume reduction procedures at our institution.

Additional File includes the re-exploration rate for all patients from 2008 to 2015, the postoperative drainage threshold for re-exploration, and the trigger for red cell transfusion, according to our institutional guidelines.

Statistical analysis

The descriptive characteristics of cases and controls were contrasted using the *t*-test or the Chi-squared test. The characteristics of the distribution of each variable were previously verified using the Shapiro–Wilk test. The Wilcoxon-Mann-Whitney test was used for variables with a nonnormal distribution. For the adjusted analysis of the data, a logistic regression model was constructed using stepwise selection with the complication of sCT as the dependent variable, postoperative anticoagulant therapy as the main independent variable, and the remaining variables related to its development as covariables. The criterion for the addition or removal of a variable in the logistic model was a bivariate probability with a $P \leq 0.20$ (variables possibly associated). The quality of fit of the model was evaluated with the Hosmer–Lemeshow test. All analyses were performed using STATA 13. For all tests, values of $P < 0.05$ were considered significant.

Table 1: Evaluated variables

Preoperative	Intraoperative	Postoperative
Age (years)	CABG	Full postoperative anticoagulation
Body weight (kg)	Aortic valve replacement	Time to initiation of full anticoagulation (h)
Height (cm)	Mitral valve repair or replacement	Type of anticoagulant
Sex	CABG + aortic valve replacement	Time to initiation of antithrombotic prophylaxis (h)
Use and type of anticoagulant medications and preoperative time of suspension*	CABG + mitral valve replacement	Time to initiation of ASA (h)
Use and type of antiplatelet medications and preoperative time of suspension*	Cardiac transplant	Maximum INR value
Diabetes mellitus	Other type of cardiac surgery	Total red blood cell units transfused (intra-and postoperatively)
Chronic obstructive pulmonary disease	Emergency surgery	Reintervention in the first 48 h postoperatively (due to bleeding or early tamponade)
Arterial hypertension	Surgery for endocarditis	Surgical site infection
Acute coronary syndrome in the 30 days prior to surgery	Number of bypasses in isolated CABG or in combined surgery	Number of days with mediastinal/chest tubes
	Perfusion time (min)	Drainage volume from tubes in the first 12 h postoperative (mL)
	Clamp time (min)	Total drainage volume from tubes in the first 24 h postoperative (mL)
	Use of tranexamic acid	Total drainage volume from tubes (mL)
	Number of red blood cell units transfused intraoperatively (units)	Maximum postoperative creatinine value (mg/dL)
	Number of plasma units transfused intraoperatively (units)	

*A patient was considered exposed to ASA if they received the medication up to 1 day prior to surgery; to unfractionated heparin if they received a dose in the 4 h prior to surgery; and to enoxaparin if there was a dose in the 10 h prior to surgery. CABG: Coronary artery bypass graft, INR: International normalized ratio, ASA: Acetylsalicylic acid

Table 2: Type of surgery

	Total (*n*=240)	Cases (*n*=80)	Controls (*n*=160)
CABG, *n* (%)	127	23 (28.7)	104 (64.2)
Aortic valve, *n* (%)	54	25 (31.2)	29 (17.9)
CABG + aorta, *n* (%)	5	0	5.162 (3.0)
CABG + mitral, *n* (%)	8	4 (5)	4 (2.4)
Mitral valve, *n* (%)	29	19 (23.7)	10 (6.1)
Other surgeries, *n* (%)	14	7 (8.7)	7 (4.3)
Transplant, *n* (%)	3	2 (2.5)	1 (0.6)
Surgery other than CABG, *n* (%)	113	57 (71.2)	56 (34.5)

CABG: Coronary artery bypass graft

Results

A total of 240 patients were included in the study: 80 cases and 160 controls. The analyzed variables can be seen in Table 1.

In the crude data analysis, various factors were significantly related to the development of sCT. In general, the cases were younger than the controls. The use of preoperative or postoperative anticoagulants was significantly higher in cases than in controls. A history of ACS in the previous 30 days and the administration of acetylsalicylic acid (ASA) were related to a lower incidence of sCT. Valvular surgeries were much more frequent in cases than in controls, while the opposite was true for CABG surgeries. Other factors, such as a longer perfusion time and the use of tranexamic acid, were significantly related to the appearance of sCT. Red blood cell (RBC) transfusion, in both the intra- and post-operative periods, was associated with developing the condition, as was the administration of fresh frozen plasma [Tables 3 and 4**]**.

The time at which postoperative anticoagulant therapy was started was noted in 73 individuals, with no significant difference being found between cases and controls. There was no significant difference between the two groups with regard to the start time of postoperative warfarin (described in 59 subjects). The start time of postoperative antithrombotic prophylaxis could be determined in 156 patients, without significant differences between cases (median 29 h, range 9–334) and controls (median 28 h, range 10–100). There was also no difference between groups with regard to the start time of postoperative ASA.

Table 3: Bivariate analysis

	Cases (*n*=80)	Controls (*n*=160)	*P*
Age (years), median (range)	58 (18-79)	61 (18-87)	0.001
Perfusion time (min), average±SD	118.8±49.9	103.8±41.5	0.03
Number of red blood cell units transfused intraoperatively, average±SD	1.1±2.0	0.33±0.76	0.0001
Number of fresh-frozen plasma units transfused, average±SD	1.5±3.7	0.2±1.1	0.002
Maximum INR postoperatively, median (range), *n*	1.6 (0.91-6), 77	1.3 (0.93-6), 126	0.001
Total number of red blood cell units transfused, median (range)	2 (0-46)	0 (0-6)	0.00001
Total postoperative drainage (mL), median (range)	650 (55-10,850)	600 (50-9560)	0.02
Maximum postoperative creatinine (mg/dL)	1.3 (0.6-9.8)	1 (0.5-3.2)	0.00001

Continuous variables with statistical significance . Unless the cell specifies *n*: It is understood that the number of cases is 80 and the number of controls is 160. SD: Standard deviation, INR: International normalized ratio

Table 4: Bivariate analysis

	OR	95% CI	Valor de *P*
Use of preoperative anticoagulants	2.41	1.80-3.22	0.002
Unfractionated heparin	3.69	1.94-7.02	0.03
Warfarin	11.1	5.0-24.4	0.0001
Aortic valve replacement	2.08	1.51-2.86	0.019
Mitral valve replacement	4.73	3.11-7.19	0.0001
Surgery other than CABG	4.69	3.48-6.31	0.0001
Use of tranexamic acid	2.08	1.57-2.75	0.008
Postoperative anticoagulation	9.08	6.60-12.5	0.0001
Unfractionated heparin	2.80	1.66-4.73	0.04
Warfarin	7.15	5.14-9.96	0.0001
Enoxaparin	7.15	3.57-14.49	0.00001
Reintervention within the first 48 h	37.15	13.09-105.45	0.0001
ASA until the day before surgery	0.3	0.2-0.5	0.01
ACS in the 30 days prior to surgery	0.2	0.18-0.35	0.0001
CABG	0.22	0.16-0.30	0.0001

Categorical variables with statistical significance.
CABG: Coronary artery bypass graft, ASA: Acetylsalicylic acid, ACS: Acute coronary syndrome, CI: Confidence interval, OR: Odds ratio

Other factors which were strongly associated with the outcome in the bivariate analysis were having undergone reintervention within the first 48 h postoperatively (significantly greater in the cases) and the maximum value of postoperative creatinine (greater in the cases). Although the drainage tubes remained in place a similar amount of time in both groups (median: 2 days; range: 1–9 days), there was more total chest tube drainage in cases than in controls [Tables 3 and 4].

In the logistic regression analysis, five variables related to the development of sCT were identified [Figure 1]. The Hosmer–Lemeshow test showed a proportion of 88.6% of patients (chi2 = 0.94) who were correctly classified (See receiver operating characteristic curve in Additional File).

Figure 2 shows how the cases were distributed throughout the tracking days.

The complication of sCT was associated with very high morbidity and mortality: nine of the cases died (11.2%) versus none of the controls (0%). The incidence of operative-site infection was 10% for cases versus 0.6% for controls.

Discussion

This study identified five independent variables significantly associated with the development of sCT: full preoperative or postoperative anticoagulation, surgery other than CABG, need for RBC transfusion, and surgical reintervention in the first 48 h after surgery.

Our data demonstrate that there is a significant increase in the incidence of sCT with the use of anticoagulants pre- or post-operatively. This factor has been identified in various prior reports, but not in all. Kuvin *et al*. found that 84% of patients presenting with CT had been receiving anticoagulation therapy with warfarin or heparin in the first 3 days after cardiac surgery and that prolonged partial thromboplastin time was significant in predicting CT.[13] Malouf *et al*. reported that the incidence of pericardial effusion was significantly higher in patients under full anticoagulation than in those without anticoagulation.[14] However, in their work, Ashikhmina *et al*. noted that in most publications, the association of anticoagulant use with an increased risk of pericardial effusion is based only on the documented administration of anticoagulants in a large proportion of patients with effusions, and few studies report the anticoagulation status of control patients without effusions.[4] In their work, these authors found no association between anticoagulation and the presence of pericardial effusion, but they did identify nine independent factors: increased body surface area, immunosuppression, pulmonary thromboembolism, renal failure, urgency of surgery, type of surgery, and prolonged cardiopulmonary bypass, which were risk factors for developing pericardial effusion. Among their conclusions, they underscored the fact that despite the introduction of new protocols for anticoagulation and new antiplatelet drugs during

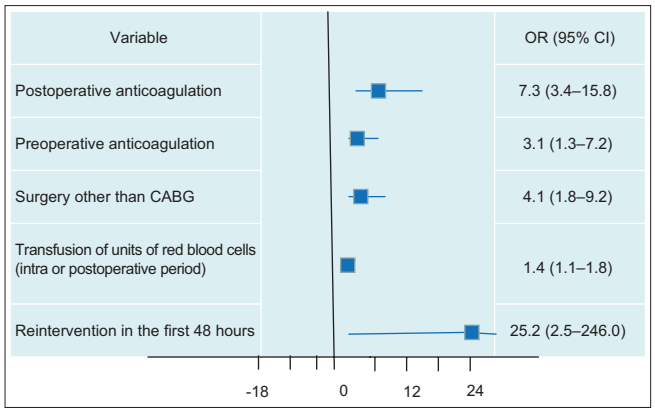

Figure 1: Multivariate analysis. Factors associated with postoperative subacute cardiac tamponade

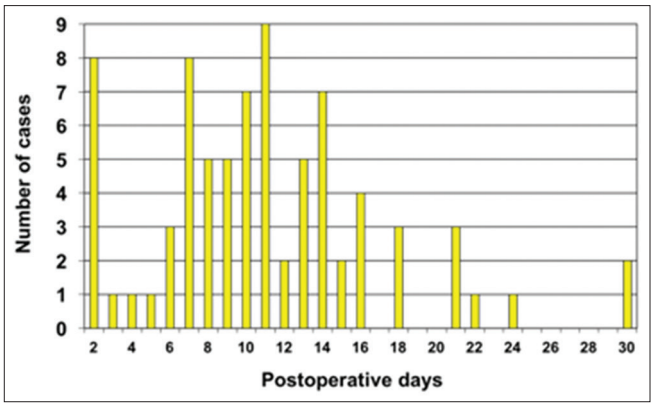

Figure 2: Distribution of the number of cases by time

the study period (13 years, ending in 2005), they found no significant change in the incidence of postoperative effusions from the beginning to the end of the study. Comparing these findings with our group's results, it is noteworthy that we analyzed sCT exclusively, while they evaluated 327 patients with any postoperative pericardial effusion (<30 days) and only 94 of these (<30%) had clinical and echocardiographic criteria for CT.[4] Perhaps, complete anticoagulation could act by increasing the volume of pericardial effusion, or its blood content, thereby generating sCT. In Pompilio *et al*.'s publication,[15] the researchers argue that the evidence of association between anticoagulant therapy and increased CT risk is controversial. They identified several independent predictors of pericardial effusion requiring drainage: additive logistic EuroSCORE, surgery on the ascending aorta, double aortic and mitral valve replacement, platelet transfusion, and postoperative renal failure. The only protective variable isolated was the administration of ASA before surgery. Furthermore, in the recent work by Khan *et al*.,[16] The authors did not directly study the effect of anticoagulation on postoperative pericardial effusion, but they concluded that late CT affects mostly young, low-risk patients undergoing valve surgery.

In our series, as well as in Ashikhmina *et al*.'s[4] and Pompilio *et al*.'s[15] data, administration of ASA seemed to confer a protective effect against CT, which these authors attributed to the drug's anti-inflammatory action. Although ASA, even at low dosages, has anti-inflammatory activity,[17] it is more probable that this finding is a confounding factor, given that patients receiving ASA have a lower risk of CT essentially because the surgery they undergo is usually CABG, as opposed to valvular or combined surgeries. The fact that in our data almost half of the controls had a history of ACS within the previous 30 days, versus 20% of the cases, reinforces this hypothesis. Similarly, Khan *et al*. reported that 63.8% of patients without CT had coronary disease versus 33.3% of patients with late CT ($P < 0.001$).[16]

It is very possible that heterogeneous inclusion criteria in each series (including the lack of a uniform definition of what constitutes a subacute or late CT) are related to the nature of the associated factors.

We created a variable for surgeries other than CABG [Table 2, non-CABG surgeries], including isolated valve procedures or CABG-associated aortic valve replacement, isolated or CABG-associated mitral valve procedures, cardiac transplant, and other combined surgeries. In our study, the majority of sCT cases were non-CABG surgeries, while CABG was the predominant surgery in the controls. The multivariate analysis demonstrated that non-CABG surgery was one of the five variables strongly related to the appearance of sCT. We believe that the type of surgery may explain not only the apparent protective effect of ASA but also other confounding variables, such as body weight and sex (Kuvin reported a hazard ratio of 2.0 for CT in women).[13] In our study, CABG was the qualifying surgery in 61.9% of men but in only 32.9% of women. These data provide evidence that supports surgery type being a very important variable in postoperative sCT incidence.

A finding not well studied previously, and demonstrated by our data, is that this risk seems to be similar with all anticoagulants (unfractionated heparin, warfarin, or enoxaparin). It is important to note that we defined that patients were under antiplatelet or anticoagulant therapy if they had received the last dose of medication within the time period defined in Table 1. Other studies do not mention this information, which may explain why this association is not always found. We also evaluated the time of postoperative initiation of anticoagulants; although it is reasonable to expect that this time is related to the development of sCT, we did not find a significant difference in this variable.

Given that there was no difference in the time of initiating postoperative antithrombotic prophylaxis between cases and controls, this study provides additional evidence that this prophylaxis, when initiated the day following surgery, does not increase the risk of clinically significant

pericardial effusion.[18] This finding is important given that thromboprophylaxis is frequently postponed for fear of increasing the incidence of hemorrhagic complications or CT.[19]

RBC transfusion and surgical reintervention in the first 48 postoperative hours were identified in the multivariate analysis of our data as variables were significantly associated with the appearance of CT. Both variables reflect that increased perioperative bleeding is likely the causal factor most closely related to sCT. We found a significant difference in the total volume of tube drainage: 650 cc median (range: 55–10.850) in cases versus 600 cc (50–9560) in controls ($P = 0.02$). The hypothesis that RBC transfusion *per se* (as opposed to being an indicator of increased bleeding) may be causally related to the appearance of sCT, cannot be discarded. We found significant differences in the number of units of RBCs and plasma transfused in both the intraoperative and postoperative periods. Coagulopathy related to polytransfusion has been well described[20] and is explained mainly by the dilution of clotting factors, thrombocytopenia, and hypothermia. Moreover, RBCs themselves contribute to hemostasis, promoting the marginalization of platelets against vessel walls and decreasing their availability to act at the vascular lesion site. It has also been shown that RBCs increase the generation of thrombin and modulate the biochemical and functional reactivity of activated platelets.[21]

An important additional finding in our research was the high incidence of surgical site infections in the cases, which may be related to the need for higher volumes of RBC transfusions. The systematic review by Ang *et al.* found that allogeneic RBC transfusions were associated with an increased risk of mediastinitis in patients undergoing cardiac surgery,[22] likely through downregulation of the immune response.[23] In their study, Falagas *et al*.[24] also found that plasma transfusion was independently associated with the development of nosocomial infections, including mediastinitis. Marked LV dysfunction with a large pericardial cavity (especially heart transplants) has been noted to be an independent risk factor for pericardial effusions.[4] This association is likely a result of a combination of factors including the potential space created when an enlarged, diseased heart is replaced with a normal organ. We did not include an evaluation of ventricular function or the size of cardiac cavities as a study variable beforehand, and the number of patients undergoing cardiac transplant in this sample ($n = 3$) did not allow for drawing conclusions.

The practical implication of this study's findings regarding patient management is obvious. Although there was a tendency toward early reintervention in those patients with significant bleeding in our surgical group, it is clear that even after a surgical revision and despite affecting the required hemostasis, the patient still carried a risk of sCT beyond the first 48 postoperative hours.

The temporal distribution of cases has an important implication, which is that, it could support the implementation of a routine, periodic echocardiogram protocol in the first three postoperative weeks in patients with the risk factors we identified in the multivariate analysis.

Among the study's limitations, it should be recognized that this analysis is based on a historical cohort and represents the experience of only one institution. Even so, all required variables were entered into the database concurrently, using prespecified definitions, and the majority of factors that may be related to sCT development were considered. Another weakness of the study is that we did not systematically record the diagnostic criteria of postpericardiotomy syndrome in our patients.

The study has strengths, mainly the inclusion of all types of surgical procedures and tracking up to 30 days, which allowed a description of postoperative CT incidence in its full temporal spectrum.

In conjunction with the factors identified in this research, the analysis shows good operative characteristics regarding the explanation of the relationships of these independent variables with the patients' case or control status. However, given the retrospective nature of the data and the limited sample size, it was not considered appropriate to describe this result in terms of a predictive model. Hopefully, future large-scale research will incorporate these variables.

Conclusions

We have successfully identified that factors such as full pre- or post-operative anticoagulation, surgery other than CABG, need for RBC transfusion, and surgical reintervention in the first 48 h following surgery are predisposing conditions for the appearance of sCT. This information is highly useful for recognizing an individual's risk of developing sCT and thus implementing measures tending to favor the condition's early detection. It is probable that implementing a routine echocardiogram protocol in the first three postoperative weeks in high-risk patients could lead to a reduction in morbidity and mortality.

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Conflicts of interest

There are no conflicts of interest.

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