The appearance of thick spontaneous echo contrast (SEC) and frank thrombosis in the intracardiac chambers and aorta after cardiac arrest has been uniformly associated with adverse outcome.1 The use of transesophageal echocardiography (TEE) during cardiopulmonary resuscitation (CPR) may be extremely helpful in making such diagnosis by enabling an early visualization of SEC. This may lead to the early initiation of specific therapy and improved outcome. A case report is presented of a patient scheduled for peripheral vascular surgery who suffered a cardiac arrest after induction of general anesthesia (GA). During resuscitation, the TEE examination showed severely depressed biventricular function and thick SEC in the ascending and descending thoracic aorta. In response to this finding, 10,000 U of unfractionated heparin were administered intravenously and inotropic therapy was initiated. The SEC gradually cleared, biventricular function improved, and he made a full recovery and was discharged from the hospital a few days later.

CASE REPORT

A 61-year-old man was scheduled for a left femoral-pedal artery bypass graft under GA because of ischemic rest pain and an ulcer on his left foot. His past medical history was significant for coronary artery disease including a history of myocardial infarction (MI) 2 years before surgery and congestive heart failure requiring hospitalization. He had a history of atrial fibrillation (AF) but had been in normal sinus rhythm for the last 6 months. His left ventricular ejection fraction was estimated to be 20% to 25% by a transthoracic echocardiogram performed a few weeks before surgery, and no SEC was reported in the intracardiac chambers. The patient had an automatic implantable cardiac defibrillator (AICD) prophylactically implanted, and preoperative interrogation of the AICD revealed no prior arrhythmic episodes or defibrillations. He also had a history of well-controlled hypertension, insulin-dependent diabetes mellitus, and chronic renal insufficiency (creatinine 2.5 mg/dl). His preoperative electrocardiogram showed evidence of a prior lateral wall MI. A preoperative chest radiograph showed cardiomegaly without evidence of any acute cardiopulmonary process.

Intravenous and radial arterial catheters were placed in the holding area after premedication with midazolam. In the operating room, standard American Society of Anesthesiologists monitors were placed, and GA was induced, after preoxygenation with 100% oxygen, with a combination of etomidate (14 mg), propofol (20 mg), fentanyl (150 µg), and succinylcholine (100 mg). A 7.5 endotracheal tube was successfully placed, and its proper position was confirmed. Anesthesia was maintained with a mixture of 50% oxygen and 50% nitrous oxide and isoflurane.

Approximately 5 minutes after the induction of GA and before placement of a central venous catheter, the patient’s blood pressure acutely dropped from an immediate postinduction level of 135/70 mmHg to 65/40 mmHg. At this time, the rhythm was noted to be AF with frequent ventricular ectopic beats at a rate of 100 beats/min. There was no evidence of ST-segment or T-wave changes. The hypertension and tachycardia did not respond to multiple repeated doses of epinephrine (800 µg), esmolol (50 mg), and fluid boluses (1,000 mL of lactated Ringer’s). Because of a lack of any improvement in the hemodynamics, a TEE probe was placed during resuscitation, and it showed severely depressed biventricular function with moderate tricuspid and mitral regurgitation. No SEC was noticed in the intracardiac chambers or the aorta. The patient’s hemodynamic condition deteriorated further, and he went into asystolic cardiac arrest. Immediate chest compression was started, and boluses of epinephrine were given intravenously. There were brief periods of return of an indeterminate wide complex rhythm with a mean arterial blood pressure in the range of 40 to 50 mmHg. TEE examination showed brief episodes of improvement of cardiac function with epinephrine and CPR without any significant sustained improvement in ventricular or valvular function. An epinephrine infusion was started at 0.5 µg/kg/min. An emergency electrophysiology consultation was requested to determine the status of the patient’s AICD and pacing capability, but interrogation of the device revealed no malfunction or any record of defibrillatory shocks or pacing.

In the meantime, a dense, gel-like SEC was observed in the left ventricle and the ascending and descending thoracic aorta (Figs 1 and 2). Heparin in a dose of 10,000 U was administered intravenously immediately, and the CPR was continued. Echocardiographic examination 2 to 3 minutes after heparin administration showed that the SEC had completely resolved and showed a slight improvement in the biventricular systolic function (Fig 2). At this stage, the patient was in rapid AF with a ventricular response rate of 130 to 140 beats/min and was cardioverted into sinus rhythm at 80 to 90 beats/min with a 200 J external shock after exclusion of thrombus in the left atrial appendage. Because of the lack of sustained improvement in biventricular function despite the increasing epinephrine dose, a milrinone bolus (5 mg) was administered intravenously followed by an infusion (0.5 µg/kg/min). With these measures, biventricular function gradually improved (ejection fraction 20%-25%), and his blood pressure also stabilized (systolic 80-90 mm/Hg). However, because he was still requiring large doses of inotropic medications, an intra-aortic balloon pump was placed, which further improved his hemodynamics. Central venous access was achieved at this stage, and a continuous cardiac output/mixed venous oxygen saturation pulmonary artery catheter (Edwards Lifesciences, Irvine, CA) was inserted. The pulmonary artery systolic pressure was 48/29 mmHg, central venous pressure was 21 mmHg, and the mixed venous saturation was 62%. Because of poor signal quality, the cardiac output could not be calculated at that time with the pulmonary artery catheter. The case was cancelled, and the patient was transported to the coronary care unit (CCU) for further management. The entire episode lasted 1 hour and 10 minutes.

In the CCU, the patient’s condition continued to improve. The inotropes were gradually turned off in the next few hours, and the intra-aortic balloon pump was weaned on postoperative day 1. He was then extubated the same day after being fully conscious, alert, and oriented without any focal neurologic deficit. His cardiac enzymes, including troponin and creatine phosphokinase, were negative for any evidence of myocardial infarction. Further interrogation of his AICD revealed 22 episodes of antitachycardia pacing and 1 internal defibril-
latory shock during the resuscitation. He remained in the CCU for a few
days because of the development of renal failure. His renal function
gradually improved to baseline. The patient was discharged home 6
days after this event.

DISCUSSION

This patient is presented to highlight the importance of
goal-directed resuscitation during a prolonged cardiopulmo-
nary arrest situation. The authors believe that the timely ad-
ministration of heparin soon after the appearance of SEC in the
left ventricle and ascending and descending aorta prevented
frank thrombus formation and a catastrophic outcome. The
availability of TEE to establish the diagnosis and continuously
assess the adequacy of resuscitation also contributed greatly
toward a favorable outcome. In this patient, the etiology of the
dramatic hemodynamic deterioration is not clear, but its rapid
onset is suggestive of an arrhythmia as the causative event. The
multiple antiarrhythmic pacing attempts by the AICD may
actually have led to the ventricular tachycardia and cardiac
arrest.

The appearance of thick SEC and thrombosis in the intra-
cardiac chambers and the aorta during CPR is uniformly asso-
ciated with adverse outcomes. It is an echocardiographic
representation of decreased flow or cardiac output, which cor-
relates with relative blood stasis and is considered a precursor
of frank thrombosis. Although the role of heparin has been
described during CPR, it primarily has been in the setting of
attempted restoration of coronary blood supply during acute MI
and in pulmonary embolism. An unbalanced activation of the
clotting cascade during cardiac arrest may be responsible for
systemic microthrombi in the vital organs leading to multiorgan
failure and an adverse outcome. Heparin administration in this
situation may help in restoration of flow in the microcirculation
and aid in organ protection and improved outcome. To the
best of the authors’ knowledge, this is the first report of the
administration of heparin during CPR given in specific re-
sponse to SEC observed in the aorta on TEE followed by its
complete resolution and a favorable outcome.

Patients with a history of severe congestive heart failure can
have downregulation of their \( \beta \)-adrenoreceptors because of a
chronic catecholamine surge and may require higher than usual
doses of catecholamines for the same hemodynamic response. A
similar etiology may have been responsible for the lack of
improvement with escalating doses of epinephrine in this pa-

Fig 1. A short-axis view of the descending thoracic aorta at 0°
showing spontaneous echo contrast in the aortic lumen.

Fig 2. Multiple composite long-axis views of descending thoracic aorta. (1) Thick spontaneous echo contrast in the descending thoracic aorta before heparin administration. (2) Partially resolved spontaneous echo contrast 1 minute after heparin administration. (3) Completely resolved spontaneous echo contrast 3 minutes after heparin administration.
tient. The decision to commence milrinone bolus and infusion in this situation was based on this pharmacologic principle. The alternative mechanism of action of milrinone complements the inotropic effects of epinephrine and is especially helpful in these situations. The gradual clearing of the SEC may be attributable to multiple factors in this case. Heparin administration may have improved the myocardial microcirculation by preventing thrombus formation and improving myocardial function. Timely administration and the correct choice of inotropes also played a crucial role in preventing an adverse outcome.

Life-threatening hemodynamic instability is considered a category 1 indication for the performance of TEE. The beneficial role of TEE during CPR is well established. TEE has also been shown to disclose new findings during periods of rapid hemodynamic instability and guide crucial decision-making. In this case, the TEE guided timely diagnosis and initiation of specific therapies (ie, heparin and milrinone) that reversed the clinical situation.

Despite prolonged intraoperative hemodynamic instability and CPR, this patient did not suffer any myocardial and/or neurologic damage. The postoperative acute plus chronic renal failure also improved, and his renal function improved to baseline. During the episode of hemodynamic instability, the patient had multiple predictors of adverse outcome (eg, biventricular failure, SEC in the intracardiac chambers, and ascending and descending aorta). It is believed that the favorable outcome was caused by the rapid diagnosis and institution of the correct therapeutic interventions during the episode, which resulted in the restoration of microcirculatory reperfusion and prevented injury to major organ systems.

REFERENCES