
Case Reports: Thrombus Formation in the Extracorporeal Circuit

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Abstract

Two cases in which thrombus was formed in the extracorporeal circuit are presented. In the first case, a small thrombus formed from packed cells added to the prime adhered to the front wall of the arterial reservoir. It proved to be incidental to the progress of the surgical procedure and recovery of the patient. In the second case, a large quantity of protaminized blood was aspirated into the circuit post-bypass. Complications at the surgical field required the immediate re-institution of bypass and, despite changing the oxygenator, bypass management was severely limited. The patient subsequently expired. As these cases represent different ends of the spectrum in regards to their origin, effects on bypass management, and patient impact, their similarities and differences are identified and discussed. The importance of circuit design and the reserve capacity of its components when presented with these abnormal situations is reviewed.

Case I

A 51 year-old woman with three-vessel atherosclerotic coronary artery disease was brought to the operating room for elective coronary revascularization. Pre-operative weight was 58 Kg. and serum chemistries and coagulation profiles were normal.

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The extracorporeal circuit consisting of an Optiflo II oxygenator,^a BCR-3000 cardiotomy reservoir,^b custom tubing pack with RF-10 recirculation filter^c and AF-10 arterial filter^b was assembled and primed with 1800 cc of LR/D₅W and 500 cc of 6% Hetastarch (Hespan). These fluids were admitted to the circuit through a filtered^c rapid prime line attached to the cardiotomy reservoir.

Shortly after induction of anesthesia with fentanyl, pancuronium and oxygen, the patient's blood was sampled for blood gases, sodium, potassium, hematocrit and baseline activated clotting time (ACT). The post-induction hematocrit was 29%, yielding an estimated bypass hematocrit of 19% before administration of cardioplegic solution.

The pump was turned off after debubbling and 750 cc of the crystalloid Hespan solution was drained from the coronary port of the arterial reservoir. A pharmacologic cocktail consisting of 3 gm Keflin, 500 mg Solu-Medrol, 500 mg ascorbic acid, 12.5 gm Mannitol and 5000 IU sodium heparin were added to the arterial reservoir. Two units of packed red cells anticoagulated with CPDA-1 solution were added to the reservoir through the rapid prime line and rinsed with 150 cc of LR solution.

As the packed cells were being added, the patient was heparinized with 18,000 IU. The tubings were passed to the sterile field and the patient was cannulated with a Sarns two-stage venous return cannula^d in the right atrium and a 21 F aortic cannula in the ascending aorta.

After cannulation but prior to initiation of bypass, three proximal anastomoses of saphenous vein to the aorta were accomplished with partial aortic occlusion. An ACT drawn 8 minutes after

administration of heparin yielded a result of 597 seconds as compared with the baseline value of 133 seconds.

Bypass was initiated 48 minutes after heparinization. A long, thin dark mass was immediately detected on the interior wall of the oxygenator, about three inches above the arterial outlet port. The formation was horizontally situated with the ends apparently anchored to the polycarbonate oxygenator shell. The center portion of the suspected thrombus oscillated in synchrony with the rotation of the arterial pump head. It was closely examined and evaluated for size, appearance and mobility. Since the formation did not expand or fragment, a decision was made not to change the oxygenator. The mass was visually monitored carefully and frequently. The surgical team was notified of the situation and constant monitoring of the ACT was achieved.

The case proceeded according to normal protocols. The patient was cooled to 30°C rectal, the aorta cross-clamped and heart arrested with cold potassium cardioplegia. Four distal anastomoses were completed and rewarming was begun 45 minutes into perfusion time. An additional 8,000 units of heparin were used to maintain the ACT between 469 and 536 seconds. A third unit of packed cells was added to maintain the hematocrit above 25%. Separation from bypass occurred without incident after 87 minutes of perfusion. The total volume of the arterial reservoir and venous tubing was transfused to the patient, allowing visualization of the formation. After decannulation of the aorta, the arterial pump head was reversed to drain the arterial blood into the reservoir for cell washing. The blood returning to the arterial outlet port caused fragmentation of the suspected clot.

The oxygenator was set aside and the section with the adhered structure was excised. The formation was rinsed gently from the surface with saline into a specimen container. There were no apparent wall irregularities on the removed section of the oxygenator. The specimen was identified as fibrin by microscopic examination in the Surgical Pathology Department.

The patient was transferred to the Surgical Intensive Care Unit (SICU) after wound closure. Her recovery was uncomplicated.

Case II

A 74 year-old male with a previous history of myocardial infarction presented with crescendo angina and electrocardiographic evidence of anterior ischemia. Left heart catheterization and selective coronary angiography illustrated three vessel atherosclerotic disease and mitral regurgitation secondary to papillary muscle rupture. Emergency surgery was performed for mitral valve replacement and bypass grafting to the right, left anterior descending and circumflex arteries.

The pump system employed was identical to that described in Case I. The priming solution used was 1800 cc of LR/D₅W with 37.5 gm Mannitol, 500 mg ascorbic acid, 25 gm dextrose and 500 IU heparin added. Pre-bypass hematocrit was measured at 30% and two units of packed red cells were added to the prime. Bypass was initiated and the perfusion progressed without incident. The patient was successfully weaned from bypass after 133 minutes and the right atrium was decannulated. The venous line was drained and the blood retransfused through the pump circuit. During decannulation, the friable tissue of the right atrium became lacerated along its posterior aspect. The heavy blood loss from this site was aspirated through the pump suckers for transfusion to the patient. Attempts to repair the laceration were not successful due to poor exposure, and it was decided the re-institution of bypass was necessary to gain proper exposure. As the venous line was being re-primed from the table, clot was noticed in the pericardial well and the surgeon asked the anesthesiologist to terminate protamine infusion and reheparinize the patient. The perfusionist was unaware that any protamine had been given until this point.

Two liters of volume had accumulated in the cardiectomy reservoir and a clot was quickly found in the distal portion of the cardiectomy drain line to the oxygenator. A second tubing was obtained, the cardiectomy drain was double clamped close to the reservoir and transected. In removing the old line from the oxygenator connector, clot was observed inside the port. Examination of the arterial reservoir found the approximately 600 cc of volume to be gelatinous. Emergency exchange of the oxygenator was performed. Total time elapsed from first discovery of clot to reinstatement of flow was

approximately four minutes.

The arterial line pressure was extremely high on re-establishing blood flow. The clamp on the arterial filter bypass line was relocated more distally and thrombus was observed in the clear fluid proximal to the clamp, ruling out bypassing the filter. The extreme hypotension of the patient precluded changing the arterial filter. At a blood flow of 2 LPM, blood was squirting between all tie-banded connections between the arterial pump head and arterial filter. After repair of the atrium and insertion of an intra-aortic balloon, the patient was weaned from bypass. Total time of impaired bypass was 28 minutes.

Consumption of clotting factors led to severe coagulopathy in the post-operative phase. During the first hour after transfer to the surgical intensive care unit, chest tube drainage exceeded four liters. Multiple blood transfusions, including fresh frozen plasma, platelets and packed red cells did not provide hemostasis or adequate volume. The patient was returned to the operating room for exploration at which time heavy bleeding was discovered from the atrial laceration and a chest wall artery in addition to a massive generalized ooze. The atrium and artery were repaired and the patient was transferred back to the SICU. Hemostasis was much improved, though still far from ideal. Two hours after the exploration the patient

suffered an intractable cardiac arrest and expired.

The arterial line filter was preserved and dissected after the case. Gross examination revealed the inflow side of the filter to be filled with thrombus. The outflow side, however, did not have gross clot adhered to any surfaces, including the 25 micron screen itself or the support mesh.

Discussion

In each of these two cases, thrombus was formed in the extracorporeal circuit. Beyond this general statement, however, the characteristics of the two cases could not be more dissimilar. Table 1 identifies the major differences between these unusual incidences.

It was theorized that the thrombus in Case I was generated from coagulation factors in the banked blood, which are present in unwashed packed cells. Apparently, there was a sufficient amount of calcium in the LR solution (3.5 mEq/L) priming the blood filter and administration line to exceed the chelating capacity of the residual CPD of the bank blood. This enabled the free calcium ions to mediate the conversion of prothrombin to thrombin before the volume came into contact with the heparinized solution in the arterial reservoir. Since heparin also acts by blocking conversion of prothrombin, it was powerless in preventing the

TABLE 1

A comparison of various parameters relevant to the incidents of thrombus formation described in the two presented cases.

	CASE I	CASE II
Cause	Freak?	Communication Breakdown
Volume of Thrombus	Less than 1 cc	Greater than 600 cc
Time relative to primary procedure	Pre-bypass	Post-bypass
Consequence	Incidental	Catastrophic

TABLE 2

Identification of extracorporeal circuit components and their performance characteristics which may be adversely affected by thrombus.

COMPONENT	CHARACTERISTIC
OXYGENATOR (Membrane or Bubble)	GAS EXCHANGE HEAT EXCHANGE FLOW RESISTANCE DEFOAMING EFFICIENCY
CARDIOTOMY RESERVOIR	FILTRATION EFFICIENCY DEFOAMING EFFICIENCY FLOW RESISTANCE
ARTERIAL FILTER	FLOW RESISTANCE FILTRATION EFFICIENCY PURGING FUNCTION
CIRCUIT TUBING	PATENCY

thrombus to form once thrombin was present.

Since the protocol used for adding the bank blood products in this case had been used hundreds of times previously without visible thrombus formation, an analytical approach was used to relate this case to all others in which bank blood was added to the prime.

If it is assumed that this case represents the first and only appearance of thrombus from banked blood, it can be attributed to abnormalities in either the blood donor (abnormally high serum calcium) or in the preparation of the product (decreased amount or activity of CPD).

On the other hand, this case could represent the first visualization of a regularly occurring event, which would mean thrombus in previous cases was located in areas not easily visualized, i.e. cardiomyotomy or oxygenator defoamer. This situation would be likely to cause slight, perhaps undetectable, changes in the performance characteristics of system components (see Table 2).

Realizing that either of these possibilities was as likely as the other and lacking the ability to retrospectively prove either one, protocol changes were made to preclude both in the future. Quite simply, LR/D₅W was replaced as a priming solution by Plasmalyte Type A solution, which does not contain calcium. Another answer to the problem would be to add a small amount of heparin (500 - 1000 cc) to each banked unit prior to administration. How-

ever, it was felt that Plasmalyte offered additional advantages (most importantly, it is buffered to a pH of 7.4) and a heparin protocol would have been a slight inconvenience to the perfusionist.

While the uncommunicated administration of protamine in Case II yielded tragic circumstances, it should be noted that the planned surgical procedure had been completed and it was the equally unusual complication at the field that compounded the original error.

The narrative of the case report should provide adequate indication as to the problems encountered in bypass management once the thrombus had formed. They are also identified in Table 2. The critical decision made in this case regarded the arterial line filter. Flowing through the filter media placed severe limitations on bypass management. The eminent dangers presented to the patient by this course of action included: 1) ischemia of all tissues with resultant metabolic acidosis due to the limited flow; 2) increased blood trauma due to the excessive pressure drop across the filter. Alternatives to the decision include bypassing the filter and changing the filter. Although both would eliminate the problems associated with flowing through the filter, each has its own inherent possible complications.

Bypassing the filter would have resulted in certain particulate embolization of the patient, as was proven when the bypass line was checked for

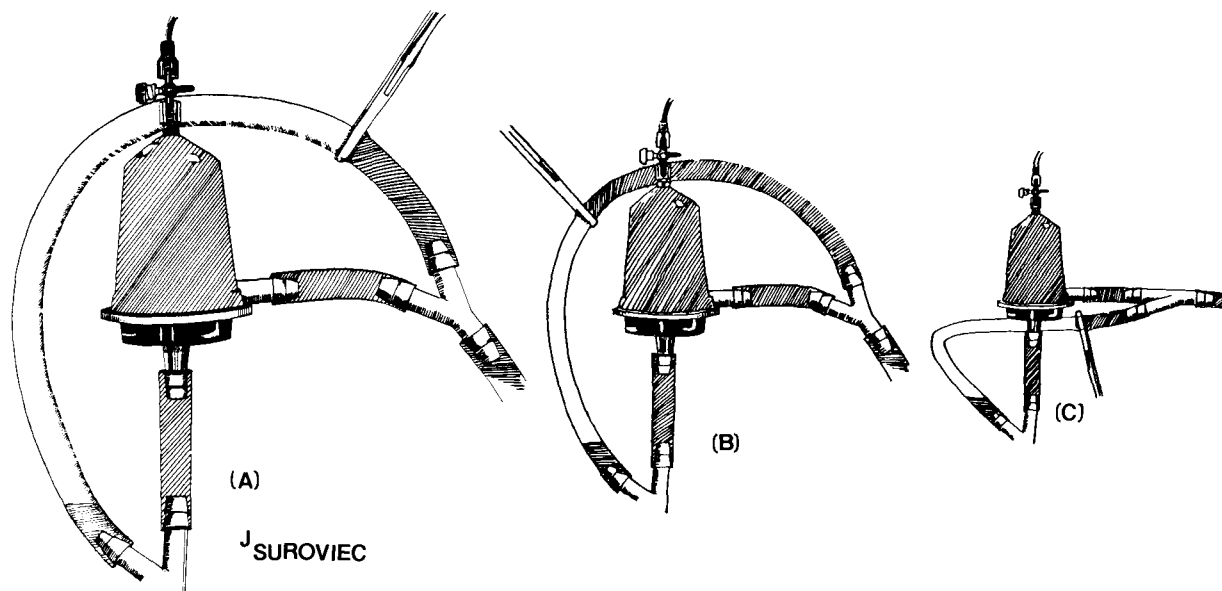


FIGURE 1. Proper placement of the bypass line and clamp are shown in (a) to allow clear visualization of the bypass line. The placements illustrated in (b) and (c) allow formed elements to totally obscure the bypass line.

thrombus. It is important to note that this observation would not have been possible without the specific geometric arrangement of the bypass line and occluding clamp that was employed in assembly of the circuit, preserving a clear crystalloid pathway through the bypass line (Figure 1).

Changing the filter would have presented a multitude of potential complications. The potential for contamination would have been high. Flushing the filter with CO₂ would have been very difficult if not impossible given the circumstances, and failure to do so is associated with neurologic sequelae. Priming and debubbling the filter would be visually obscured if the patient's blood was used, increasing the possibility of gaseous embolus. A brief period of perhaps two minutes of zero flow would have been necessary. And finally, performing the exchange would have consumed a great deal of the perfusionist's time and attention which would be better used in managing other aspects of the situation given the anticipated brevity of the surgical repair.

Perhaps filter exchange would have been possible if a pre-planned protocol were established in anticipation of such an event. This protocol would have to account for the specific system of components.

The possibility of streptokinase infusion to the pump system was mentioned. Such a course of

action would have to be considered experimental as the benefits in terms of line pressure reduction would be anyone's guess. Also, it would compound the already expected coagulopathy.

If the absence of thrombus on the outflow side of the excised filter is not attributed to shedding, the potential benefits of arterial line filtration are proven by this incident. If shedding of thrombus had occurred, one would expect some vestige of thrombus along the seams of filter structures because of the low flow employed. Gross examination revealed nothing, justifying the decisions made in both cases.

Regardless of the extent, any formation of thrombus in the perfusion circuit must be recognized and appropriately responded to. The central location of the thrombus makes access by circulating platelets easy. The platelets would adhere and release their prothrombin activators into the general circulation, potentiating expansion of the existing thrombus or formation of secondary thrombi in either the circuit or the patient's vasculature. This course of events increases the likelihood of circuit component impairment and patient morbidity. Most important is constant monitoring of the ACT. Fibrin formation has been demonstrated at ACT less than 400 seconds,¹ and pre-existing thrombus can only increase the rate of fibrin formation should the ACT fall below accept-

able values.

Conclusion

Though interruption of the clotting mechanism is of primary importance to extracorporeal circulation, intra-circuit clotting can and does occur though the frequency is not available in the literature. The means by which thrombus is introduced to the circuit covers a large range. Once formed, the perfusionist must respond appropriately to thrombus such that patient safety, component per-

formance and progress of the procedure are all considered. A certain amount of compromise must be made between these areas, though patient safety is always foremost. In this respect, arterial line filtration is a very valuable tool, though it too must be properly used.

Reference

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