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Incidence, risk factors, and outcome of retrograde type A aortic dissection after TEVAR

A systematic review. English version

Retrograde type A aortic dissection after thoracic endovascular aortic repair (TEVAR) has recently been recognized as a new pathophysiological entity [1, 2]. Initial case reports in the literature were published as early as in 2002 [3, 4]. As the incidence in early TEVAR series was rare, it took several efforts in order to understand the mechanisms behind the disease [5]. Initial conceptual approaches were provided by a large consecutive series of 443 patients undergoing TEVAR reporting 11 cases with retrograde type A aortic dissection from China as well as by the European Registry of Endovascular Complications in 2009 [1, 2]. Furthermore, biomechanical studies and finite element analyses provided better insights into the underlying mechanisms [6]. Still, little is known about the incidence, risk factors, and outcome.

The aim of this systematic review was to evaluate the incidence, risk factors, and outcome of retrograde type A aortic dissection after TEVAR in the current literature.

Methods

Literature search strategy

The search terms used were ("aorta"[MeSH Terms] OR"aorta"[All Fields]) AND ("dissection"[MeSH>Terms]OR"dissection"[All Fields]) AND retrograde[All Fields] (450 hits). All abstracts were studied and reports without any relation to endovascular therapy were excluded. Forty-five hits remained. After excluding case reports, reviews, technical tips, and reports not written in English, 28 hits remained. Furthermore, reports where endovascular therapy was used as a therapeutic strategy to treat retrograde type A dissection/intramural hematoma (IMH) were excluded (26 hits remaining). Furthermore, a hand search adding the term"TEVAR" was performed. Thereby, two other studies could be identified. Studies were considered relevant if they presented a continuing series of patients from the same institution. A minimum requirement was the reporting of the incidence of retrograde type A aortic dissection, of the potential pathogenesis, and of the management. Registries were excluded. Thereby, 17 studies were considered relevant. Of these, one institution isolatedly reported a subset of their entire cohort, and this this was regarded as redundant. Finally, 16 studies remained for further analysis (**Fig. 1**, **Tab. 1**).

Outcome measures

We focused on incidence, underlying aortic pathology, pathogenesis, location of entry tear, time between TEVAR and occurrence of retrograde type A aortic dissection, as well as outcome.

Results

Incidence

The incidence of retrograde type A aortic dissection after TEVAR was given in all 16 studies and was between 1.3 and 24% [5, 7]. Incidence varied substantially with regard to the underlying aortic pathology treated as well as with regard to the application or nonapplication of supra-aortic transpositions prior to TEVAR. Grabenwoger reported a 1.3% incidence of retrograde type A aortic dissection in a consecutive series of 80 patients undergoing TEVAR between 1996 and 2003 [5]. This very patient sustaining retrograde type A aortic dissection was treated for an acute type B aortic dissection. As 20 patients in this series were treated for acute type B aortic dissection, the incidence in this subgroup is 5%. The highest incidence was observed in a series of 17 patients undergoing hybrid repair of aortic arch dissections [7].

M. Czerny and M. Rieger contributed equally to this work.

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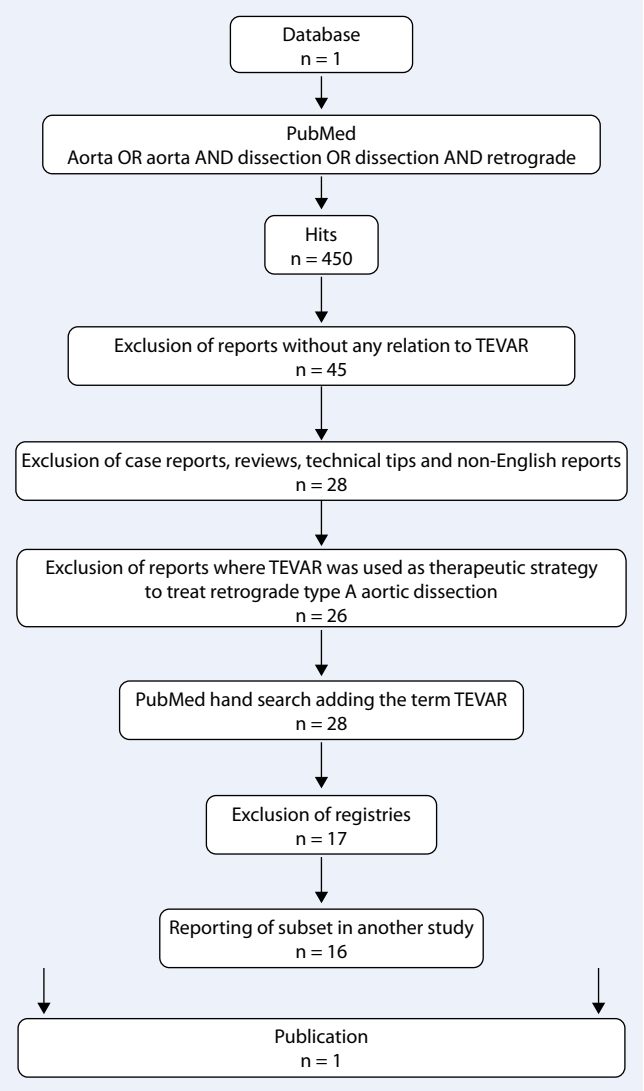

Fig. 1 ◀ Study selection

Underlying aortic pathology

The incidence was substantially higher in patients undergoing TEVAR for treatment of type B aortic dissection as well as in patients undergoing total arch rerouting in order to gain a sufficient landing zone prior to TEVAR. Of 14 patients who sustained retrograde type A aortic dissection after TEVAR in a consecutive series of 766 patients reported by the Cleveland group, seven had the underlying diagnosis of type B aortic dissection and five had the underlying diagnosis of type B intramural hematoma (i.e., 85.7% with acute aortic syndrome) [8]. The strong association between acute aortic syndromes and retrograde type A aortic dissection was consistent in all other studies, being highest in the combination of an acute aortic syndrome and supra-aortic rerouting prior to TEVAR [7].

Pathogenesis

Both proven and suspected pathophysiological mechanisms causing retrograde type A aortic dissection were consistent in all studies. The association with proximal bare stents was frequently mentioned [2, 7, 9, 10, 11]. Furthermore, three authors mentioned fragility of the aortic wall, both proximal thoracic aortic segments not serving as landing zones as well as segments serving as a proximal landing zone of the stent graft [8, 9, 12]. An enlarged ascending aorta of ≥40 mm was identified as another surrogate of increased incidence of retrograde type A aortic dissection [10, 11]. Manipulation by wire was mentioned as a cause in three studies [1, 13, 14]. Oversizing was identified as a causative factor in one study [14]. Balloon dilatation in order to enhance conformability of the stent graft to the aortic wall was mentioned in two studies [9, 15]. Clamp injury to the ascending aorta during accomplishment of the proximal anastomosis for total arch rerouting was described in two series [10, 13]. Finally, the ascending aorta used as a landing zone per se was mentioned in six studies [7, 8, 10, 11, 12, 16]. Two studies noted that the incidence of patients with bovine aortic arches was remarkable in their series at 43 and 41%, respectively [12, 14].

Location of entry tear in retrograde type A aortic dissection

The new entry tear was identified at the tips of the proximal bare springs in three studies [1, 5, 10]. Another two studies stratified entry tears according to concavity and convexity, identifying 75% of entry tears as convex and 25% as concave in the one study, and 34 and 66%, respectively, in the second study [8, 9]. One study identified the site of the proximal clamp for the anastomosis in total arch rerouting as the location of the primary entry tear [13]. Further locations described were the mid-ascending aorta in two studies, potentially representing an unrelated secondary aortic event [15, 17].

Time between TEVAR and the occurrence of retrograde type A

The time interval between TEVAR and the occurrence of retrograde type A aortic dissection varied between immediate occurrence during the procedure and up to 7 years after treatment [1, 8, 18, 19].

Outcome

Outcome varied according to the time of occurrence, being best in patients who were treated surgically where the diagnosis was made in hemodynamically stable conditions [8, 10]. These two studies reported 0% mortality. The worst outcome was reported in patients where retrograde type A aortic dissection occurred during or immediately after TEVAR (57 and

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Abstract

Background. Retrograde type A aortic dissection after thoracic endovascular aortic repair (TEVAR) has recently been recognized as a new pathophysiological entity. Little is known about its incidence, risk factors, and outcome.

Methods. Electronic searches were performed in Medline until 8 November 2013. Relevant studies were identified. We looked for consecutive series reporting on incidence, risk factors, and outcome. Registries were excluded. Data were extracted and analyzed according to predefined outcome measures.

Results. Sixteen studies were identified for inclusion for qualitative and quantitative analyses. The incidence of retrograde type A aortic dissection after TEVAR was given in all 16 studies and was between 1.3 and 24%. Incidence varied substantially with regard to the underlying aortic pathology treated as well as with regard to the application or non-application of supra-aortic transpositions prior to TEVAR. The strong association between acute aortic syndromes and retrograde type A aortic dissection was consistent in all other studies, being highest in the combination of an acute aortic syndrome and supra-aortic rerouting prior to TEVAR (24%). Both proven and suspected pathophysiological mechanisms causing retrograde type A aortic dissection were consistent in all studies. The time interval between TEVAR and the occurrence of retrograde type A aortic dissection varied between immediate occurrence during the procedure and up to 7 years after treatment. Outcome varied according to the time of occurrence, being best in patients who were treated surgically where the diagnosis was made in hemodynamically stable conditions.

Conclusion. Retrograde type A aortic dissection after TEVAR is not rare in patients with the underlying pathology of type B aortic dissection, type B intramural hematoma as well as after total arch rerouting aimed for zone 0 landing zone deployment. Retrograde type A aortic dissection after TEVAR may occur early or late. Further studies are needed to clarify the pathophysiological mechanisms and to prevent this complication in the future.

Keywords

Aortic dissection, type A · Thoracic endovascular aortic repair · Incidence · Risk factors · Outcome

Retrograde Typ-A-Aortendissektion nach TEVAR: Inzidenz, Risikofaktoren und Outcome. Ein systematischer Review

Zusammenfassung

Hintergrund. Vor kurzem wurde die retrograde Typ-A-Aortendissektion nach TEVAR als neue pathophysiologische Entität anerkannt. Zu Inzidenz, Risikofaktoren und Outcome ist noch wenig bekannt.

Methoden. Bis zum 08.11.2013 wurde in der Datenbank Medline recherchiert, für die Fragestellung relevante Studien wurden identifiziert. Gesucht wurde nach Fallserien mit Dokumentation von Inzidenz, Risikofaktoren und Outcome. Nach vordefinierten Outcome-Kriterien wurden Daten extrahiert und analysiert.

Ergebnisse. Für die qualitativen und quantitativen Analysen wurden 16 Studien herangezogen. Die Inzidenz einer retrograden Typ-A-Aortendissektion nach TEVAR war in sämtlichen Studien angegeben, sie lag zwischen 1,3 und 24%. Die Inzidenz war je nach zugrundeliegender aortaler Pathologie und je nach (Nicht-)Anwendung supraaortaler Transpositionen vor der Intervention sehr unterschiedlich. Die deutliche Assoziation zwischen akuten Aortensyndromen und retrograder Typ-A-Aortendissektion war konsistent in allen anderen Studien zu finden, am höchsten war sie bei der Kombination akutes Aortensyndrom und supraaortalem „Rerouting" vor TEVAR (24%). Nachgewiesene und angenommene pathophysiologische Mechanismen, die eine retrograde Typ-A-Aortendissektion verursachen, waren in sämtlichen Studien konsistent. Zwischen TEVAR und retrograder Typ-A-Aortendissektion lagen sehr verschiedene Zeitintervalle: zwischen sofortigem Eintreten schon während der Behandlung bis zu 7 Jahren postinterventionell. Das Outcome war je nach Zeitpunkt des Auftretens unterschiedlich: Am günstigsten war es bei chirurgisch behandelten Patienten wo die Diagnosestellung unter hämodynamisch stabilen Bedingungen erfolgt ist.

Fazit. Bei Patienten mit den zugrundeliegenden Pathologien Typ-B-Aortendissektion und/bzw. intramurales Typ-B-Hämatom, ferner nach komplettem Aortenbogenumbau, kommt es nicht selten nach TEVAR zu einer retrograden Typ-A-Aortendissektion. Sie kann früh oder spät TEVAR auftreten. Weitere Untersuchungen sind erforderlich, um die pathophysiologischen Mechanismen zu klären und dieser Komplikation künftig vorzubeugen.

Schlüsselwörter

Aortendissektion Typ A · Endovaskuläre Therapie thorakaler Aortenläsionen · Inzidenz · Risikofaktoren · Outcome

60%) [14, 20]. However, also in these conditions, favorable reports were available stating a 0% mortality in three patients where the diagnosis was made immediately during the TEVAR procedure in the angiosuite [5].

Discussion

Retrograde type A aortic dissection after TEVAR is not rare in patients with the underlying pathology of type B aortic dissection, type B intramural hematoma, as well as after total arch rerouting aimed for zone 0 landing zone deployment. Retrograde type A aortic dissection after TEVAR may occur early or late. Further studies are needed to clarify the pathophysiological mechanisms and to prevent this complication in the future.

The incidence of retrograde type A aortic dissections ranges widely in the current literature being between 1.3 and 24% in studies included in this systematic review [5, 7]. When going into de-

Tab. 1 Studies included in the present analysis

Reference	Author[a]	Journal[b]	Patients (*n*)	Incidence (%)[c]	Type B as underlying pathology (%)[d]	Time to event (range in days)[e]	Outcome (% dead)[f]
[2]	*Dong*	*Circulation 2009*	443	2.5	100	0–1,080	18
[5]	*Grabenwoeger*	*Eur J Cardiothorac Surg 2004*	80	1.3	5	na	0
[7]	*Cochennec*	*J Vasc Surg 2013*	17	24	100	na	50
[8]	*Idrees*	*J Thorac Cardiovasc Surg 2013*	766	1.8	85.7	0–2,766	0
[9]	*Gorlitzer*	*Eur J Cardiothorac Surg 2012*	29	13.8	100	0–14	0
[10]	*Luehr*	*Eur J Cardiothorac Surg 2013*	9	22	50	10–12	0
[11]	*Andersen*	*J Vasc Surg 2013*	87	3.4	na	0–3	66
[12]	*Williams*	*J Vasc Surg 2012*	309	1.9	66.7	0–6	33
[13]	*Geissbüsch*	*J Vasc Surg 2011*	47	6.3	100	0–na	33
[14]	*Kpodonu*	*Eur J Cardiothorac Surg 2008*	287	2.4	85.7	1–900	57
[15]	*Steingruber*	*J Endovasc Ther 2008*	35	11.4	100	3–1,770	na
[16]	*De Rango*	*J Vasc Surg 2013*	104	3.8	na	0–10	50
[17]	*Neuhauser*	*Eur J Cardiothorac Surg 2008*	28	17.9	100	3–1,170	20
[18]	*Ehrlich*	*Ann Thorac Surg 2010*	32	3.1	100	na	na
[19]	*Andacheh*	*J Vasc Surg 2012*	73	5.5	na	na	40
[22]	*Bavaria*	*J Thorac Cardiovasc Surg 2013*	47	2.1	na	155	na

na not available[a]First author's last name[b]Journal abbreviation and year of publication[c]Incidence of retrograde type A aortic dissection in percent on the entire cohort reported[d]Percentage of patients sustained retrograde type A aortic dissection with the underlying pathology of type B aortic dissection[e]Time from TEVAR to the event of retrograde type A aortic dissection reported as range in days[f]Percentage of nonsurvivors with the diagnosis of retrograde type A aortic dissection

tail it becomes evident that certain subgroups are at increased risk of developing this complication such as patients undergoing TEVAR for the treatment of type B aortic dissection, in particular patients with acute type B aortic dissection as well as patients with type B intramural hematoma [8]. Several reasons may account for these findings. Primarily, the aortic wall in patients with (acute) type B aortic dissection is more frail than in other thoracic aortic pathologies and in many patients there will be no disease-free landing zone as length is limited by the offspring of supra-aortic vessels. As such, a sealing zone is present but no classic landing zone, meaning morphologically and functionally healthy tissue. Secondarily, the compliance mismatch between the highly elastic aortic wall and the rigid stent graft may well account for substantial shear stress and may therefore lead to intimal disruption [6].

Another subgroup of patients at elevated risk are patients after total arch rerouting aimed at gaining a sufficient proximal landing zone for TEVAR. These patients where zone 0 landing is intended may present several reasons why retrograde type A aortic dissection occurs. Tangential clamp injury, compliance mismatch, and alteration of hemodynamics in the aortic arch by total arch rerouting may account for initiation of the pathophysiological process [6, 10, 20]. If a combination of these risk factors converges, incidence increases excessively as shown in a French series describing a 24% incidence in patients undergoing arch rerouting with an underlying diagnosis of aortic dissection [7].

It seems that two main reasons account for the stroke rate after total arch rerouting: the underlying pathology and incomplete rerouting. Regarding the underlying pathology, the ascending aorta being affected by the degenerative process might be decisive, in particular patients with penetrating atherosclerotic ulcers as this disease is the phenotype of an aggressive underlying obliterative arteriopathy and embolization is likely. Tradeoffs regarding the left subclavian artery may account for strokes since exposure and transposition are sometimes challenging. We feel it is important to underline that left upper extremity perfusion is the weakest argument for additional subclavian artery transposition as this is usually well collateralized. However, posterior cerebellar circulation and spinal cord perfusion strongly depend on subclavian inflow. Secondary ischemic events after initial asymptomatic courses when omitting subclavian revascularization are also reported [21].

Proximal bare stents have been traditionally associated with retrograde type A aortic dissection [2, 7, 9, 10, 11]. The first description of this context was given by the European Registry for Endovascular Complications in 2009 [1]. However, it should be taken into account that more than 80% of patients included in this registry received a stent graft with proximal bare stents. This was because at that time only one commercial provider of stent grafts was on the market. In the authors' experience and in the experience of many others, retrograde type A aortic dissection may occur with and without proximal bare stents. The overall low incidence of retrograde type A aortic dissection in the EuREC registry might be due to the low vigilance for the problem (at that time), in other words, patients dying suddenly might have been stratified as cardiovascular instead of looking for retrograde type A aortic dissection. Nevertheless, a substantial difference between acute aortic syndromes and chronic thoracic aortic pathology was already noted [1].

An enlarged ascending aorta was mentioned as a potential trigger for retrograde type A aortic dissection in several reports [10, 11]. As a consequence, many authors have changed their strategy and now perform prophylactic ascending aortic replacement in these patients [1, 11, 22]. This eliminates the risk of sustaining an event. However, the extent of repair is clearly widened. It would be interesting to gain information on the cuspidity of the aortic valve in patients sustaining retrograde type A aortic dissection, since an association between the bicuspid aortic valves and the occurrence of type A aortic dissection is known [23]. There seems to be a subgroup of patients with bicuspid aortic valves who have at least a minor connective tissue disease component, which itself is a risk factor for sustaining acute aortic syndromes [24].

Manipulation by wire and oversizing were also mentioned. It is clear that any injury to the intima may lead to a functional primary entry tear with ante- and retrograde propagation and that manipulation should be done with caution [1, 13, 14]. Oversizing was also named as a causative factor. Oversizing per se increases radial force to the aortic wall and may thereby amplify compliance mismatch and shear stress leading to intimal disruption with its known consequences [14]. By contrast, EuREC recorded a 6% median oversizing, which seems low [1]. As a consequence, the underlying aortic pathology might be more decisive than the degree of oversizing.

Balloon dilatation was mentioned [9, 15]. The use of this adjunctive method to increase conformability to the aortic wall is less frequently used since devices with better alignment and more even radial force distribution have become widely available.

Two studies established an association between a bovine trunk and retrograde type A aortic dissection [12, 14]. This is interesting as little is known about the mechanisms. Also here, it would be of great interest to know the cuspidity of the aortic valve as it might give another hint on the occurrence of retrograde type A aortic dissection [23, 24].

A new entry tear was identified at the tip of the proximal bare stent in three studies [1, 5, 10]. This seems logical as chronic radial force to the intimal surface will have led to disruption. Another two studies stratified the entry tear according to convexity and concavity. This was interesting as differences regarding the propagation of naturally occurring dissection according to a primary entry tear at the concavity or the convexity are well known [25, 26]. However, in retrograde type A aortic dissection it may well be related to the point of maximal radial force of the proximal end of the stent graft than to other reasons. Interestingly, two studies described an entry tear remote from the proximal end of the stent graft, which might be interpreted as a new and isolated type A aortic dissection [15, 17]. This is known in patients after type B aortic dissection. Because no further detailed description in these two reports was available, this theory remains speculative.

The time interval between TEVAR and the occurrence of retrograde type A aortic dissection varied considerably between immediate occurrence during the procedure and up to 7 years after treatment [1, 8, 18, 19]. This is somewhat worrisome as remaining risk persists years after TEVAR. This circumstance mirrors our very limited knowledge regarding this disease.

Outcome varied according to the time of occurrence. Favorable as well as dismal results have been reported both after immediate and late occurrence [5, 8, 10, 14, 20]. The presence or absence of hemodynamic instability, speed of diagnosis and treatment, as well as clinical conditions may contribute to outcome. Little information was given with regard to clinical symptoms. Interestingly, in a substantial number of patients—consistent with the EuREC findings—the diagnosis was made by chance and a specific event of chest pain could not be determined [1, 10]. The reasons remain speculative. The best explanation might be provided in patients after total arch rerouting and TEVAR where dissective pain might be interpreted as unspecific poststernotomy discomfort.

Limitations

This systematic review has all the limitations inherent to the studies included, which were single-center series. Moreover, late retrograde type A aortic dissections might have been missed, since late deaths may have been classified as sudden cardiac deaths but may have been due to retrograde type A aortic dissection.

Conclusion

In summary, retrograde type A aortic dissection after TEVAR is not rare in patients with the underlying pathology of type B aortic dissection, with type B intramural hematoma, as well as after total arch rerouting aimed for zone 0 landing zone deployment. Retrograde type A aortic dissection after TEVAR may occur early or late. Further studies are needed to clarify the pathophysiological mechanisms and to prevent this complication in the future.

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Compliance with ethical guidelines

Conflict of interest. M. Czerny, M. Rieger, and J. Schmidli state that there are no conflicts of interest.

The accompanying manuscript does not include studies on humans or animals.

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