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Appendix A. Conference discussion

Dr D.C. Miller (Stanford, California, USA): Nine of the 31 had the left subclavian covered? Is it correct that 9 of the 31 patients required covering the left subclavian artery covered by the stent graft?

Dr Buz: Yes. In 9 of the 31 patients, the origin of the left subclavian artery was primarily overstented; 5 of them later needed bypass.

Dr Miller: Five needed revascularization?

Dr Buz: Yes.

Dr Miller: Because of arm claudication or posterior fossa cerebral insufficiency?

Dr Buz: The arm symptoms, i.e. arm ischemia at exercise or at rest, were the main indications for revascularization.

Dr Miller: It should be remembered that you cover the left subclavian ostium when you have to in an emergency situation, but you can get burned by a posterior fossa stroke if the left vertebral is large and the right vertebral is hypoplastic or occluded. We use the pre-procedure CTA to assess the vertebral artery anatomy and size to determine whether to revascularize (transposition preferred over carotid–subclavian bypass) the left subclavian system before or during the stent graft. Also remember that the left vertebral artery originates directly from the arch in about 2% of patients – a four-vessel arch – which can be very important.

Dr Buz: In patients with polytrauma, especially with severe head injury, the distinction of neurological complication related to occlusion of the left subclavian artery is difficult. But in this series with 31 patients we have not shown any neurological complication because of LSA occlusion. (Comment: In an oral presentation in Stockholm, we reported 31 patients treated with endovascular stent grafting. After the update of all patients until 2007, the paper reports on 39 patients (endovascular group). In this series we have two patients with neurological complications following LSA occlusion, reported in the paper).

Dr Miller: Yet... As your experience gets larger it will happen. Believe me, this is a tricky and controversial subject.

Dr T. Sundt (Rochester, Minnesota, USA): I may have missed this. In your conventional group, how was the repair performed? Was it performed with left heart bypass? Did you give any heparin? Or did you use full cardiopulmonary bypass? I am concerned about your conclusions, because you seem to have an extraordinarily high CVA rate in your control group of conventional surgery. I’m concerned about how much heparin was administered in the control group.

Dr Buz: We had five patient deaths on intracranial hemorrhage in this group. In all patients who were operated on with cardiopulmonary bypass, systemic heparinization was established.

Dr Sundt: In your control group, how was the operation done? Was full bypass with full heparinization used?

Dr Miller: Two were off-pump, or clamp and sew. What perfusion strategy for spinal cord and lower body protection was used for the others in the conventional surgery control group – left heart bypass, total bypass?

Dr Buz: We had only two patients without cardiopulmonary bypass in the conventional group. Except for two patients who were operated on without cardiopulmonary bypass, in all patients femorofemoral bypass was established. Out of them in 9 patients circulatory arrest and deep hypothermia were performed.

Editorial comment

Management of traumatic aortic rupture: endovascular is the winner

Massive deceleration either horizontal or vertical can cause rupture of the aorta typically at the level of the ligamentum arteriosum (the aortic isthmus) distal to the origin of the left subclavian artery. The moment of inertia displaces the relatively mobile heart together with the aortic arch, while the descending aorta tethered to the spine via

the intercostals pedicles remains fixed. With the vast majority of these injuries incurred through car crashes, nearly 80% of the victims die at scene of the accident as a result of complete aortic transection including the adventitia and attached connective tissue [1]. Approximately 20% reach the hospital alive due to an incomplete disruption of the

tunica intima and media. Tensile strength is provided by the intact adventitia, and the parietal pleura contain the hematoma. However, if left untreated, 5–20 % of these patients are at risk of secondary rupture and intrapleural exsanguination within the first week [2]. In surgically untreated survivors, the natural course of aortic rupture is false aneurysm formation with secondary rupture after months or years.

For decades the treatment of choice has been immediate repair by a left thoracotomy, aortic cross clamping and direct suturing, or prosthetic graft interposition. The outcome has been constantly improved and in particular, the risk of paraplegia resulting from open surgery has been decreased to 2% by the use of partial cardiopulmonary bypass, active distal perfusion, and heparin-bonded circuits. However, mortality remains high with 12–26% due to associated injuries [3,4].

Since the first report of an endovascular treatment of traumatic aortic rupture in 1997 [5], numerous studies including our own experience [6,7] demonstrate a high degree of technical success and low complication rates, although based on a small number of patients. The recent report of Buz and co-workers [8] represents the largest series to date, and corroborates the results of the previous studies. It gives, once again, supporting evidence of an improved early outcome following endovascular repair. The literature gives clear proof of a shift in the management from open surgical to endovascular repair. Although endovascular aneurysm repair was first introduced 16 years ago, and a decade from the first stent graft repair of an acute traumatic rupture, no prospective randomized trial has been performed so far. Endovascular repair has several obvious advantages over open repair:

Increased efficiency: operative trauma load is minimal with a relatively short operating time of 60–90 min.

Improved outcomes: overall early mortality seems to be lower by 3–6%, as well as procedure-related morbidity including pulmonary complications. Heparin administration, even if necessary, is minimized and, therefore, less dangerous in patients with cerebral injuries. The endovascular repair is usually performed in a supine position, which is preferable in the presence of instable spine fractures.

Lower risk: paraplegia as an inherent and tragic complication of open repair, is *not* associated with endovascular repair, as aortic cross clamping is avoided.

Minimized delay: the endovascular approach removes the issue of delaying the repair due to its minimal invasiveness.

While the techniques and technology used in endovascular repair will continue to improve, there are a number of challenges that remain, namely:

Design improvement: there is a demand for smaller device diameters (20 or 22 mm) in these mostly younger patients with a small aorta and a tight aortic arch. Improvements of the devices are likely to mitigate device-related complications such as endoleaks and device collapse. Enhanced flexibility in order to better accom-

modate the steep inner curve of the aortic arch and a covered flap at the proximal end of the device designed to fully appose the inner aortic curve, will enable tight sealing of the aortic tear. A scallop at the outer proximal end of the device could avoid covering the left subclavian artery orifice. However, its covering by the actually available devices is necessary in about one third of the cases, and is rarely associated with impaired perfusion of the extremity. Finally the risk of iliac artery laceration could be decreased by smaller introducer sheaths.

Embolization risk: an inherent risk is the possibility of cerebral embolization resulting from manipulations within the aortic arch, although most of these trauma victims are young and their aortas free of atheroma.

Repair suitability: certain types of lesions are not favorable to an endovascular repair. There is such a circumferential disruption with pseudocoarctation because of the risk of dissection of the aortic arch by endovascular manipulations, or an impending instability of the device because of insufficient anchorage. An aortic disruption with extension into the arch also requires an open repair.

Device durability: the main argument against the endovascular repair, or indeed in favor of a randomized trial, is the unknown outcome in the long term. There are some concerns about device collapse, compression of the left main stem bronchus or aortoesophageal fistula, however, they seem to be very rare, and mid-term results are encouraging [9,10].

In summary, reduced early mortality and procedure-related morbidity, combined with the absence of paraplegia, are strong arguments in favor of an endovascular repair. Although early results are favorable, long-term outcome is still lacking. Considering the very encouraging experience we have to date, it is difficult to recommend prospective randomized trials. In the challenging management of traumatic rupture of the aorta, the endovascular approach with a record of improved outcomes, is a winning strategy.

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