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Management of Catheter-Related Injuries to the Coronary Sinus

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Background. **Although coronary sinus catheter-related injuries (CSCRIs) are rare, they are potentially lethal. The purpose of this study was to evaluate such injuries, the repair methods used, and to identify related risk factors for mortality.**

Methods. **A retrospective review of 10,552 cardiac surgical procedures from 1995 to 2000 in which retrograde cardioplegia was used revealed 10 cases (n = 10) of CSCRIs (0.095%) at our center. These injuries occurred during coronary bypass, valve replacement, and combined procedures. Management included direct suture, vein patch, or pericardial "on-lay" patch repair.**

Results. **Two deaths occurred (20% mortality) from failure of CSCRI repair; 8 of 10 injuries (80%) were successfully repaired. One patient had delayed, localized pericardial tamponade, which resolved spontaneously. Two patients had recurrent angina that was assessed 3 and 5 years later by coronary angiography; the coronary sinus was found to be patent in both cases. The remaining 6 patients have been asymptomatic.**

Conclusions. **Repair of CSCRIs can be challenging as it can be complicated by inadequate myocardial protection, inadvertent coronary artery injuries, and possibly, subsequent coronary sinus thrombosis. Repair of CSCRIs should be carried out on an arrested, well-protected heart providing secure hemostasis and coronary sinus patency.**

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Use of retrograde cardioplegia with catheters placed (directly or indirectly) into the coronary sinus is widespread. Coronary sinus catheter-related injury (CSCRI) is rare (0.6%) [1], but carries a potential mortality [2]. This study is a retrospective review of our experience with CSCRIs; we wanted to identify risk factors for mortality and suggest an optimal strategy for managing this complication.

Material and Methods

In a retrospective review of 10,552 cardiac surgical procedures in which coronary sinus (CS) cardioplegia was used from January 1995 to December 2000, 10 cases (0.095%) of CSCRIs were identified. For each case, the location and nature of the injury, the method of repair and its rationale, and early outcome were analyzed. Late follow-up was accomplished by telephone contact with all surviving patients.

The patients were 5 men and 5 women aged 63 ± 15 years. Four had aortocoronary bypass grafting (CABG), 2 had isolated valve replacement, and 4 had a combined CABG and valve procedures (Table 1). The same type CS cannula was used in all cases (RMI, RCO14 MIB, Edwards Lifescience LLC, Irvine, CA), with the same infusion protocol (150 mL/min, with CS pressures never exceeding 40 mm Hg).

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All CSCRIs were detected intraoperatively at different times during the procedures (Table 1). For this retrospective study, we could not obtain data regarding first time or repeated insertion attempts or the volume used for inflation of the CS catheter balloon.

Location of Coronary Sinus Catheter-Related Injuries

For purposes of injury classification, the CS was arbitrarily divided into three sections. Most (n = 6) of the CSCRIs occurred in the middle section of the CS, followed by the proximal (n = 2) and distal (n = 2) sections (Fig 1).

Mechanism of Injury and Time of Discovery

Proximal injuries (n = 2) were perforations of the CS wall by the catheter tip, occurred during catheter insertion, were discovered early (unexpected bleeding on cardioplegia infusion and very low CS catheter-recorded pressures), and could be visualized directly. Middle CSCRIs (n = 6) were CS ruptures or "blowouts," which occurred during infusion of retrograde cardioplegia while the heart was inverted for exposure of the obtuse marginal coronary arteries. All middle CSCRIs (except one) were discovered during the procedures as large atrioventricular groove hematomas. Distal CSCRIs (n = 2) were discovered late, just before or after CPB separation, despite the fact that usual infusion pressures had been recorded. They were most likely the result of balloon overinflation and overpressurization of the CS lumen from "wedging" the CS catheter too distally, or in a small CS branch.

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Table 1. Data From Patients With Coronary Sinus Catheter-Related Injuries

Age/Sex	Procedure	Type and Site of CS Injury	Method of Repair	Outcome
48/M	CABG × 3	"Blowout" middle portion	Ante C-plegia, vein patch repair and with PPFP coverage	Uncomplicated recovery, LOS 6 days
73/M	CABG × 3, MV plasty	Distal CS portion, discovered before CPB separation	Ante C-plegia coverage with PPFP, anchored with fine (6-0) sutures	Uncomplicated recovery, LOS 7 days
68/F	MVR	Tip perforation, proximal CS section, discovered during catheter insertion	Repair with fine sutures, coverage with PPFP, insertion of smaller MV prosthesis, retaining of posterior leaflet	Uncomplicated recovery, LOS 8 days
72/F	AVR, CABG × 1	Tip perforation, proximal CS, discovered during catheter insertion	Repair with fine (6-0) sutures, coverage with PPFP	Uncomplicated recovery, LOS 8 days
75/M	CABG × 3, RCA endarterectomy	Middle CS, "blowout," discovered during C-plegia infusion	Antegrade C-plegia, vein patch repair, coverage with PPFP	LOS 8 days
67/F	CABG × 3	Middle CS "blowout," discovered after all distal anastomoses performed	Ante C-plegia, vein patch repair with fine sutures (7-0), coverage with PPFP.	LOS 9 days
72/F	CABG × 3	Middle CS, "blowout", discovered after X-clamp off	Repaired on CPB with the heart arrested, covered with autologous, pericardium, sutured around the hematoma.	Discharged. Readmissions for pericardial mass compressing inferolateral portion of the RV. Improved slowly.
71/F	AVR (redo)	Distal CS, discovered after separation CPB and protamine administration	On CBP, antegrade arrest. Autologous pericardium, sutured around the hematoma and "Bio-glue" application	Uneventful recovery, LOS 8 days
79/M	AVR, CABG × 2	Middle CS "blowout," discovered after X-clamp off	Repair with pericardial "onlay" patch with fine sutures, on a CPB-supported, beating heart	Inotropic agents, IABP support, open sternum. High levels of cardiac enzymes. Massive stroke, death from sepsis (POD9)
71/M	AVR, CABG × 3	Middle CS "blowout," discovered after separation from CPB and protamine administration	Initial repair with pledgeted sutures, on a non-CPB-supported ejecting heart, followed by coverage with autologous pericardium on a CPB-supported, beating heart	Inotropes, IABP support open sternum. Rebleeding. To surgery, RV hemorhagic, infarction, and rupture. On CPB. Repair with Hemashield patch. IABP support, high-dose inotropic agent high cardiac enzyme levels, Death (48h)

AVR = aortic valve replacement; C-plegia = cardioplegia; CABG = coronary artery bypass graft; CPB = cardiopulmonary bypass; CS = Coronary sinus; IABP = intraaortic balloon pump; LOS = length of stay; MVR = mitral valve replacement; POD = postoperative day; PPFP = pedicled pericardial fat pad; RCA = right coronary artery; RV = right ventricle; X-clamp = cross-clamp.

Methods of Repair

Proximal CSCRIs were repaired directly with fine Prolene (Ethicon, Somerville, NJ) sutures during a period of cardioplegic arrest. The repair was reinforced with a pedicled pericardial fat pad (PPFP) [3] (Table 1).

Of the six middle CS "blowouts," it was possible to identify the edges of the CS injury in three (n = 3), keeping the CS catheter in place as a "stent" for the repair. A vein patch was sutured to the edges of the injury and the area was covered with a PPFP (Fig 2A). In the remaining three middle CSCRIs, an autologous pericardial patch was sutured around the subepicardial hematoma, because identifying the edges of the CS injury of itself was impossible (Fig 2B). One repair was carried out durinmg CPB with an arrested heart. In one case the repair was carried out on a CPB-supported, beating heart; in the third case the pericardial "on-lay" patch repair followed a failed direct, pledgeted suture repair of the CSCRI on an ejecting heart after CPB separation (Table 1).

Distal CSCRIs were initially observed. When a "weeping" hematoma surface was noted after the protamine administration, either a PPFP or a pericardial patch was used to cover the hematoma. The repair was reinforced with "Bio-Glue" (Cryo-Life, Kennesaw, GA) in one instance.

Results

Eight of 10 CSCRIs (80%) were repaired successfully. All successful, uncomplicated repairs were carried out on an arrested, vented heart with direct fine suture, or with use of a vein or a pericardial patch.

Two deaths (20%) were recorded. Both patients had an

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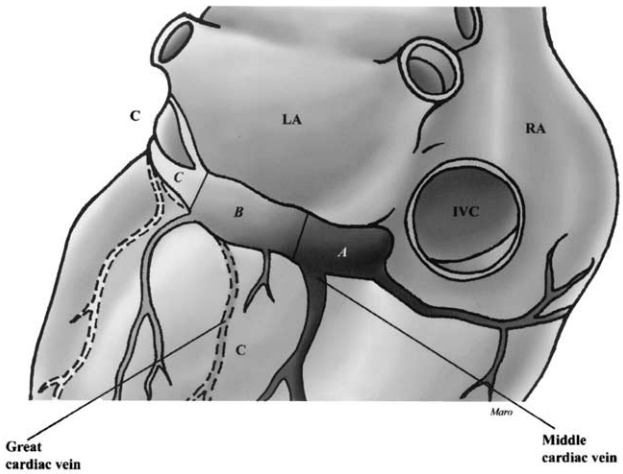

Fig 1. Coronary sinus (CS) sections. A = *proximal CS. From the ostium up to the junction with the middle cardiac vein, the proximal CS is almost never covered by fat and is close to major coronary branches from the right coronary artery system.* B = *middle CS, which lies in the atrioventricular groove, adjacent to the circumflex coronary artery, around the base of the left atrial appendage. This part is almost always covered with epicardial fat and receives a number of venous tributaries from the lateral wall of the left ventricle.* C = *distal CS, which extends from the distal base of the left atrial appendage, and receives the great cardiac vein from the anterior left ventricle. (*IVC = *inferior vena cava;* LA = *left atrium;* RA = *right atrium.)*

"on-lay" pericardial patch repair of a middle CS "blowout" injury on a CPB-supported, beating heart, after an unsuccessful direct suture repair after separation from CPB. Both had increased myocardial enzyme activity (CK-MB and troponin levels), despite the presence of patent and functioning coronary grafts, and they required intraaortic balloon pump counterpulsation, inotropic support, and open sternum because of unstable hemodynamics. One of these patients died from low cardiac output syndrome, after a Hemashield (Boston Scientifics, Watertown, MA) patch repair of an inferior right ventricular wall hemorrhagic infarction, and the other from sepsis, after a large hemispheric stroke (Table 1).

One patient with middle CS "blowout" injury successfully repaired with an on-lay pericardial patch developed late localized pericardial tamponade, with caval hypertension, pedal edema, and right ventricular dysfunction. The patient refused surgery and required several hospital readmissions, but finally improved with medical treatment.

Coronary sinus patency was verified by catheterization of the CS in 2 of 8 survivors who underwent cardiac catheterization for recurrent angina 5 and 3 years later. These 2 patients had direct vein patch repair of their injuries. The remaining 6 patients remained in good condition (telephone interview).

Comment

According to Menache and colleagues [1, 4], most CS injuries are caused by overpressurization of the sinus, overinflation of the CS catheter balloon, or traumatic catheter insertion. Each of these mechanisms produces an injury, the outcome of which depends on its magnitude, location, and the effectiveness of repair.

Typically, proximal CSCRIs are caused by forceful catheter insertion or repositioning attempts (especially on an empty, arrested heart) resulting in punctate, or "tip" perforations of the CS wall [5, 6]. Bright red bleeding from the back of the heart and inability to achieve adequate pressure during cardioplegia infusion suggest the presence of a CSCRI. A direct, accurate fine suture

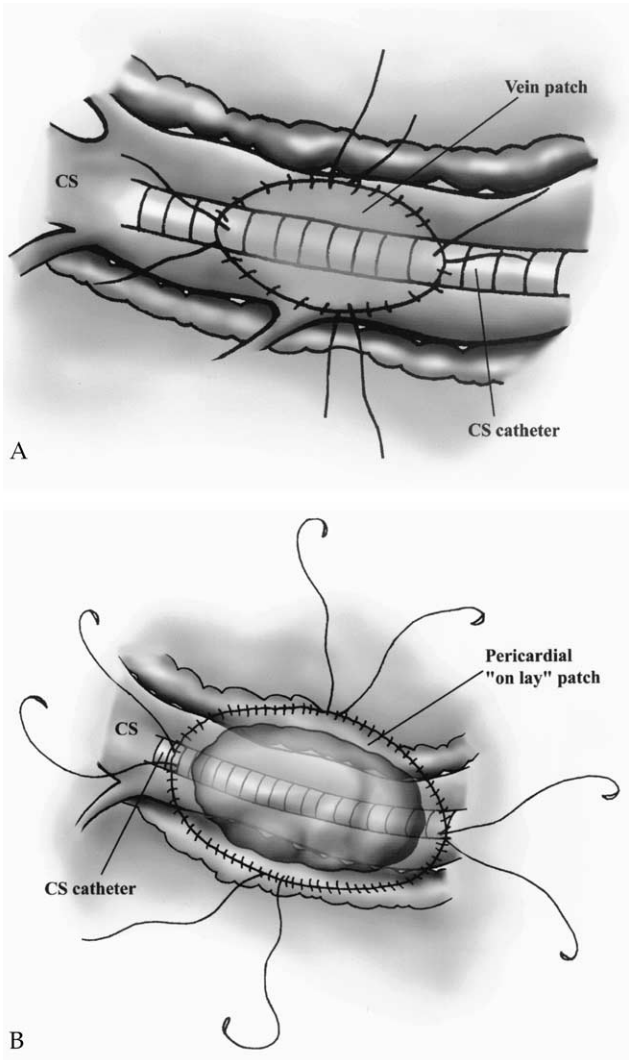

*Fig 2. (A) Vein patch repair of a coronary sinus catheter-related injury with fine Prolene sutures. (B) The "on-lay" pericardial patch repair. The pericardial patch is sutured on the epicardium around the hematoma with fine Prolene sutures. The size of the coronary sinus (*CS*) catheter is reduced for clarity in this figure.*

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repair carried out on an arrested, well-protected heart suffices most of the time [5, 6].

Major rupture, the so-called blowout injury, usually occurs in the middle section of the CS due to either overpressurization during infusion of cardioplegia at excessive flow or by balloon overinflation resulting in stretching and tearing of the walls of the sinus, especially when elevation of the apex for circumflex coronary artery exposure is used [6]. This development is most relevant in hearts with septal hypertrophy, and most often presents as a large AV groove hematoma. Repair of these injuries is difficult because the hematoma obscures the actual margins of the CS injury, tissues are fragile, and the proximity to the circumflex coronary artery branches makes injury to these arteries possible. It is essential to abandon all CS cardioplegic infusions, leave the CS catheter in place, carry out the CSCRI repair, and complete the planned cardiac procedure with another cardioprotection strategy [4–8].

If injury margins can be identified, a direct repair can be accomplished, or a vein or pericardial patch can be used if a major tissue defect exists. If the margins of the defect cannot be identified, a pericardial patch sutured on the epicardium around the overlying hematoma, keeping the CS catheter in place during the repair, has been recommended by some authors [7] to display the edges of the injury and serve as a "stent" to avoid CS lumen distortion. Although we and others [5–10] have used the "on-lay" pericardial patch method successfully in a number of cases, CS lumen distortion from outside compression and persistent bleeding from high intraluminal pressures [6] was noted. We maintain as others have that postrepair CS distortion sets the stage for CS thrombosis [11].

Coronary sinus thrombosis has been proven experimentally to cause electrocardiographic, enzymatic, and histologic changes similar to hemorrhagic infarction [12, 13] and can be a potentially lethal complication of any CS injury [14–17]. Although autopsy was not performed for the 2 deaths in our series, direct inspection, cardiac enzymes, and echocardiography were suggestive of hemorrhagic infarction related to possible thrombosis of the CS.

Coronary sinus catheter-related injuries in the distal CS are usually discovered late, unless overt bleeding occurs from rupture of the hematoma during cardioplegia administration. Most authors recommend observation if no overt bleeding occurs [1, 4, 5]. In the event of bleeding, coverage with a pericardial patch or a PPFP can be successful.

Undoubtedly the best treatment of these serious injuries is prevention [4, 6, 18, 19] and involves at least four considerations. First is avoidance of forceful CS catheter insertion. In reoperations, before inserting the CS catheter, adhesions to the inferolateral part of the heart should be released, to avoid distortions of the CS lumen. When inverting the heart, deflation of the catheter balloon or discontinuation of retrograde cardioplegia infusion (for flow inflatable balloons) is warranted. Last, careful and continuous monitoring of the pressure recordings during cardioplegia infusion is necessary, with immediate adjustments of the catheter position when either extremely high or low pressure readings are noted.

Conclusion

Coronary sinus catheter-related injuries are rare, frequently difficult to repair, and are potentially lethal, due to inadequate myocardial protection, inadvertent coronary artery injuries, and possibly postrepair CS thrombosis. Meticulous technique to prevent CSCRI is essential. Nonetheless, if a CSCRI occurs, an alternative cardioprotective plan should be implemented to carry out the originally planned procedure.

A meticulous repair carried out on an arrested, empty, and well-protected heart is recommended to achieve secure hemostasis and CS patency. It is essential that the surgeon resists any temptation to repair a CSCRI on a beating, non-CPB-supported heart, especially if the injury is discovered late after CPB separation and protamine administration.

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Mark your calendars for the Fortieth Annual Meeting of The Society of Thoracic Surgeons, which will be held in San Antonio, Texas, January 26–28, 2004. The program will provide in-depth coverage of thoracic surgical topics selected to enhance and broaden the knowledge of practicing thoracic and cardiac surgeons. Traditional abstract presentations as well as topic-specific ancillary sessions and courses will make up the continuing medical education opportunities that will be offered at the Fortieth Annual Meeting.

Advance registration forms, hotel reservation forms, and details regarding transportation arrangements, as well as the complete meeting program, will be mailed to Society members. Also, complete meeting information will be available on the Society's Web site located at http://www.sts.org. Nonmembers wishing to receive information on attending the meeting may contact the Society's Secretary, Gordon F. Murray.

Abstracts for the meeting must be submitted electronically. The electronic submission form may be accessed at http://www.ctsnet.org/abstracts/sts. There is no charge for submitting your abstract electronically. The electronic abstract submission deadline is August 18, 2003, at 12 Midnight, CDT. Video submission deadline is August 8, 2003. All abstracts must be submitted using the electronic forms located at http://www.ctsnet.org/abstracts/sts. Any questions may be directed to the STS headquarters.

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