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SHORT REPORT

What We Know From Reports on Type III Endoleak in the Literature

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Objective: To analyse case reports published on the latest generations of endograft (EG) and understand the mechanisms of type III endoleak (EL) development.
Methods: A literature review was undertaken of English language case reports and series that concerned modular junction or component disconnection (type IIIa EL) and fabric perforations (type IIIb EL) after endovascular aneurysm repair.
Results: Of the 2 785 studies, 56 full texts were chosen to review 73 cases. Type III EL was diagnosed with computed tomography angiography in 67.1% and digital subtraction angiography in 12.3%; the rest were identified during surgery. Of the 73 EG, 65 (89.0%) were made of polyethylene terephthalate and seven (9.6%) were polytetrafluoroethylene. The type of material was not mentioned in one (1.4%) case report. There were 25 (34.2%) type IIIa and 48 (65.8%) type IIIb EL. The most frequent were trunk–trunk in nine (12.3%) and trunk–limb overlap separations in 14 (19.2%). Type IIIb EL in the trunk area was identified in 27 (37.0%) cases, while 21 (28.8%) defects were found in the limbs. Stent fractures were recognised as an underlying mechanism of type IIIb EL development in one report. A combination of fabric lesions in the trunk and limb area was found in one case. Seven type IIIb EL were related to suture disruption or suture–fabric abrasions. Four cases were related to stent–fabric abrasions, and two developed as a result of fabric fatigue owing to kinking. Information on the mechanisms of degradation was only occasionally and scarcely presented. Given the small number of reports and lack of detailed analysis, no definitive conclusions could be drawn.
Conclusion: The available information is scarce and does not allow any definitive conclusions to be drawn on the mechanisms that lead to the development of type III EL. Further explant analyses would be beneficial.
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INTRODUCTION

Endovascular aneurysm repair (EVAR) has become routine in the treatment of abdominal aortic aneurysm (AAA). Technological improvements that include better fixation, flexibility, and durability of endografts (EG) have reduced the risk of migrations, stent fractures, and component separation. Despite the progress, endoleak (EL) can still affect the treatment outcome.[1] Approximately 30% of patients undergoing EVAR develop various types of EL;[1,2] this increases the risk of consequent sac expansion and aneurysm rupture.

Most publications focus on type I and II EL. Type I, being a high flow and high pressure EL, is a frequent cause of re-intervention for rupture prevention. Conversely, patients with type II EL may only require surveillance.[3] As with type I, type III EL may expose patients to the same level of risk, elevating pressure inside the aneurysm. The precise mechanisms of this complication remain undefined. Regularly published case reports might present accurate and valuable information. This review aimed to analyse them in the hope of understanding the reasons for failure in order to take further steps towards discussions on precise examinations or making changes in treatment strategy.

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REPORT

Materials and methods

An electronic search of Medline and Cochrane Library databases was conducted to obtain English language reports about type III EL to reveal possible mechanisms of development after EVAR. The search was aimed at case reports or case series published between January 2003 and December 2021. The following medical subject headings, informal terms, and their combinations were applied in the search engine: Endoleak, Type III/3 (Endoleak), Endovascular Aneurysm Repair, Endovascular Aortic Repair, Stent Grafting, Aortic Aneurysm, and Ruptured Aneurysm. Two investigators (JG and NC) independently verified the obtained

publications for appropriateness based on the titles and abstract information. Experimental, pharmacological studies, cases of thoracic aortic EG, EG lesions following trauma or iatrogenic impact, and cases without precise description of lesion type and their location were excluded from further analysis. Disagreements were resolved by means of a re-investigation of the data, discussion, and a consensus. Consequently, full text versions of the selected reports were obtained. A standardised form was used for data collection. The extracted data comprised study characteristics (year of publication, number of cases), EG characteristics (brand and model), EL type and or subtype, indications for diagnostic imaging, treatment modality, reported EG lesions, and macroscopic or microscopic analysis when available. No ethical approval or patient consent were required for the study.

Results

The article selection process is described in Fig. 1. A total of 2 785 studies were identified, and 56 articles were chosen.

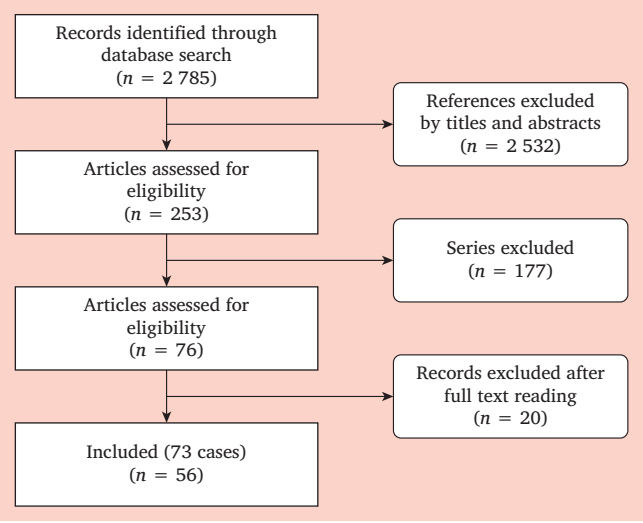

Figure 1. Flow diagram of the selection process for identifying suitable case reports on type III endoleak. The exclusion criteria are provided in the text.

This number comprised case reports ($n = 45$) or small series ($n = 11$) published between 2003 and 2021 on the topic of type III EL, including a total of 73 patients. All patients underwent computed tomography angiography (CTA) but 49 (67.1%) of them were diagnosed with EL. Nine patients (12.3%) underwent endovascular repair with EL identified on the aortography. Thirteen (17.8%) EL were diagnosed during open surgery. The examination modality was not mentioned for one patient (1.4%), and one case (1.4%) was defined as a diagnosis of exclusion, with no visible findings on the initial CTA. A summary of reported indications for imaging diagnostics or treatment, with the EL subtypes, is presented in Table 1. The EL of both subtypes in routinely followed and symptomatic AAA patients were generally recognised with imaging diagnostics. Conversely, preoperative imaging confirmation of EL was less frequent in patients with sac enlargement and AAA rupture. Type IIIb EL was predominantly reported in those cases. Endovascular management of patients with EL was common in elective cases, followed by urgent patients with symptomatic AAA. At least half of the sac enlargements and AAA ruptures were treated with open surgery. Hybrid procedures were infrequent.

Endograft characteristics. Of the 73 cases, 65 (89.0%) represented polyester EG of the polyethylene terephthalate (PET) subgroup. There were seven expanded polytetrafluoroethylene EG (9.6%). The type of material was not mentioned in one case (1.4%). There were 25 type IIIa (34.2%) and 48 type IIIb EL (65.8%).

Causes of type IIIa endoleak. A summary of lesion locations by fabric type is presented in Fig. 2. The most frequently reported were contralateral limb component separations from the main body in 14 (19.2%) or separations of a trunk from a trunk or an extension cuff in nine (12.3%).

Causes of type IIIb endoleak. Type IIIb EL was caused by fabric defects, most of which could be identified in the trunk (Fig. 2). Eighteen reports presented specified macroscopic investigation data. However, few provided detailed information on failure mechanisms. Thus, seven type IIIb ELs

Table 1. Indications, characteristics of type III endoleak, and treatment modalities.

Indications	Total number of EL cases	EL cases confirmed by imaging	Type IIIa EL	Type IIIb EL	Endovascular repair	Open repair	Hybrid repair
Routine examination*	26 (100.0)	26 (100.0)	11 (42.3)	15 (57.7)	22 (84.6)	2 (7.7)	1 (3.8)
Symptomatic abdominal aortic aneurysm†	15 (100.0)	14 (93.3)	6 (40.0)	9 (60.0)	9 (60.0)	4 (26.7)	1 (6.7)
Sac enlargement	12 (100.0)	1 (8.3)	1 (8.3)	11 (91.7)	4 (33.3)	7 (58.3)	1 (8.3)
Rupture	16 (100.0)	6 (37.5)	4 (25.0)	12 (75.0)	8 (50.0)	8 (50.0)	0 (0.0)
Other reasons‡	4 (100.0)	2 (50.0)	3 (75.0)	1 (25.0)	4 (100.0)	0 (0.0)	0 (0.0)

Data presented as *n* (%) for each indication group. EL = endoleak; Hybrid repair = a unilateral endograft and a femorofemoral bypass.
* Includes one patient with a symptomatic abdominal aortic aneurysm at a routine follow up visit (type IIIa + IIIb).
† One patient (type IIIa) died before surgery.
‡ Other reasons: non-abdominal aortic aneurysm related indication or non-reported indication.

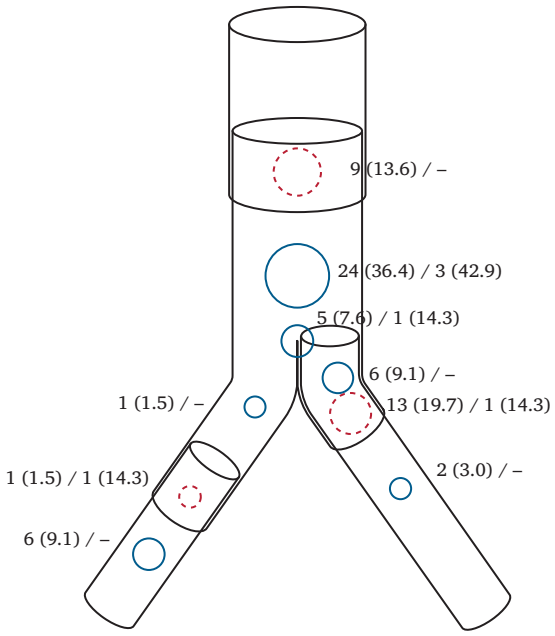

Figure 2. A schematic illustration of the distribution of type IIIa (dashed) and type IIIb (solid) endoleak. Data are presented as *n* (%) for the polyester and polytetrafluoroethylene endograft groups. One case with unknown type of material is included in the polyester trunk group (24 cases).

(38.9%) were related to suture disruption or suture—fabric abrasions, leading to hole formation. Five cases (27.8%) were related to stent—fabric abrasions (one stent fracture), and two developed as a result of fabric fatigue owing to kinking (11.1%).

DISCUSSION

This study focused on available information concerning the mechanisms of type III EL development. Based on the obtained case reports, the actual mechanisms were scarcely reported and insufficiently studied. Few publications provided limited data on the mechanisms of several types of fabric disruption that possibly led to perforation.

EG evolution has brought better device stability and reduced the risk of type I and, according to some series, type III EL.[4,5] Nonetheless, the data are insufficient and there is a lack of reliable information on the distribution and comparison of infrarenal PET EG and expanded polytetrafluoroethylene grafts in the setting of type III EL.

This review found that a third of the cases demonstrated type IIIa EL. One of the potential reasons is an inherent weakness in multicomponent grafts that could entail a certain risk of type IIIa EL. Type IIIa EL could also develop because of material degradation, including stent properties. These issues require additional attention. The studied case reports lacked information on the adherence to the instructions for use. It is well known that complications are more common in cases in which the instructions for use are not followed. The typical reasons for failure were insufficient overlap, inadequate sizing (could lead to a type Ia EL managed with a proximal extension cuff, which can separate), kinking in a tortuous or calcified artery, and some other technical issues that could probably be prevented. The more common type IIIb EL could be related to the construction of the endograft, fabric, stent wires, and stitches. The inherent weakness of the construction, which runs the risk of breakdown in a number or cases, or exceptional stress on the material could perhaps be explained by suboptimal placement. On the other hand, the identified mechanisms suggest friction of the fabric against stitches or metal stents as a primary cause of perforation. The tortuous anatomy of the aorta and presence of large folds could possibly increase the extent of the rubbing effect, particularly in the PET EG that were predominant in this review. A previous publication[6] summarised possible fabric damage mechanisms based on an EG explant analysis. According to that study, ageing of the implanted device could be the factor that increased the risk of fabric perforation. Although this suggestion is logical, the current review could not support or reject this statement, since the data lacked comprehensive clinical information. In the other review conducted by this team, two main mechanisms of fabric damage were identified: compression and abrasion.[7] The first type was related to possible compression damage associated with excessive material packing inside the delivery system. The second type included the cyclical stress that the material is exposed to in vivo. This type of deterioration under certain conditions may potentially involve all kinds of woven and non-woven materials, such as fabric, stitches, and fixation knots. They would obviously undergo degradation before metal stents, which tend to be more durable.[7]

The diagnostic accuracy of type III EL detection is low. Computed tomography angiography revealed one fifth of all type IIIb EL,[8] suggesting that better image analysis could be beneficial.[9] However, a negative CTA with significant sac growth should be considered for treatment.[10] There was also publication bias, and the cases were under reported. The previous study demonstrated both the lack of data and interest in the degradation processes and device failures.[7]

A key point to ensure that the provided patient care is adequate and safe is the durability of the implanted EG. This is the reason why similar events should be reported in case reports and also in physician driven registries. The lack of detailed analysis prevented definitive conclusions being made on the possible mechanisms of degradation or on further strategies. Comprehensive explant analyses are needed to further elucidate the mechanisms involved in fabric and stent degradation processes.

CONCLUSION

The cornerstone of understanding the mechanisms of degradation that lead to the development of type III EL is a detailed analysis of the devices. Nonetheless, the relevant data have not been provided by surgeons. The available information is scarce and does not enable any definitive conclusions to be drawn on the mechanisms that lead to

the development of type III EL. Explant analyses in an overall program of material surveillance should be encouraged.

CONFLICTS OF INTEREST

None.

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