Title: Preoperative Endoleak Type in Patients of Aneurysmal Sac Expansion After Endovascular Aneurysm Repair and Intraoperative Blood Loss in Aneurysmorrhaphy: Analysis of a Case Series of 18 Patients

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Research Article

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Abstract

Purpose

EVAR (endovascular aneurysm repair) has become a possible treatment option of abdominal aortic aneurysms, and endoleak is the most common complication after EVAR. The purpose of this study was to determine the difference of intraoperative blood loss in aneurysmorraphy for aneurysmal sac expansion after EVAR according to type of endoleak.

Methods

Perioperative data of 18 patients underwent aneurysmorrhaphy for aneurysmal sac expansion after EVAR from July 2010 to November 2017 at the Jikei University Hospital are collected. Patients were divided in two group, endoleak type II or type V (endotension) according to preoperative imaging. Patient demographic and quantitative data, including intraoperative blood loss and intraoperative red blood cell transfusion volume, were collected from electronic medical and anesthetic records. Intraoperative blood loss in aneurysmorrhaphy were compared between the two groups.

Results

In 18 patients, 13 patients were diagnosed type II endoleak, and 5 patients were diagnosed endotension. There were statistically differences in intraoperative blood loss and red cell transfusion volume between type II endoleak and endotension groups ($P = 0.0016$ and $P = 0.018$, respectively).

Conclusion

These results suggest that the type of endoleak after EVAR may be associated with intraoperative blood loss in aneurysmorrhaphy. Pre-operative estimation of intraoperative blood loss depending on endoleak type may be helpful for the decision making of anesthetic management in aneurysmorrhaphy.

Introduction

Aortic aneurysm has been treated mainly by open surgical repair. Since the aortic stent graft was approved in Japan in 2006, endovascular aneurysm repair (EVAR) has become a possible treatment option of abdominal aortic aneurysms, especially in elderly patients and patients with high risk complications, because of its low invasiveness and rapid postoperative recovery. Previous studies have shown that the incidence of laparotomy- related complications after EVAR is lower in comparison with open surgery, however the long-term prognosis of EVAR remains unknown [1–3]. Device-related complications of EVAR include endoleaks, stent graft migration, stent graft fracture, aortic neck expansion, and development of other aortic aneurysms. Secondary intervention is required in generally

15–20% of patients [2–4]. Endoleak is the most common complication requiring secondary intervention after EVAR, and type II endoleak is the most common type of endoleak [5, 6].

Type II endoleak that caused by a backflow through collateral vessels is most problematic in the chronic phase. Current guidelines recommend treatment of type II endoleaks associated with aneurysm enlargement to prevent rupture and aorto-enteric fistula, and transarterial coil or embolization is the choice of intervention [7, 8]. Conventional open surgical treatment, including open graft replacement and aortobiliac bypass grafting is a secondary choice in type II endoleak or type V (also called endotension) cases if previously transarterial coil or embolization failed [9]. Recently, aneurysmorrhaphy is considered as a less invasive treatment for patients with enlargement of aneurysm. This procedure is a modified technique that can avoid aortic cross clamp, graft explantation, and systemic heparinization [10, 11]. In our institution, aneurysmorrhaphy is standard surgical treatment in type II or endotension cases of aneurysm enlargement after EVAR. It is recognized among vascular surgeons that there is a large difference in the amount of intraoperative blood loss depending on the type of endoleak in aneurysmorrhaphy. However there has been no discussion in the literature. The purpose of the present study was to evaluate the intraoperative blood loss according to the type of endoleak in patients treated with aneurysmorrhaphy. Therefore, we hypothesized that the amount of intraoperative blood loss in endotension cases is less than in endoleak type II cases, and conducted a retrospective study.

Methods

The protocol of this retrospective study was reviewed and approved by the Institutional Ethics Committee of the Jikei University Hospital. There were 921 cases who underwent EVAR for AAA at the Jikei University Hospital from July 2010 to November 2017. Patients younger than 20 years old were excluded from this retrospective study. Totally 18 cases treated by aneurysmorrhaphy were enrolled in this study. Patient data and comorbid conditions (age, sex, hypertension, diabetes mellitus, chronic kidney disease, coronary artery disease, anticoagulation therapy, aneurysm diameter, EVAR to aneurysmorrhaphy duration, preoperative hemoglobin, preoperative hematocrit) were recorded retrospectively. Endoleak type was defined as follows; type I : blood flow entering through the end of the stent graft, type II : caused by a backflow through collateral vessels include lumbar artery or inferior mesenteric artery, type III : blood flow entering the aneurysm through the stent graft connection, type IV : blood flow through the stent graft membrane into the aneurysm, type V (endotension): increment in aneurysm diameter without identifiable blood flow. At our hospital, post- EVAR CT (computed tomography) or CTA (computed tomography angiography) or angiography are performed at every 1 to 12 months. First, coil embolization or additional stent placement were performed when the aneurysm was enlarged, and if there was no improvement, an aneurysmorrhaphy was performed. Pre- and post-operative endoleak type was diagnosed by the specific attending vascular surgeon of The Jikei University Hospital with angiography, enhanced CT, and operation records. The method of image evaluation in the follow-up period was left to the vascular surgeons. All of the operation of aneurysmorrhaphy were performed in general anesthesia and the trigger of intraoperative blood transfusion depended on each anesthesiologist. Intraoperative outcomes were collected from electronic medical records. Differences in patient variables (sex, hypertension, diabetes mellitus, chronic kidney disease, coronary artery disease, anticoagulation therapy, aneurysm diameter, duration from EVAR to aneurysmorrhaphy, preoperative hemoglobin, preoperative hematocrit) between the type II endoleak group and endotension group were analyzed by unpaired t-test and Fisher exact test. The others were analyzed using the Mann-Whitney U test. Statistical significance was defined as $P < 0.05$. Statistical analyses were performed using GraphPad Prism 5.0 (GraphPad Software Inc., La Jolla, CA, USA).

Results

A total of 18 patients underwent aneurysmorrhaphy for aneurysmal sac expansion after EVAR during this study period. Clinical characteristics of patients and pre- and post-operative diagnosis of endoleak type were described in Table 1. In only one case, type III endoleak was revealed intraoperatively and all of others had type II endoleak or endotension. In 18 patients, 13 patients were diagnosed Type II endoleak, and 5 patients were diagnosed endotension postoperatively. Patient demographic characteristics and medical comorbidities were described in Table 2. Complications included hypertension in 16/18 patients (88.9%), diabetes in 2/18 patients (11.1%), chronic kidney disease in 11/18 patients (61.1%), history of CAD (coronary artery disease) in 5/18 patients (27.7%), and anticoagulation in 10/18 patients (55.6%). Preoperative anemia (male < 13.0 g/dL, female < 12.0 g/dL, elderly < 11.0 g/dL) was observed in 5 cases of type II endoleak and one case of endotension. 8 cases of type II endoleak and 2 cases of endotension were treated with coil embolization and one case of type II endoleak and one case of endotension were treated with additional stent graft before aneurysmorrhaphy. Plain and contrast-enhanced CT images in type II endoleak or endotension after EVAR were shown (Fig. 1.2). In preoperative imaging, contrast agents in aneurysm that indicates inflow vessels are detected in all endoleak cases but not in endotension cases. Operation time, Postoperative diagnosis, intraoperative blood loss, and RBC transfusion are described in Table 3. Intraoperative blood loss and RBC transfusion were compared retrospectively between 2 groups (Table 4). The median intraoperative blood loss was 2280 mL in type II endoleak group and 80 mL in endotension group (Table 4). There were statistically significant differences in intraoperative blood loss and red blood cell transfusion volume between two groups ($P = 0.0016$ and $P = 0.018$, respectively) (Table 4 and Fig. 3).

Discussion

The current study showed that intraoperative blood loss and RBC transfusion in aneurysmorrhaphy were significantly less in endotension cases than type II endokleak cases.

In the past, open graft replacement was the main treatment for aortic aneurysm. However, since the devices for EVAR was approved in Japan in 2006, EVAR has been generally accepted as the optional treatment for repairing abdominal aortic aneurysms, especially in elderly patients because of its low invasiveness and rapid postoperative recovery. Endoleak is a major complication of EVAR, and management of endoleaks has been a major issue for more than 20 years. Type II endoleak has leakage through retrograde collateral blood flow such as lumbar, inferior mesenteric or internal iliac arteries. Type

II endoleaks are generally benign and have little adverse effect on the outcome of EVAR. However, Van Marrewijk [12] found that type II endoleak were significantly associated with dilatation of the aneurysm sac over time and it was widely recognized that such patients should be treated with endovascular therapy or conversion to open repair.

The incidence of endotension after EVAR is 1.5-5% [13, 14]. Endotension causes enlargement of the vessel and aneurysm diameter in spite of no apparent endoleak. The mechanism of endotension is not clear. It has been hypothesized that if the leak is small, the small hole in the stent graft may be blocked with a thrombus, resulting in an undetectable leak on imaging studies [15]. Some investigators have suggested that endotension results from direct pressure transfer from the adjacent aortic lumen to the aneurysm [16]. Another hypothesis was that leakage fluid accumulates progressively in the aneurysm sac due to increased permeability of covering material of the stent graft. This increased permeability has been reported primarily with polytetrafluoroethylene stent grafts and inhibits thrombus formation in the aneurysm sac [17]. In endotension, fibrinolysis in the aneurysm sac was also observed [18]. This hyperfibrinolysis may be a risk for bleeding.

The largest published series was from European Collaborators on Stent/graft Techniques for aortic Aneurysm Repair (EUROSTAR) registry (3595 patients), with a 9 % of type II endoleak cases diagnosed during follow-up [12]. It was also reported that 2.4 ~ 6 % of patients were required secondary treatment due to endotension after EVAR [5, 19]. CT (computed tomography), MRI (magnetic resonance imaging), and US (ultrasound) are commonly used to diagnose endoleak types, although guidelines recommend CTA (computed tomography angiography) after 6 months [20]. In this study, preoperative endoleak type was diagnosed by CT, CTA, and angiography performed every 1 ~ 12 months after EVAR. It has been suggested that four-dimensional flow-sensitive MRI after EVAR was useful for identifying the type of endoleak and predicting dilatation of the aneurysm one year later [21], however there were limitations in the number of facilities available for testing and the inability to test with stainless stent-grafts. Contrast-enhanced ultrasound may be highly accurate in detecting and classifying endoleaks.

Treatment options for type II endoleaks include transarterial chemoembolization, coil embolization, translumbar embolization, laparoscopic surgery, open graft replacement, and aneurysmorrhaphy [22]. While transarterial chemoembolization and coil embolization are the most common treatments, often require repeated intervention. In particular, 51 % of patients treated with only coil embolization required secondary intervention [23]. Patients receiving these secondary interventions were reported to have a higher probability of subsequent additional embolization [23]. Recently translumbar embolization using liquid embolization agents (Onyx, glue, thrombin, polymers) is popular, however these agents are expensive and unintentional leakage has been reported to cause various complications [23]. Laparoscopic branch ligation is less invasive than open surgery, however this procedure is technically difficult and may eventually convert to open surgery if the periaortic area is inflamed [9]. Open graft replacement is a standard surgical treatment, however it is highly invasive because of large incision, aortic cross clamp and, graft explantation and is associated with significant mortality (often ~ 20 %) [8, 24, 25]. Aneurysmorrhaphy, a minimally invasive open surgical repair against aneurysm expansion, is recently reported in some case reports [10, 11]. This procedure is a modified technique that is not require aortic cross clamp, graft explantation, and systemic heparinization. In our institution, aneurysmorrhaphy is standard surgical treatment in patients of aneurysm enlargement after EVAR.

In type II endoleaks, the number of vessels entering the aneurysm varies. Previous studies have reported that risk factors for persistent type II endoleaks are large, patent inferior mesenteric artery, and more than two lumbar arteries on preoperative CTA [26]. Therefore, it has been shown to be reasonable to evaluate the inflow vessels preoperatively. In our institution, aneurysmorrhaphy often caused massive bleeding in the cases of type II endoleak, but not in cases of endotension. The present study demonstrated that intraoperative blood loss is much less in endotension comparing to type II endoleak. Furthermore, intraoperative blood loss in our 18 cases have some tendency to increase in type IIb endoleak (two or more inflow vessels) than type IIa endoleak (one inflow vessel), indicating that the greater the number of inflow vessels, the greater the intraoperative blood loss. Preoperative precise prediction of blood loss helps anesthesiologists to plan an optimum strategy of intraoperative cardiohemodynamic management and leads to reduce unnecessary preoperative blood orders.

We recognize several limitations to this study. Firstly, the blood transfusion trigger was not defined because anesthesiologists were not unified in these 18 cases. Secondly, the total number of aneurysmorrhaphy was small, and this study design was a retrospective and observational.

Conclusion

In conclusion, this case series suggested that the type of endoleak after EVAR may be associated with the amount of intraoperative blood loss in aneurysmorrhaphy. Especially blood loss in endotension cases was much less than in other type of endoleak cases. Preoperative estimation of intraoperative blood loss depending on endoleak type will be helpful for the decision making of anesthetic management in aneurysmorrhaphy.

Abbreviations

EVAR
endovascular aneurysm repair; EL:endoleak; ET:endotension; HT:hypertension; CKD:chronic kidney disease; DM:diabetes mellitus; CAD:coronary artery disease; SD:standard deviation; BMI:body mass index; CAD:coronary artery disease; RBC:red blood cell; CT:computed tomography; CTA:computed tomography angiography; MRI:magnetic resonance imaging; US:ultrasound.

Declarations

Funding

No funding was received.

Availability of data and materials

The datasets used and analyzed during the present study are available from the corresponding author on reasonable request.

Authors' contribution

KO: data collection and drafting the manuscript. KI: study protocol and drafting the manuscript. YS: statistical analysis. MH: data collection and drafting the manuscript. SU: study protocol and revising the manuscript. All authors read and approved the final manuscript.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of Jikei University (29-316[8932]). The requirement for written informed consent was waived by the Ethics Committee of Jikei University because the procedures did not require any additional intervention, and all data were analyzed anonymously. All methods were carried out in accordance with relevant guidelines and regulations.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing of interests.

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References

1. Zarins CK, White RA, Schwarten D, Kinney E, Diethrich EB, Hodgson KJ, Fogarty TJ: AneuRx stent graft versus open surgical repair of abdominal aortic aneurysms: multicenter prospective clinical

trial. J Vasc Surg 1999, 29:292–305.

2. EVAR Trial Participants: Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. Lancet 2005, 365:2179–86.
3. Patel R, Sweeting MJ, Powell JT, Greenhalgh RM: Endovascular versus open repair of abdominal aortic aneurysm in 15-years' follow-up of the UK endovascular aneurysm repair trial 1 (EVAR trial 1): a randomised controlled trial. Lancet 2016, 388:2366–74.
4. Matsumura JS, Brewster DC, Makaroun MS, Naftel DC: A multicenter controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysm. J Vasc Surg 2003, 37:262–71.
5. Mehta M, Sternbach Y, Taggert JB, Kreienberg PB, Roddy SP, Paty PS, Ozsvath KJ, Darling RC 3rd: Long-term outcomes of secondary procedures after endovascular aneurysm repair. J Vasc Surg 2010, 52:1442–9.
6. Lal BK, Zhou W, Li Z, Kyriakides T, Matsumura J, Lederle FA, Freischlag J: Predictors and outcomes of endoleaks in the Veterans Affairs Open Versus Endovascular Repair (OVER) Trial of Abdominal Aortic Aneurysms. J Vasc Surg 2015, 62:1394–404.
7. Chaikof EL, Brewster DC, Dalman RL, Makaroun MS, Illig KA, Sicard GA, Timaran CH, Upchurch GR Jr, Veith FJ: Society for Vascular Surgery. The care of patients with an abdominal aortic aneurysm: the Society for Vascular Surgery practice guidelines. J Vasc Surg 2009, 50:S2-49.
8. Moulakakis KG, Dalainas I, Mylonas S, Giannakopoulos TG, Avgerinos ED, Liapis CD: Conversion to open repair after endografting for abdominal aortic aneurysm: a review of causes, incidence, results, and surgical techniques of reconstruction. J Endovasc Ther 2010, 17:694–702.
9. Yamada M, Takahashi H, Tauchi Y, Satoh H, Matsuda H: Open Surgical Repair Can Be One Option for the Treatment of Persistent Type II Endoleak after EVAR. Ann Vasc Dis 2015, 8:210–4.
10. Ferrari M, Sardella SG, Berchiolli R, Adami D, Vignali C, Napoli V, Serino F: Surgical treatment of persistent type 2 endoleaks, with increase of the aneurysm sac: indications and technical notes. Eur J Vasc Endovasc Surg 2005, 29:43–6.
11. Hiraoka A, Yoshitaka H, Chikazawa G, Ishida A, Totsugawa T, Kuinose M: A Modified Technique of Open Surgical Treatment for Aneurysmal Sac Enlargement after Endovascular Repair. EJVES Extra 2012, 23:e45-e7.
12. van Marrewijk CJ, Fransen G, Laheij RJ, Harris PL, Buth J: Is a type II endoleak after EVAR a harbinger of risk? Causes and outcome of open conversion and aneurysm rupture during follow-up. Eur J Vasc Endovasc Surg 2004, 27:128–37.
13. van Marrewijk C, Buth J, Harris PL, Norgren L, Nevelsteen A, Wyatt MG: Significance of endoleaks after endovascular repair of abdominal aortic aneurysms: The EUROSTAR experience. J Vasc Surg 2002, 35:461–73.
14. White GH, May J, Petrasek P: Specific complications of endovascular aortic repair. Semin Interv Cardiol 2000, 5:35–46.
15. Lin PH, Bush RL, Katzman JB, Zemel G, Puente OA, Katzen BT, Lumsden AB: Delayed aortic aneurysm enlargement due to endotension after endovascular abdominal aortic aneurysm repair. J

Vasc Surg 2003, 38:840–2.

16. White GH, May J, Petrasek P, Waugh R, Stephen M, Harris J: Endotension: an explanation for continued AAA growth after successful endoluminal repair. J Endovasc Surg 1999, 6:308–15.
17. Trocciola SM, Dayal R, Chaer RA, Lin SC, DeRubertis B, Ryer EJ, Hynececk RL, Pierce MJ, Prince M, Badimon J, Marin ML, Fuster V, Kent KC, Faries PL: The development of endotension is associated with increased transmission of pressure and serous components in porous expanded polytetrafluoroethylene stent-grafts: characterization using a canine model. J Vasc Surg 2006, 43:109–16.
18. van Nes JG, Hendriks JM, Tseng LN, van Dijk LC, van Sambeek MR: Endoscopic aneurysm sac fenestration as a treatment option for growing aneurysms due to type II endoleak or endotension. J Endovasc Ther 2005, 12:430–4.
19. Turney EJ, Steenberge SP, Lyden SP, Eagleton MJ, Srivastava SD, Sarac TP, Kelso RL, Clair DG: Late graft explants in endovascular aneurysm repair. J Vasc Surg 2014, 59:886–93.
20. Moll FL, Powell JT, Fraedrich G, Verzini F, Haulon S, Waltham M, van Herwaarden JA, Holt PJ, van Keulen JW, Rantner B, Schlosser FJ, Setacci F, Ricco JB: Management of abdominal aortic aneurysms clinical practice guidelines of the European society for vascular surgery. Eur J Vasc Endovasc Surg 2011, 41:S1-S58.
21. Katahashi K, Sano M, Takehara Y, Inuzuka K, Sugiyama M, Alley MT, Takeuchi H, Unno N: Flow dynamics of type II endoleaks can determine sac expansion after endovascular aneurysm repair using four-dimensional flow-sensitive magnetic resonance imaging analysis. J Vasc Surg. 2019, 70:107–16.
22. Baum RA, Carpenter JP, Golden MA, Velazquez OC, Clark TW, Stavropoulos SW, Cope C, Fairman RM: Treatment of type 2 endoleaks after endovascular repair of abdominal aortic aneurysms: comparison of transarterial and translumbar techniques. J Vasc Surg 2002, 35:23–9.
23. Sarac TP, Gibbons C, Vargas L, Liu J, Srivastava S, Bena J, Mastracci T, Kashyap VS, Clair D: Long-term follow-up of type II endoleak embolization reveals the need for close surveillance. J Vasc Surg 2012, 55:33–40.
24. Lipsitz EC, Ohki T, Veith FJ, Suggs WD, Wain RA, Rhee SJ, Gargiulo NJ, McKay J: Delayed open conversion following endovascular aortoiliac aneurysm repair: partial (or complete) endograft preservation as a useful adjunct. J Vasc Surg 2003, 38:1191–8.
25. Brewster DC, Jones JE, Chung TK, Lamuraglia GM, Kwolek CJ, Watkins MT, Hodgman TM, Cambria RP: Long-term outcomes after endovascular abdominal aortic aneurysm repair: the first decade. Ann Surg 2006, 244:426–38.
26. Arko FR, Rubin GD, Johnson BL, Hill BB, Fogarty TJ, Zarins CK: Type-II endoleaks following endovascular AAA repair: preoperative predictors and long-term effects. J Endovasc Ther 2001, 8:503–10.

Tables

Table 1
Clinical characteristics and summary of the diagnosis

Case	Age	Sex	Comorbidities	Diameter of aneurysm (mm)	Preoperative hemoglobin (g/dl)	Preoperative diagnosis	Postoperative diagnosis
1	77	M	HT, CKD	75	12.7	EL2b	EL2b
2	66	M	HT, DM	88	15.1	EL2b	EL2b
3	74	M	HT, CKD, Anticoaglation	94	12.2	EL2a	EL2a
4	84	M	HT, CKD, CAD, Anticoaglation	88	8.3	EL2a	EL2a
5	85	M	HT, DM, CKD	135	8.9	EL2b	EL2b
6	81	M	HT, CKD	132	11.0	EL2b	EL2□EL3
7	67	M	CAD, Anticoaglation	90	14.0	EL2b	EL2b
8	86	F	HT, CKD, Anticoaglation	82	10.9	EL2b	EL2b
9	71	M	HT, Anticoaglation	88	13.2	EL2a	EL2a
10	81	M	HT, Anticoaglation	90	10.7	EL2b	EL2b
11	73	M	Anticoaglation	70	13.3	EL2a	EL2a
12	91	M	HT, CKD	100	9.6	EL2b	EL2b
13	64	M	HT	111	15.1	EL2b	EL2b
14	87	M	HT, CKD, CAD, Anticoaglation	100	10.3	ET	ET
15	79	M	HT, CKD, CAD, Anticoaglation	96	10.2	ET	ET
16	75	M	HT, CAD	76	11.9	ET	ET
17	81	M	HT, CKD	65	14.2	ET	ET
18	86	M	HT, CKD, Anticoaglation	74	11.1	ET	ET

EL: endoleak; ET: endotension; HT: hypertension; DM: diabetes mellitus; CKD: chronic kidney disease; CAD: coronary artery disease

Table 2
Group characteristics and preoperative data in 18 patients

Characteristics	Type ▯ Endoleak (n = 13)	Endotension (n = 5)	P value
Age	76.9 ± 8.2	80.2 ± 4.5	0.14
Male, n (%)	13 (100)	4 (80)	0.28
BMI (kg/▯)	23.7 ± 3.8	22.8 ± 1.2	0.30
Hypertension, n (%)	11 (85.6)	5 (100)	1.00
Diabetes mellitus, n (%)	2 (15.3)	0	1.00
Chronic kidney disease, n (%)	7 (53.8)	4 (80)	0.59
Coronary artery disease, n (%)	2 (15.4)	3 (60)	0.10
Anticoagulation, n (%)	7 (53.8)	3 (60)	1.00
Diameter of aneurysm (mm)	96 ± 19	82 ± 14	0.19
EVAR to aneurysmorrhaphy Duration (month)	68 ± 25	87 ± 19	0.14
Preoperative hemoglobin (g/dl)	11.9 ± 2.2	11.5 ± 1.6	0.73
Preoperative hematocrit (%)	35.7 ± 6.5	35.3 ± 4.9	0.90

Values are expressed as mean ± SD. No significant differences were observed among the groups.

SD: standard deviation; BMI: body mass index; EVAR: endovascular aneurysm repair

Table 3
Clinical outcomes of perioperative period

Case	Operation time (minutes)	Postoperative diagnosis	Intraoperative blood loss (mL)	RBC transfusion (ml)
1	231	EL2b	1,570	240
2	236	EL2b	1,610	630
3	161	EL2a	620	560
4	254	EL2a	1,300	1680
5	361	EL2b	9,170	5690
6	280	EL2□EL3	2,940	1960
7	272	EL2b	3,830	1270
8	602	EL2b	8,130	6300
9	320	EL2a	2,280	440
10	378	EL2b	6,420	4620
11	161	EL2a	310	0
12	460	EL2b	12,500	7480
13	320	EL2b	1,437	0
14	235	ET	80	0
15	268	ET	30	0
16	198	ET	285	0
17	128	ET	30	0
18	197	ET	100	560

EL: endoleak; ET: endotension; RBC: red blood cell

Table 4
Comparison of Intraoperative blood loss and RBC transfusion between endoleak and endotension group

	Endoleak (n = 13)	Endotension (n = 5)	P value
Intraoperative blood loss (ml)	2,280 [310–12,500]	80 [30–285]	0.0016
RBC transfusion (ml)	1,270 [0–7,480]	0 [0–560]	0.018
Values are expressed as median [minimum and maximum].			
RBC: red blood cell			

Figures

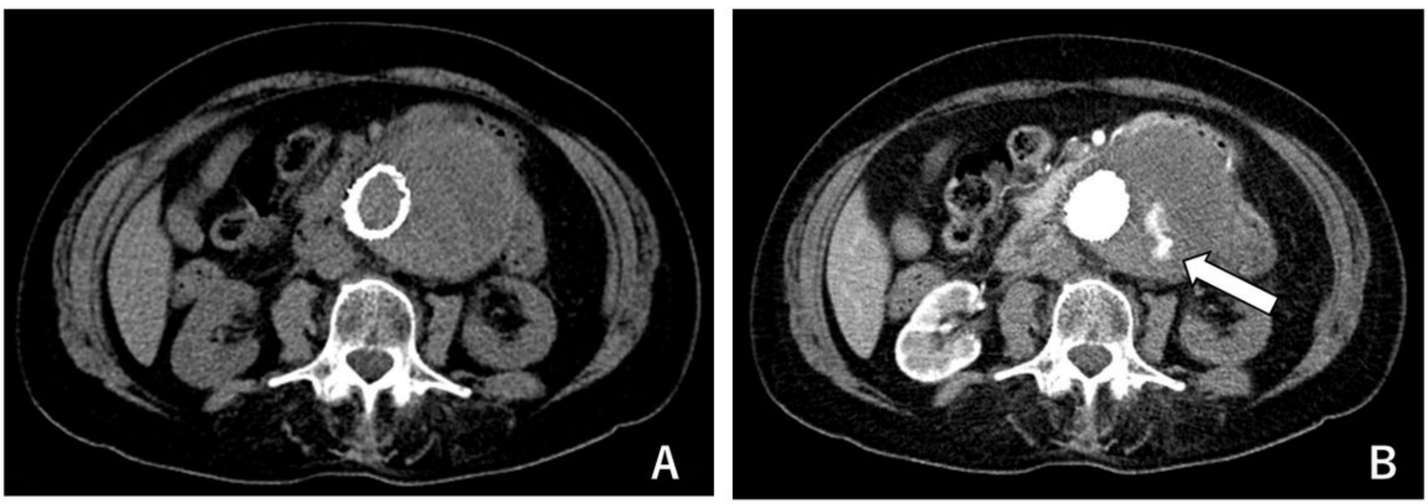

Figure 1

Preoperative plain and contrast-enhanced CT on type 2 endoleak after EVAR. (A) A plain CT image shows the high intensity prosthetic stent graft inside enlarged abdominal aortic aneurysm. (B) On contrast-enhanced CT, a contrast agent inflow outside of endoprosthesis lumen and within the aneurysm sac is seen developing from lumber artery (arrow). CT: computed tomography; EVAR: endovascular aneurysm repair.

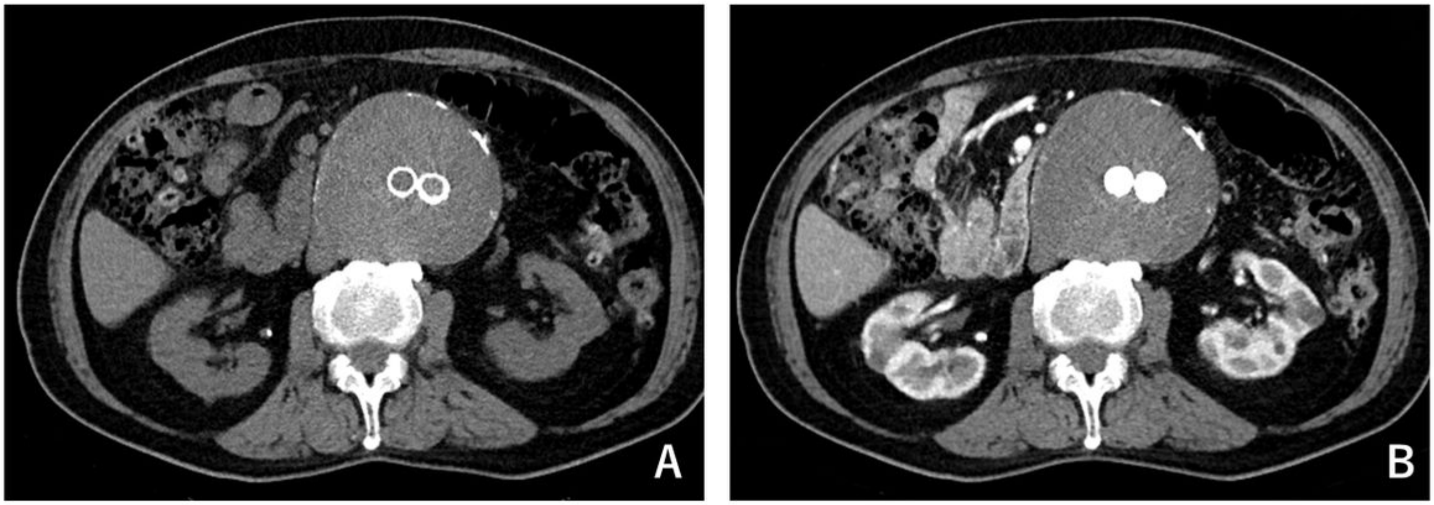

Figure 2

Preoperative plain and contrast-enhanced CT on endotension after EVAR. (A) On plain CT, high intensity prosthetic stent graft is seen inside enlarged abdominal aortic aneurysm. (B) On contrast-enhanced CT, stent graft lumen is well visualized with contrast agent, however no contrast agent is seen in the aortic aneurysm, suggesting no inflow vessels. CT: computed tomography; EVAR: endovascular aneurysm repair.

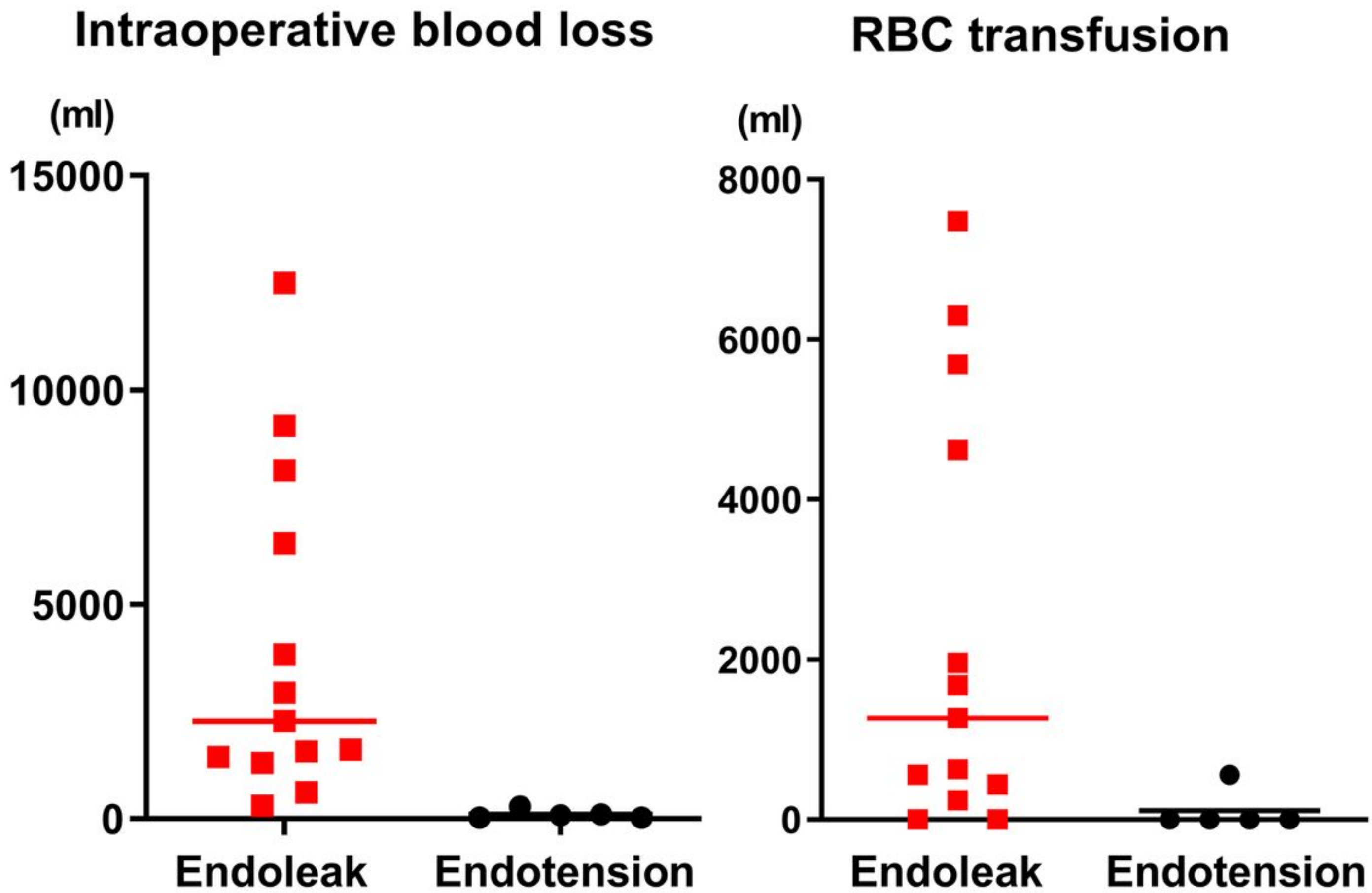

Figure 3

Comparison of intraoperative blood loss and RBC transfusion volume between endoleak and endotension groups in aneurysmorrhaphy. * P=0.0016 and # P=0.018 versus Endoleak, respectively. Each column is expressed as scattered dot plots with median bar. RBC, red blood cells.