Hemodynamic instability during superior vena cava crossclamping: Predictors, management, and clinical consequences

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Particular replacement of the superior vena cava (SVC) has been shown to be a feasible and safe technique in the surgical treatment of selected mediastinal and lung tumors. SVC crossclamping is the most utilized technique for reconstruction of the vessel but it causes intraoperative hypotension that can be severe in some instances. The aim of this study was (1) to evaluate the incidence of hemodynamic instability during SVC crossclamping, (2) to evaluate the clinical impact on the postoperative period, and (3) to search for factors influencing its occurrence.

Clinical Summary
Since January 2002, all hemodynamic data concerning patients who had prostatic SVC replacement at the Thoracic Surgery Department of the European Institute of Oncology were prospectively recorded during surgery with a dedicated software. Severe hemodynamic instability during SVC crossclamping was defined as any hypotension requiring (1) rapid colloid infusion (500 mL/15 min), (2) additional administration of vasoactive agents, (3) suspension of SVC clamping. Intraoperative fluid administration was managed to obtain a mean systemic pressure >80 mm Hg at SVC crossclamping, reducing the risk of cerebral edema. Patients received 15 to 25 mL · kg⁻¹ · h⁻¹ of crystalloids; if the mean systemic pressure of 80 mm Hg was not reached before SVC crossclamping, 2 mg etilephrine bolus was repeated every 5 minutes up to 3 times; if that was ineffective, a continuous infusion of dopamine 5 y · kg⁻¹ · min⁻¹ was started and then adjusted. Before SVC clamping, heparin 5000 U was administered.

Several variables were investigated as possible predictive factors of intraoperative severe hemodynamic instability: age, sex, antihypertensive therapy, preexisting SVC obstruction, need of azygos vein clamping, duration of SVC crossclamping, decubitus (lateral versus supine), amount of fluid infusion before SVC crossclamping.

The study population was composed of 22 patients (15 men and 7 women, mean age 58.8 ± 8.2 years) who had SVC replacement from January 2002 to February 2006.

Fifteen patients had non–small cell lung cancer invading SVC, 6 patients had a thymic tumor infiltrating SVC or innominate vein or both, 1 patient had a metastatic lesion from a leiomyosarcoma infiltrating the apex of the chest. Symptoms of SVC syndrome or signs of collateral venous circulation were present in 7 patients. In 12 cases, SVC resection was performed through a muscle-sparing right thoracotomy and in 10 cases, through a sternotomy or a sternothoracotomy (hemiclamshell). Overall median SVC clamping time was 33 minutes.

Six patients (27.2%) developed severe hemodynamic instability at the time of SVC crossclamping. In 2 cases, hemodynamic instability required SVC clamp removal. In 1 case, instability was due to the clamp position, which was too close to the right atrium. Repositioning of the clamp more distally allowed SVC clamping time of 53 minutes without further instability. In the other case, the patient did not tolerate SVC crossclamping, and the reconstruction was performed with tangential SVC clamping with an infiltrated resection margin on frozen section. Hypotension was managed by fluid rapid infusion in all patients; 3 patients required additional vasoactive agents (epinephrine in 2 cases and dopamine infusion in 1 case). A satisfactory mean pressure was obtained within 6 minutes from SVC clamping in all the cases. Six hours after awakening, Glasgow scale was 15 in all patients.

All 6 patients who developed hemodynamic instability had concomitant definitive or temporary closure of the azygous vein (P = .04) and received more fluids before crossclamping as compared with stable patients (median 65.5 mL/kg vs 21.9 mL/kg, P = .03). Four patients were on antihypertensive therapy (angiotensin-converting enzyme inhibitors in 2 cases, angiotensin II receptor blockers in 2 cases; P = .04), which was stopped 24 hours before surgery.

Multivariate analysis confirmed the role of azygous vein clamping (odds ratio 2.2) and preoperative antihypertensive treatment (odds ratio 2.5) in determining intraoperative hemodynamic instability.

Discussion
Results from this study showed that SVC crossclamping causes a major hypotensive response in about 30% of cases. Its management is difficult because central venous pressure is not a reliable indicator of left ventricle filling during SVC occlusion. Moreover, most patients having SVC replacement are also candidates for extensive and time-demanding lung resections (pneumonectomy or tracheal sleeve pneumonectomy) in which fluid overload is a well-known risk factor for acute respiratory distress syndrome. Therefore, fluid implementation required to maintain intracranial...
Massive hemoptysis: Successful treatment with surgical ligation of the thyrocervical artery

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Bronchial artery embolization (BAE) is an effective treatment for massive hemoptysis and has a primary success rate of 94% owing to blood flow interruption. The morbidity rate of BAE is low compared with that of emergency lung resection.1 We report a case of recurrent massive hemoptysis after primary BAE and further controlled by surgical ligation of the left thyrocervical artery.

Clinical Summary
A 57-year-old man was admitted to our hospital for recurrent hemoptysis. The patient had a history of pulmonary tuberculosis with a previous episode of hemoptysis, which required a BAE, 20 years ago. Coronary artery bypass grafting (CABG) was performed following a myocardial infarction, using both right and left internal thoracic arteries. Six months later, a moderate hemoptysis (10 to 20 mL) occurred without any other symptoms, but because of increasing bleeding, the patient was admitted 4 days later. At admission, there was no respiratory distress and no fever. The chest radiograph showed retractile opacities in the left upper lobe and interstitial infiltrate in the lower left lobe. The fiber-optic bronchoscopy confirmed bleeding from the lingula. Primary angiography demonstrated that the left upper bronchus was vascularized by a collateral artery of the thyrocervical trunk.

References