It is better to be young and healthy than the opposite

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Related article
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In the article presented in this issue of *Kardiologia Polska* (*Polish Heart Journal*) [1], its authors describe the incidence of major complications after abdominal aortic surgery, including myocardial injury after non-cardiac surgery (MINS), acute renal injury (AKI) and bleeding independently associated with mortality (BIMS). Such data may be useful to clinicians participating in preoperative assessment, those taking care of patients in the perioperative time, and those involved in the long-term care of those patients after surgery. Reading about those patients leads me to reflect on my place of work — an intensive care unit — where such patients frequently are admitted after surgery.

First, patients with indications for aortic surgery are all high-risk patients. Parts of the cardiovascular system usually do not get diseased in isolation from other parts — aortic disease is frequently associated with disease in the coronary, cranial, mesenteric, and peripheral circulation. As a group, those patients have a high prevalence of risk factors for complications: hypertension, diabetes, heart failure, previously diagnosed coronary artery disease (CAD), and, if not, in most cases undiagnosed CAD disease. Moreover, most are older (regardless of how we define it). The authors report one-month mortality among all those patients as 6.9% although it may well differ dramatically depending on circumstances, for example, ruptured aneurysm versus elective surgery.

The second issue, and the first surprise, is the frequency of those events: >40% in the case of MINS, >40% in the case of BIMS, and over 15% in the case of AKI. Admittedly, the criteria for finding them are lax — for example, a small increase in troponins or 6-hour urine output below 0.5 cc/kg/h or transfusion of 1 unit of packet red blood cells (pRBC, about 300 ml). No question that if we look for such events, there will be plenty of them.

The third issue is the prognostic significance of the occurrence of those events (MINS, BIMS, AKI). This is another surprise for most of us — having MINS diagnosed (vs. not) is associated with mortality of 12.4% vs. 2.6%; in the case of BIMS the corresponding numbers are 12.3% vs. 1.7% and for AKI 32.6% vs. 1.1%. These are likely eye-opening numbers.

The fourth and fifth issues are of utmost importance. To start, could we improve prognosis by preventing those events? We know the characteristics of those patients (older, with multiple comorbidities). We know at least some of the perioperative factors associated with them — for example, tachycardia, or depth and duration of hypotension — even mean blood pressure below 65 mm Hg and certainly below 55 mm Hg, and even for a few minutes [2, 3]. Could we prevent those events and their consequences? The answer is not clear, and, so far, a series of well-done randomized controlled trials have failed to confirm the usefulness of several interventions, including antiplatelet therapy (ASA), beta-blockers, clonidine, or even a strategy aimed at avoiding hypotension [4]. However, it seems that a strong focus on avoiding hypotension (dehydration, bleeding, excessive sedation) and tachycardia, even as a marker of ongoing problems (dehydration, pain, or not taking pre-op beta-blockers) in the postoperative time, will not hurt and may well help our patients. The importance of rapid recognition of intraoperative and postoperative bleeding

seems crucial. Speed of reaction to those events, as well as speed of reaction to renal dysfunction (usually related to hypovolemia or hypotension), is likely to play a role. All this requires system-wide ability to detect and appropriately react to the physiological phenomena (monitoring technology and presence of trained personnel to react to observations) [5].

The last issue is what to do once we detect those events. This is not entirely clear, and the course of action may depend not only on the nature and severity of complications but also on a host of other factors. Renal injury and the need for transfusion are likely consequences of a one-time event. The issue of troponin elevation in a person at high risk requires more in-depth considerations. There is reasonably compelling although non-conclusive observational evidence suggesting use of ASA and lipid-lowering therapy (statin) in MINS patients. Otherwise, the management (investigations and treatment) may range from "not much" to multi-pronged pharmacological treatment, either permanent or until further risk stratification is completed. Those investigations may vary in scope and intensity from regular stress tests (likely in patients considered very low risk due to lack of major cardiovascular risk factors and minor troponin elevation without ongoing or documented dynamic ECG changes), through echocardiogram checks to look for wall motion abnormalities, pharmacologic stress testing looking for ischemia and its extent, and non-invasive imaging tests (for example, computed tomography angiography), to regular coronary angiography investigations with PCI intervention if needed. What can be done will also depend on patients' values and preferences and the system's ability to "process" a large number of patients, hence there will be different thresholds for action in different geographic areas, further modified by patients' choices. In terms of treatments, attention to and control of modifiable risk factors (smoking, hypertension, hyperlipidemia, and diabetes) are crucial, with consideration given to the use of renin-angiotensin blockade (ACEi or ARB), beta-blockers, and, when indicated, SGLT-2 inhibitors.

In conclusion, from the perspective of an ICU clinician, the occurrence of complications is common and has significant prognostic implications. Once detected, complications should not be ignored but managed with an explicit plan of action, even if it includes 'only' referral for future risk assessment.

Article information

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