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Endovascular Treatment of Chronic Type B Aortic Dissection; Current Updates and Perspectives

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Short Communication

Currently, emergent thoracic endovascular aortic repair (TEVAR) is considered the treatment of choice for complicated acute type B aortic dissection, while there is growing support for its use in treating uncomplicated acute and chronic type B aortic dissection [1]. TEVAR is performed in type B aortic dissection with the expectation that proximal entry tear occlusion and redirection of all ante-grade flow into the true lumen will promote true lumen (TL) expansion, false lumen (FL) thrombosis, and depressurization thereby setting the stage for favorable aortic remodeling [2]. Aortic remodeling refers to reversal in aortic aneurysmal enlargement. Early and long term survival analysis after TEVAR have shown comparable or possibly superior early mortality rates with open surgical repair, but at increased overall treatment failure and re-intervention rates approximating 30%~35% and complications ranging from new dissection, aneurysmal enlargement, degenerative aneurysmal rupture or end-organ malperfusion in 20%~50% [3-9]. Although complications are mostly landing zone related, namely retrograde dissection or type I endoleak, type II and III endoleaks were also reported [10-12]. Van Bogerijen et al. in a propensity adjusted study, identified TEVAR (p=0.046) as an independent predictor of treatment failure for chronic type B dissection, despite propensity score adjustment with open surgery [12]. Arguments questioning the efficacy of TEVAR in chronic type B aortic dissection relate to concerns regarding the established nature of the dissection flap and the presence of multiple downstream fenestrations which may prevent complete thrombosis, FL depressurization, and favorable FL remodeling [2]. Although acute and chronic type B aortic dissection were shown to behave differently after endovascular intervention with divergent outcomes, TEVAR outcomes have frequently been reported over a heterogenic population of patients comprising both acute and chronic dissections [13-15] while only few studies have reported TEVAR outcomes exclusively in the setting of chronic type B dissection and much less in the setting of a more stratified classification according to dissection duration. All of this has added to the confusion regarding the efficacy of TEVAR in these patients. The definition of aortic dissection within 14 days after onset of symptoms is generally referred as being acute in most trials and clinical practice, and anything beyond as chronic. However, there is a growing awareness of a transitional period immediately following the acute phase or a sub-acute phase (15-90 days) that is distinct from the established chronic dissection phase. [16-18] Analysis of the intimal thickness as a function of time showed stabilization in the thickening rate occurring after about 83 days and reaching a plateau after about 235 days (about 8 months) [11]. This is consistent with the view that there is a sub-acute period (~ first 3 months) corresponding to a transitional period of septal remodeling that is characterized by a period of markedly accelerated growth before entering the established chronic phase [11]. Based on this temporal remodeling behavior of the aorta in dissection, TEVAR performed in the subacute period may be expected to show a different outcome compared to the chronic phase [18]. The INSTEAD trial, which was the only prospective randomized study investigating the impact of TEVAR in a population of uncomplicated chronic type B dissection, by definition enrolled chronic patients beyond the interim period of 14 days ranging between 2 and 52 weeks with a median of 39 days for the combined OMT and TEVAR patients. However, the dissection duration represented a subset of patients that actually corresponded to sub-acute type B dissection [19,20]. Therefore, the remodeling behavior after TEVAR observed in this study does not strictly apply to chronic type B dissection patients who had thicker and less compliant intimal septum. At the same token, the relatively non expansible nature of the thickened flap in chronic dissection may cause erosive injury of the thickened intima causing so called stent graft induced new entry (SINE) tear which may lead to sudden FL pressurization and rupture risk. Although SINE was reportedly associated with both acute and chronic dissection, the onset after the primary procedure ranged between 12 ± 9 months to 24.8 ± 5.9 months, which would be well into the chronic phase

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of the dissection flap [2,21,22]. Differences in remodeling behavior according to the temporal dissection classification are methodically described in the VIRTUE Registry Investigators study comparing the outcomes by acute, subacute, and chronic aortic dissection. The true lumen showed significant expansion with time in all three subgroups after TEVAR. While the FL lumen area in the acute and subacute groups showed significant decreases at all three anatomical index study sites, in chronic dissection the FL did not show a definitively decreasing pattern with time. Furthermore, although there were no significant mid-term differences in all cause and dissection related mortalities there was a greater need for additional TEVAR in chronic dissection with more than 30% requiring further TEVAR distal to the original stent graft [23]. Therefore, a more limited remodeling response to TEVAR in the chronic type III group as opposed to the acute and sub-acute groups was suggested to be the case.

Achieving complete false lumen thrombosis with TEVAR as a primary treatment objective is based on the observation that patients with complete FL thrombosis displayed better long term prognosis than those with a patent false lumen [24]. Progressive aortic enlargement and increased risk of rupture was more likely in patients with a patent FL [25]. Although the overall complete thrombosis rate after TEVAR is generally low at approximately 39%, patients with a limited dissection had a significantly greater treatment success and higher complete thrombosis rate than those with more extensive dissections at 78% vs. 13%, P<0.001, respectively [26,27]. In addition, FL thrombosis in extensive type III dissection which is reported to occur in about 75 to 83% is usually limited to the covered segment of the false lumen, with the risk of aneurysmal degeneration distal to the distal margin of the stent graft remaining even after treatment [27]. To treat the non-covered distal communicating channels, a more liberal coverage of the thoracic segment may be contemplated, but even with this approach the stent graft cannot cover beyond the visceral vessels at the celiac axis level [26]. Therefore, retrograde flow from fenestrations beyond this aortic segment remains intact. Kölbel et al. described a technique of paving the descending thoracic aorta with an oversized stent-graft in the true lumen and tearing the dissection membrane to allow expansion of the graft into the FL thereby preventing retrograde flow from distal fenestrations pressurizing the proximal FL. Although this technique may be effective in preventing the distal retrograde flow it is nevertheless unable to fundamentally occlude the reentry sites distal to the visceral vessels [13]. Therefore, the inability to occlude intimal tears at or below the visceral vessels remains a limitation of supra-celiac TEVAR. Strategies aimed at overcoming this limitation such as fenestrated or branched stent grafts, and hybrid thoracoabdominal aortic procedures are difficult to standardize with access limited to a few highly specialized experts. Furthermore, the long term durability remains uncertain with evidence leaning towards significantly greater morbidity and mortality risks [28-30]. Therefore, various methods to promote FL thrombosis by selectively excluding the distal entry tears endovascularly are being investigated [31,32]. These approaches offer a relatively simple and significantly less invasive alternative. Kim TH et al performed false lumen procedures in 25 chronic type III b patients (>3 months) either adjunctively to TEVAR or as an isolated procedure. The mean imaging follow up duration (CT) was 5 months. Complete thrombosis was achieved in 20 patients or in 80%. The aortic dimensions were measured at the left subclavian artery (LSA), the pulmonary artery bifurcation (PA), and abdomen (celiac artery) levels. Significant positive remodeling as indicated by TL expansion and FL regression at all three levels indicated promising results. However, the follow up duration was relatively short and the before and after changes were significant but not noticeably large. The basic assumption of the FL procedure is that occluding all distal fenestrations and thereby retrograde flow into the FL may be sufficient to induce the desired remodeling effect. However, Qing KX et al. reported that although the FL pressure in a non-thrombosed FL remained unchanged and intraluminal pressure in a thrombosed FL was significantly reduced after TEVAR, the pressure in the thrombosed FL was still nearly at 80% of the pre-procedure pressure [32]. Therefore, the pressure in the thrombosed chamber remains at significant levels warranting further long term studies to assess the impact of this reduced but nonetheless high intra-thrombus pressure on delayed aneurysm formation and rupture. The possibility of inflows from significant type II endoleak contributing to this persistent pressurization cannot be ruled. In EVAR patients it is a frequent and major cause of treatment failure [33]. The available literature to date indicate that TEVAR may be highly efficacious in the setting of chronic type B aortic dissection but probably in a selective manner limited to those with favorable anatomy and flow communications that can be effectively addressed. The implications of the encouraging results with distal FL procedures suggest that it is worthwhile to focus efforts at effectively identifying anatomic and flow conditions that are conducive to successful endovascular treatment of chronic type B aortic dissection. Currently, the evidence in the existing literature suggests a significantly poorer overall response to remodeling and greater likelihood of treatment failure with time in chronic type B dissection (with endovascular treatment). Therefore, at the present stage, there is a definite beneficial role of endovascular treatment in the treatment chronic type B aortic dissection. However, until the issues raised in this review can be effectively resolved, open surgical repair remains the treatment of choice, at least in those patients in whom the raised issues remain pertinent. Until then, a more objective and truly balanced approach to treating chronic type B dissection seems warranted.

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