ELSEVIER

Clinical Research

Heterogeneous Characteristics of Patients with Inflammatory Abdominal Aortic Aneurysm. Systematic Review of Therapeutic Solutions

Antonio V. Sterpetti,[1] Vittorio Arici,[2] Milamaria Franciscone,[2] Giuseppe D'Ermo,[1] Luca Di Marzo,[1] Maria Vittoria Carati,[1] Umberto Costi,[1] Franco Ragni,[2] Eloisa Arbustini,[2] and Antonio Bozzani,[2] Rome and Pavia, Italy

Background: Endovascular repair of inflammatory abdominal aortic aneurysms (IAAAs) has emerged as an alternative to open surgery, but direct comparisons are limited. The aim of the study was to compare clinical outcomes of endovascular and open repair for IAAA according with specific clinical characteristics.

Methods: We performed a literature review of reports describing patients who had open or endovascular repair for IAAA. A literature search was performed in June 2022 by 2 investigators who conducted a review of papers reported in PubMed, Embase, MEDLINE, and Cochrane Database. The strings ''Inflammatory aneurysm'' and ''Abdominal Aortic Aneurysms'' were used. There was no language restriction and screened reports were published from March 1972 to December 2021. We identified 2,062 patients who had open (1,586) or endovascular repair (476) for IAAA. Primary outcomes were operative mortality and morbidity. Secondary outcomes were complications during follow-up (mean follow-up: 48 months). Propensity score matching was performed between patients who had open or endovascular surgery.

Results: In Western countries, propensity-weighted postoperative mortality (in-hospital) (1.5% endovascular vs. 6% open) and morbidity rates (6% vs. 18%) were significantly lower in patients who had endovascular repair ($P < 0.0001$); patients with larger aneurysm (more than 7 cm diameter), signs of active inflammation, and retroperitoneal rupture of the aneurysm had better outcomes after endovascular repair than after open surgery. Hydronephrosis was present in 20% of the patients. Hydronephrosis regressed in most patients when signs of active inflammation were present suggesting an acute onset of the hydronephrosis itself (fever, elevated serum C Reactive Protein) either after endovascular or open surgery. Long-standing hydronephrosis as suggested by the absence of signs of active inflammation rarely regressed after endovascular surgery despite associated steroid therapy. During a mean follow-up of 48 months, propensity-weighted graft-related complications were more common in patients who had endovascular repair (20% vs. 8%). For patients from Asia, short-term and medium-term results were

The authors have no conflicts of interest to declare.

No funds were received for this work.

Author Contributions: Antonio Sterpetti conceived the research and wrote the manuscript. Umberto Costi, Maria Vittoria Carati, Luca Di Marzo, and Giuseppe D'ermo collected data from literature. Antonio Bozzani, Franco Ragni, and Vittorio Arici gave suggestions and organized the research.

The registration number at PROSPERO was CRD 4201808633.

[1]*Sapienza University of Rome, Rome, Italy.*

[2]*IRCCS Policlinico San Matteo, Pavia, Italy.*

Correspondence to: Antonio V. Sterpetti, Sapienza University, Viale del Policlinico, 00167, Rome, Italy; E-mail: antonio.sterpetti@uniroma1.it

Ann Vasc Surg 2023; ■: 1–9

https://doi.org/10.1016/j.avsg.2023.06.036

Manuscript received: May 13, 2023; manuscript accepted: June 27, 2023; published online: ■ ■ ■

similar after open and endovascular repair. IAAAs related with aortitis were more common in Asia. In Western countries, IAAAs were commonly associated with atherosclerosis.

Conclusions: Patients with IAAA represent a heterogeneous population, suggesting biological differences from continent to continent; conservative therapy and endovascular or open surgery should be chosen according to the patient clinical condition. Endovascular repair presents advantages in patients with signs of active inflammation and contained rupture of the IAAA and larger aneurysms. Hydronephrosis, without signs of active inflammation, rarely regresses after endovascular repair associated with steroid therapy. Further studies are needed to establish the long-term results of endovascular repair.

De Weerd et al.[1] in 1955 reported on a 45-year-old patient with bilateral hydronephrosis. At the operation, both ureters were encased in a thick, whitish, fibrotic reaction, starting from an aneurysm of the abdominal aorta. Bilateral ureterolysis was performed with normalization of renal function. The aneurysm was left in place. The same year Shumacker and Garrett[2] performed a successful resection of an inflammatory abdominal aortic aneurysm (IAAA), with bilateral ureterolysis. The postoperative course was uneventful with return of a regular renal function. Walker et al.[3] reviewed the clinical outcome of 187 patients who had resection of an abdominal aortic aneurysm (AAA): in 19 patients (10%) the aneurysm was surrounded by a ''thick, whitish fibrosis''. They used the term ''inflammatory aneurysm'', noting the higher surgical mortality (26%) for these patients. Crawford et al.[4] defined some of the operative principles to prevent complications in surgery for IAAA, including minimal dissection of the aneurysm: Open surgery for IAAA is complications prone for the peri-aneurysmal fibrosis involving the adjacent structures (Figs. 1 and 2).[5,6] Endovascular repair offers several advantages, avoiding a complex dissection with the possibility of injuries to the duodenum, small bowel, and vena cava. Concerns remain about the medium-term and long-term results of endovascular surgery in this clinical setting. The aim of this study was to analyze the results of open and endovascular surgery for patients with IAAA.

MATERIALS AND METHODS

The methods used for the study and inclusion criteria were based on PRISMA and AMSTAR recommendations. A literature search was performed in June 2022 by 2 investigators who conducted a review of papers reported in PubMed, Embase, MEDLINE, and Cochrane Database. The strings ''Inflammatory aneurysm'' and ''Abdominal Aortic Aneurysms'' were used. There was no language restriction and screened reports were published from March 1972 to December 2021 (Supplement). Papers reporting patients with a follow-up less than 6 months were not included. Data extraction was performed by 2 reviewers independently; a third reviewer was involved to solve any question in interpreting data. The primary outcomes were operative mortality and morbidity. Secondary outcomes were complications related to therapy and survival. Data were analyzed according to the geographical areas. The presence of an IAAA was defined for a significant peri-aneurysmal fibrosis at computed tomography (CT) scan or at operation. Patients with Beçhet disease and AAA were considered separately. The registration number at PROSPERO was CRD4201808633. This systematic review was approved by the Council of our Department and from the Ethics Committee of our University and Hospital. Being a systematic review of already published data, written consent was not required.

Quality Assessment

Two independent reviewers assessed the quality and risks of biases of the analyzed studies by using the Newcastle-Ottawa scale. Papers with a score more than 6 were considered of good quality (scale 0 to 9).

Geographical Regions

Results were analyzed by an overall point of view as well as according to specific geographical areas: (1) USA-Canada-Europe, (2) North Africa and Middle East, (3) India-Pakistan, (4) Japan-China-South Korea, and (5) Mexico-Central and South America. USA, Canada, and Europe were considered together for similar clinical characteristics of the patients and similar operative results.

Statistical Analysis

Interstudy heterogeneity was assessed by I^2 statistics as a measure describing degree of heterogeneity. I^2 results less than 50% were considered as low

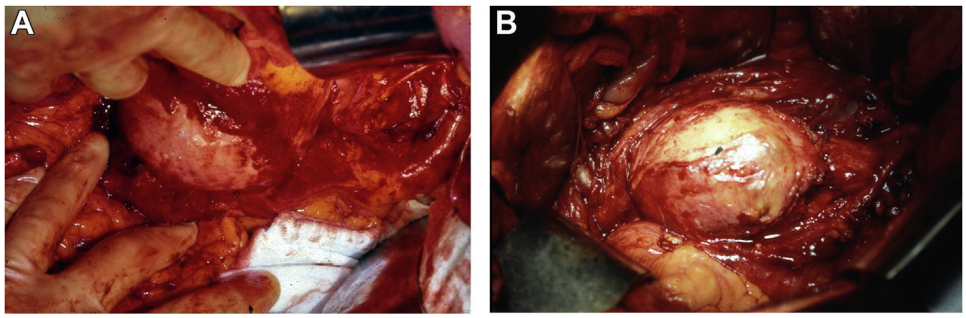

Fig. 1. (A–B) Intraoperative view of inflammatory abdominal aortic aneurysm: The dissection is kept to a minimum to avoid injuries to the adjacent structures.

heterogeneity; I^2 more than 50% were considered with high heterogeneity. Comparisons were made by Chi-square test and Student's *t*-test where appropriate. Log-rank test was used to compare actuarial survival rates after surgery between selected groups of patients. Multivariate analysis was not possible because of incomplete data in the majority of the study.

Propensity Score Matching

Results were compared for selected patients who had open surgery or endovascular surgery in USA-Canada-Europe. Two hundred patients who had open surgery were compared to 100 patents who had endovascular surgery. Patients were selected by random sampling within strata: all observations were ranked on their propensity score, and the data were then divided into quantiles of the propensity score. Within each stratum, equal sample sizes in the treatment and control groups were selected. Matching within calipers was proposed to protect against a treated and control observation not similar to each other in their propensity score. For covariate balance, we used the standardized differences after weighting. These were all <0.1, indicating adequate balancing by the propensity score model. Patients were matched on characteristics that predict receipt of 1 treatment strategy over another. Analyzed preoperative clinical characteristics of the patients were age, gender, symptoms if present (fever, abdominal pain, and weight loss), general clinical conditions, comorbidities, associated autoimmune diseases, and previous cardiovascular events. Other included variables were maximum transverse diameter of the AAA, intact or ruptured aneurysm (free or contained rupture), and presence of aneurysms in other arterial segments.

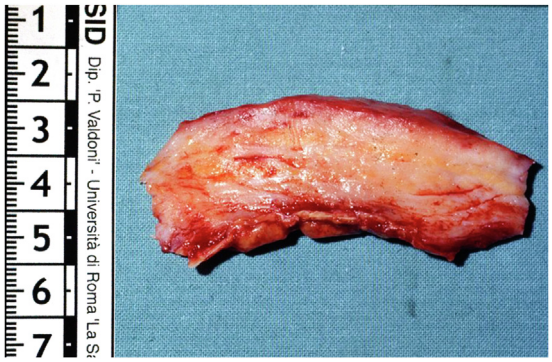

Fig. 2. Aneurysmal wall of an IAAA.

For covariate balance, we used the standardized differences after weighting. These were all <0.1, indicating adequate balancing by the propensity score model. We used weighted Cox regression to compare overall survival and overall disease-free survival.

Definition of Complications

The postoperative morbidity (in-hospital) was recorded as a total percentage and based on the presence of significant complications according to the Clavien-Dindo Classification (Class III-IV-V).[7] If the Clavien-Dindo Classification was not mentioned in the reports, grades were assigned based on the information provided. We also considered as major complications all negative events which required a new intervention either after open or endovascular repair.

RESULTS

Two thousand and six hundred articles were screened: 94 papers were included in the analysis (Supplement Methods). There was no randomized

Table I. Comparison between the overall results of open and endovascular surgery for IAAA in USA-Canada-Europe

Early and late results	Open repair (1,276 patients)	Endovascular surgery (166 patients)
Operative mortality (30 days)	6.8%	1.8%
Operative morbidity (30 days)	25%	5%
Perianeurysmal fibrosis	(124 patients)	(55 patients)
Significant regression	73%	62%
Unchanged	26%	34%
Progression	1%	4%
Ureteral entrapment	(85 patients)	(32 patients)
Significant regression	69%	44%
Unchanged	22%	44%
Progression	9%	12%

study. Most studies were retrospective. Few prospective studies (10 papers) were included, but details about the organization of the study were not clearly reported. The average Newcastle Ottawa score was low. Study heterogeneity ranged from 30% to 60%, with an average of 46%. A total of 2,054 patients were collected: 1,578 had open surgery (only 1,394 are included because 184 patients had incomplete information or very short follow-up) and 476 had endovascular surgery. Postoperative mortality ranged from 20% to 0% and morbidity rates from 50% to 10%. We considered separately patients from different continents because of a significant difference in results of open and endovascular surgery and specific clinical characteristics suggesting various etiologies and pathophysiology of the IAAA.

USA-Canada-Europe

Open surgery—clinical characteristics. Overall, 1,276 patients with intraoperatively defined IAAA were considered for the analysis. Twenty of these patients had associated autoimmune disease (1.6%), but histology did not show a significant difference from patients with IAAA and no associated autoimmune disease. Three patients had Takayasu disease; histology confirmed the nature of the aneurysm. The clinical characteristics of 200 patients with IAAA were compared with those of 200 patients with simple atherosclerotic-degenerative aneurysm. Patients with inflammatory AAA complained more often from pain ($P < 0.001$) and they had increased serum inflammatory markers (C-reactive protein) ($P < 0.001$). Comorbidities, age, and sex distribution were similar between patients with IAAA and simple atherosclerotic-degenerative aneurysm. IAAAs had lower prevalence of rupture (5%), and the rupture was generally contained into the retro-aortic space ($P < 0.0001$).[7–18]

Open surgery—results. Tables I and II show the operative mortality and morbidity. There was a significant improvement in results after the year 1985, when the operative details of minimal aneurysm dissection were popularized, and after the year 2008, when endovascular surgery started to be accepted and performed in patients with IAAA (Table III). Reports from the same centers showed improved results in time ($P < 0.0001$), namely after the introduction of endovascular surgery.[7–13] Five papers compared the results for patients with IAAA with those for patients with simple atherosclerotic-degenerative aneurysm and no statistically significant difference was found for the low number of observations.[14–18] However, aggregated data of the 5 papers show a significant increased operative mortality (7% vs. 2%) and postoperative morbidity (25% vs. 15%) in patients with IAAA in comparison with patients with simple AAA ($P < 0.0001$). Peri-aneurismal fibrosis regressed in 73% of the patients in whom serial postoperative CT scans were performed (Table I).

Endovascular Surgery—Clinical Characteristics

One hundred and sixty six patients who had endovascular repair for IAAA were collected. The aneurysm was defined for the increased thickness of the peri-aneurysmal tissue at CT scan. The incidence of associated hydronephrosis (22%) was similar with that of patients with IAAA who had open surgery.

Endovascular Surgery—Results

Table I shows the results of endovascular surgery compared with those of open surgery. Ten patients

Table II. Results of open surgery for inflammatory abdominal aortic aneurysms

Author	Type of study	*N* patients	Analyzed years	Postop mortality	morbidity
Paravastu et al.[7]	Systematic Review	999	1972–2008	6.9%	NA
Kakkos et al.[13]	Systematic Review	459	1972–1999	9.8%	NA
Kakkos et al.[13]	Systematic Review	300	1999–2013	4%	NA
Current Review	Systematic Review	205	1972–1984	12%	30%
Current Review	Systematic Review	350	1985–1994	7.5%	20%
Current Review	Systematic Review	580	1995–2007	6.2%	15%
Current Review	Systematic Review	443	2008–2019	3%	10%

Table III. Overall results of open and endovascular surgery for IAAA performed in the same period in the same centers in the last 10 years

Authors year publication		Mortality EVAR[a]	Mortality open	Overall mortality (30 days from Surgery)
Coppi et al.[19]	2010	0/9 (0%)	?/9	?
Stone et al.[10]	2012	0/10 (0%)	1/59 (1.6%)	1/69 (1%)
Zhang et al.[20]	2014	0/8 (0%)	0/5 (0%)	0/13 (0%)
Kakkos et al.[13]	2015	0/9 (0%)	0/10 (0%)	0/19 (0%)
Kasashima at al[21]	2017	0/17 (0%)	0/23 (0%)	0/40 (0%)
Georgakarakos et al.[22]	2017	0/1 (0%)	0/1 (0%)	0/2 (0%)
Duques Santos et al.[23]	2018	0/5 (0%)	1/29 (3.4%)	1/34 (2.9%)
Total		0/59 (0%)	2/128 (1.5%)	2/187 (1.0%)

[a]30-days mortality.

had endovascular surgery, after open surgery was abandoned for difficulties in the dissection. Operative mortality and morbidity rates were significantly lower than those after open surgery. One hundred patients who had endovascular surgery were compared with 200 patients who had open surgery by propensity score matching 1 to 2. Operative mortality and morbidity were significantly lower for patients who had endovascular repair in comparison with patients who had open surgery (endovascular repair operative mortality 1.5% and operative morbidity 6%; open surgery operative mortality 6% and operative morbidity 18%; $P < 0.001$). This difference was more evident in patients who were operated upon in emergency conditions, in patients who had clinical (fever-abdominal pain and tenderness) and serologic signs (increased serum C-reactive protein) of active inflammation and in patients with an aneurysm with transverse diameter larger than 7 cm ($P < 0.001$). Endovascular surgery resulted in a lower rate and speed for regression of peri-aneurismal fibrosis. During a mean follow-up of 48 months, reinterventions were more common in patients who had endovascular repair (20% vs. 8%) ($P < 0.0001$). After the introduction of new graft materials and the routine use of steroids, immediately before and after endovascular surgery, worsening of the peri-aneurismal fibrosis was seldom reported. Hydronephrosis, when present, regressed more frequently when associated with clinical and/or hematological sings of inflammation (12/13). In patients with no evidence of clinical and/or hematological signs of acute inflammation, hydronephrosis rarely regressed (3/19).

Asian Countries

Open surgery—clinical characteristics. Overall, 124 patients who underwent open surgery were collected. Seventeen patients had IG4-related disease, 4 patients IG4 not related chronic periaortitis, and 1 patient Takayasu disease. The remaining 102 were defined as having atherosclerotic-degenerative AAA; however, patients with atherosclerotic-degenerative disease had lower prevalence of associated disease in comparison with patients with atherosclerotic IAAA from USA-Canada and Europe, suggesting the possibility of a less virulent form of atherosclerosis. This was indirectly confirmed by the higher 5-year actuarial survival rate (78% vs. 70%, $P < 0.001$).

Open surgery—results. There was only one postoperative mortality (1/124 = 0.8%). Reported postoperative complication rate was less than 5%. Postoperative mortality and morbidity were significantly lower in comparison with patients operated

upon in USA-Canada-Europe ($P < 0.001$). Mean follow-up was 48 months. Four patients had an anastomotic pseudoaneurysm and 1 patient an aorto-enteric fistula.

Endovascular surgery—clinical characteristics. Three hundred nine patients with inflammatory aneurysm were reported: 278 were extracted from the Japan National Statistics.[24]

Thirty one patients were reported in single-institution studies. There were 2 cases of postoperative mortality (2/309 = 0.6%). During a mean follow-up of 28 months, there were 3 patients with graft rupture (3/309 = 1%). Early results after endovascular repair were similar with those after open surgery. Reintervention rates were also similar after open and endovascular surgery. Worsening of peri-aneurysmal fibrosis was reported in 9 of 10 patients with IG4-related disease; none of those patients had associated steroid therapy.[21,25]

We were able to find only 2 reports of 2 young patients (aged 38 and 39 years; 1 male; 1 female) who underwent successful open resection for a Takayasu-related IAAA.[26–33]

North Africa-Middle East

The possibility of abdominal aneurysm or pseudoaneurysm related with Beçhet disease was high in these regions, with a higher prevalence in young males (average age: 35 years). There was an increasing use of endovascular surgery, with improved results in comparison with open surgery.[34–36] We found only 1 report from Turkey which described the clinical outcome of 17 patients after open surgery for repair of IAAA probably atherosclerotic in nature.[16] The postoperative mortality was 11% (2/17) and complication rate was 29%, mainly related with renal failure.

DISCUSSION

Several theories have been proposed about the etiology of IAAA. Rose and Dent in 1981[35] demonstrated an inflammatory reaction in almost all AAAs: in 6 patients, the inflammatory reaction was severe. Other reports have hypothesized a major role of autoimmunity in the genesis of IAAA, extrapolating the etiological events associated with the formation of chronic aortitis to the development of IAAA in general.[36–39] The Society for Cardiovascular Pathology has provided guidelines to distinguish IAAA associated with severe atherosclerosis from those associated with aortitis and peri-aortitis.[40] The term Inflammatory Atherosclerotic Abdominal Aneurysms should be used to introduce a clear distinction between atherosclerosis-related IAAA and other forms of aortitis and peri-aortitis, which can result also in aneurysmal degeneration. Atherosclerosis-related IAAA shows severe atherosclerosis and excessive degree of adventitial inflammation, consisting mainly on lymphocytes and plasma cells. The thickness of the aortic wall should exceed 4 mm to make a diagnosis of IAAA. The presence in the adventitia of histological findings not typical for atherosclerosis (like granulomata, extensive IG4+) combined with no significant or mild atherosclerosis favors the diagnosis of peri-aortitis.[40] In our review, we found a significant difference in the distribution and etiology of IAAA. Atherosclerotic IAAA was the most common form in USA-Canada and Europe; in Asia, there was a significant prevalence of IAAA related with aortitis. CT scan shows in IAAA related with aortitis a homogeneous distribution of the peri-aneurysmal fibrosis, involving also the posterior wall. In patients with atherosclerotic-related IAAA, the peri-aneurysmal fibrosis is confined to the anterior and lateral walls.

Patients from Asia with atherosclerotic IAAA had less comorbidities, better operative results, and survival rates. These findings support the hypothesis of different forms of atherosclerosis between Western and Eastern countries. Moreover, patients with Takayasu disease had different characteristics when comparing patients from Asia, with a high prevalence of AAA, with those from Mexico and South America in whom associated AAA was uncommon.

Endovascular surgery resulted in reduced mortality and morbidity in comparison with open surgery in patients with contained retro-aortic rupture, evidence of active inflammation (either clinically—abdominal pain, fever, weight loss—or based on serologic inflammatory markers), and with larger aneurysms in USA-Canada-Europe. In an average follow-up of 48 months, complications and reinterventions were higher after endovascular repair than after open surgery in USA-Canada-Europe, but not in East Asia. Regression of peri-aneurismal fibrosis was more common and faster after open surgery. Regression of hydronephrosis related to IAAA was more common in patients with active, recent-onset inflammation either after open or endovascular surgery. The absence of active inflammation, presuming a chronic inflammation with long-standing peri-aortic fibrosis, was associated with reduced rates of regression of the hydronephrosis, especially after endovascular surgery.[7,13,41–44]

These findings represent the theoretical basis for an aggressive medical and surgical therapeutic approach in patients with IAAA and clinical and

Fig. 3. CT scan of a patient with an atherosclerotic IAAA. The peri-aneurysmal fibrosis is present only in the anterior and lateral segments.

Fig. 4. CT scan of a patient with an IAAA related with aortitis. The peri-aneurysmal fibrosis involves also the posterior segment.

serologic evidence of active inflammation before chronic fibrosis supervenes. In presence of markers for active inflammation, endovascular surgery associated with steroids represents a valid therapeutic approach. Short course of steroids followed by open surgery may be a realistic alternative. In patients with IAAA and no signs of active inflammation, the presence of hydronephrosis should be considered related with chronic fibrosis. In this clinical setting, regression of the hydronephrosis is uncommon after endovascular surgery, even if combined with steroid therapy. Open IAAA repair and ureterolysis may be a more appropriate solution: steroid therapy may help to prevent recurrent ureteric obstruction.

CONCLUSION

Patients with IAAA represent a significant heterogeneous group. Conservative treatment in older patients, with reduced life expectancy, with smaller aneurysm (<5 cm in maximum transverse diameter), without associated hydronephrosis, is an acceptable therapeutic option.[45–47] Endovascular surgery associated with steroids is followed by good clinical outcomes, with significantly reduced operative mortality and morbidity in comparison with open surgery, and it should be considered the optimal treatment for patients with contained rupture, active inflammation, and larger aneurysms.[48,49] The initial concerns that endovascular surgery might lead to worsening of the peri-aneurysmal inflammation seem to reduce because the evidence of improved early result of endovascular surgery with lower perioperative complications and the reduced incidence of the problem for new graft materials and diffusion of associated steroids therapy. Open surgery in patients with longer life expectancy, with chronic inflammatory conditions, and hydronephrosis represents a valid choice for the higher possibility of hydronephrosis regression in comparison with endovascular repair.

The therapeutic approach in patients with IAAA should be chosen according to the specific clinical characteristics and expectations of each single patient[50–52] (Figs. 3 and 4). Conservative medical treatment and open or endovascular surgery should be considered valid complementary options. Table III shows the improved results reported in centers where endovascular or open repair were selected according to the characteristics of the patients.

Limitations

Our review implies several possible biases: retrospective nature of most studies, incomplete and not uniform reported data, and low quality of papers by New Castle grading.

The definition of an IAAA was approximate and not uniform and information about clinical presentation (elective and emergency) was not reported in several papers. Despite these limitations, the review offers an overall view of the current controversies about the proper treatment of patients with IAAA and underlines the importance of personalized therapy in treating this heterogeneous group of patients.[53,54] Patients with IAAA have been often classified as a homogeneous group of patients; general guidelines for treatment are not appropriate,

without considering the specific conditions and needs of each singe patient.

REFERENCES

1. DeWeerd JH, Ringer MG, Pool TL, et al. Aortic aneurysm causing bilateral ureteral obstruction: report of a case. J Urol 1955;74:78–81.
2. Schumacker HB Jr, Garrett R. Obstructive uropathy from abdominal aortic aneurysm. Surg Gynecol Obstet 1955;100:758–61.
3. Walker DI, Bloor K, Williams G. Gille inflammatory aneurysms of the abdominal aorta. Br J Surg 1972;59:609–14.
4. Crawford JL, Stowe CL, Safi HJ, et al. Inflammatory aneurysms of the aorta. J Vasc Surg 1985;2:113–24.
5. Caradu C, Ammollo RP, Dari L, et al. Management of inflammatory aortic aneurysms - a scoping review. Eur J Vasc Endovasc Surg 2023;65:493–502.
6. Xu J, Bettendorf B, D'Oria M, et al. Multidisciplinary diagnosis and management of inflammatory aortic aneurysms. J Vasc Surg 2023;78:231–242.e2.
7. Paravastu SC, Ghosh J, Murray D, et al. A Systematic review of open versus endovascular repair of inflammatory abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2009;38:291–7.
8. Pennell RC, Hollier LH, Lie JT, et al. Inflammatory abdominal aortic aneurysms: a thirty-year review. J Vasc Surg 1985;2:859–69.
9. Nitecki SS1, Hallett JW Jr, Stanson AW, et al. Pairolero PC inflammatory abdominal aortic aneurysms: a case-control study. J Vasc Surg 1996;23:860–8.
10. Stone WM, Fankhauser GT, Bower TC, et al. Money SR comparison of open and endovascular repair of inflammatory abdominal aortic aneurysms. J Vasc Surg 2012;56:951–6.
11. Paravastu SC, Murray D, Ghosh J, et al. Inflammatory abdominal aortic aneurysms (IAAA): past and present. Vasc Endovascular Surg 2009;43:360–3.
12. Wieker CM, von Stein P, Bianchini Massoni C, et al. Long-term results after open repair of inflammatory infrarenal aortic aneurysms. J Vasc Surg 2019;69:440–7.
13. Kakkos SK, Papazoglou KO, Tsolakis IA, et al. Open versus endovascular repair of inflammatory abdominal aortic aneurysms: a comparative study and Meta-analysis of the literature. Vasc Endovascular Surg 2015;49:110–8.
14. Lindblad B, Almgren B, Bergqvist D, et al. Abdominal aortic aneurysm with perianeurysmal fibrosis: experience from 11 Swedish vascular centers. J Vasc Surg 1991;13:231–7.
15. Bonamigo TP1, Bianco C, Becker M, et al. Inflammatory aneurysms of infra-renal abdominal aorta. A case-control study. Minerva Cardioangiol 2002;50:253–8.
16. Yusuf K, Murat B, Unal A, et al. Inflammatory abdominal aortic aneurysm: predictors of long-term outcome in a case-control study. Surgery 2007;141:83–9.
17. Arroyo A, Rodríguez J, Porto J, et al. Management and course of hydronephrosis secondary to inflammatory aneurysms of the abdominal aorta. Ann Vasc Surg 2003;17:481–5.
18. Bonati L, Rubini P, Japichino GG, et al. Long-term outcome after inflammatory abdominal aortic aneurysm repair: case-matched study. World J Surg 2003;27:539–44.
19. Coppi G, Rametta M, Aiello S, et al. Inflammatory abdominal aortic aneurysm endovascular repair into the long term follow up. Ann Vasc Surg 2010;24:1053–9.
20. Zhang M, Guo W, Du X, et al. Treatment strategies of inflammatory abdominal aortic aneurysms. Zhonghua Yi Xue Za Zhi 2014;94:348–51.
21. Kasashima S, Kasashima F, Kawashima A, et al. Clinical outcomes after endovascular repair and open surgery to treat immunoglobulin G4 related and not related inflammatory abdominal aortic aneurysms. J Endovasc Therapy 2017;24:833–45.
22. Georgakarakos E, Schoretsanitis N, Koufopoulos G, et al. Abdominal pain in the presence of small abdominal aortic aneurysms: if in doubt, cut it out!. Ann Vasc Surg 2017;40:300.e17–21.
23. Duque Santos A, Valdivia AR, Romero Lozano MR, et al. Outcomes of open and endovascular repair of inflammatory abdominal aortic aneurysms. Vascular 2018;26:203–8.
24. Hoshina K, Ishimaru S, Sasabuchi Y, et al., Japan Committee for Stentgraft Management. Outcomes of endovascular repair for abdominal aortic aneurysms: a nationwide survey in Japan. Ann Surg 2019;269:564–73.
25. Sakai K, Watanabe T, Yoshida T. Endovascular treatment of immunoglobulin G4-related inflammatory abdominal aortic aneurysm. J Vasc Surg Cases Innov Tech 2018;4:189–92.
26. Kallappa Parameshwarappa S, Mandjiny N, Kavumkal Rajagopalan B, Radhakrishnan N, et al. Intact giant abdominal aortic aneurysm due to Takayasu arteritis. Ann Vasc Surg 2013;27:671.e11–4.
27. Bhardwaj N1, Babu R, Behra A. Anaesthetic management of a pregnant patient with Takayasu's disease undergoing abdominal aortic aneurysm repair. Int J Obstet Anesth 2009;18:392–5.
28. Bonilla-Abadía F, Echeverri AF, Carbonell JP, et al. Multiple endovascular stent-graft implantations in a patient with aortic thoracic and abdominal aneurysms due Takayasu arteritis. Rheumatol Int 2014;34:723725.
29. Robles M, Reyes PA. Takayasu's arteritis in Mexico: a clinical review of 44 consecutive cases. Clin Exp Rheumatol 1994;12:381–8.
30. Clemente G, Hilário MO, Len C, et al. Brazilian multicenter study of 71 patients with juvenile-onset Takayasu's arteritis: clinical and angiographic features. Rev Bras Reumatol Engl Ed 2016;56:145–51.
31. Dabague J, Reyes PA. Takayasu arteritis in Mexico: a 38-year clinical perspective through literature review. Int J Cardiol 1996;54:S103–9.
32. Cañas CA, Jimenez CA, Ramirez LA, et al. Takayasu arteritis in Colombia. Int J Cardiol 1998;66:S73–9.
33. Watanabe Y, Miyata T, Tanemoto K. Current clinical features of new patients with Takayasu arteritis observed from cross-country research in Japan: age and sex specificity. Circulation 2015;132:1701–9.
34. Desbois AC, Wechsler B, Cacoub P, et al. Aortic inflammatory disease in Behçet disease. Rev Med Interne 2016;37:230–8.
35. Shen C, Li W, Zhang Y, et al. Outcomes of surgery for patients with Behcet's disease causing aortic pseudoaneurysm: a shift from open surgery to endovascular repair. Clinics 2016;71:302–10.
36. Rose AG, Dent DM. Inflammatory variant of abdominal atherosclerotic aneurysm. Arch Pathol Lab Med 1981;105:409–13.
37. Baskerville PA, Blakeney CG, Young AE, et al. The diagnosis and treatment of peri-aortic fibrosis (inflammatory aneurysm). Br J Surg 1983;70:381–5.
38. Haug ES, Skomsvoll JF, Jacobsen G, et al. Inflammatory abdominal aneurysm is associated with increased incidence of autoimmune disease. J Vasc Surg 2003;38:492–7.

39. Rasmussen TE, Hallett JE. Inflammatory aortic aneurysms: a clinical review with new perspectives in pathogenesis. Ann Surg 1997;225:155–64.
40. Stone JR, Bruneval P, Angelini A, et al. Society for Cardiovascular Pathology ''consensus statemen on surgical pathology of the aorta from the Society for Cardiovascular Pathology and the Association for European Cardiovascular Pathology: I. Inflammatory diseases''. Cardiovasc Pathol 2015;24:267–78.
41. Stella A, Gargiulo M, Faggioli GL, et al. Postoperative course of inflammatory abdominal aortic aneurysm. Ann Vasc Surg 1993;7:229–38.
42. Hechelhammer L, Wildermuth S, Lachat ML, et al. Endovascular repair of inflammatory abdominal aortic aneurysm: a retrospective analysis of CT follow-up. J Vasc Interv Radiol 2005;16:737–41.
43. Sterpetti AV, Feldhaus RJ, Schultz RD, et al. Identification of abdominal aortic aneurysm patients with different clinical features and clinical outcomes. Am J Surg 1988;156:466–9.
44. Deleeasnijder R, Daenens K, Fourneau I, et al. Endovascular repair of Inflammatory Abdominal Aortic Aneurysms with special reference to concomitant ureteric obstructuiobn. Eur J Vasc Endovasc Surg 2002;24:146–9.
45. de la Motte L, Kehlet H, Vogt K, et al. Preoperative methylprednisolone enhances recovery after endovascular repair: a randomized double-blind, placebo controlled trial. Ann Surg 2014;260:540–8.
46. van Bommel EHF, van der Veer SJ, Hendriksz TR, et al. Persistent chronic aortitis (inflammatory aneurysm) after abdominal surgical repair: systematic review of the literature. Vasc Med 2008;13:293–303.
47. Yabe T, Hamada T, Kubo T, et al. Inflammatory abdominal aortic aneurysm successfully treated with steroid therapy. J Am Coll Cardiol 2010;55:2877.
48. Hellmann DB, Grand DJ, Freishlad JA. Inflammatory abdominal aortic aneurysm. JAMA 2007;297:395–400.
49. Sterpetti AV, Lepidi S, Borrelli V, et al. Growth factors and experimental arterial grafts. J Vasc Surg 2016;64:1444–9.
50. Sterpetti AV, Cavallari N, Allegrucci P, et al. Seasonal variation in the incidence of ruptured abdominal aortic aneurysm. J R Coll Surg Edinb 1995;40:14–5.
51. Cucina A, Borrelli V, Di Carlo A, et al. Thrombin induces production of growth factors from aortic smooth muscle cells. J Surg Res 1999;82:61–6.
52. Borrelli V, Sterpetti AV, Coluccia P, et al. Bimodal concentration-dependent effect of thrombin on endothelial cell proliferation and growth factor release in culture. J Surg Res 2001;100:154–60.
53. Bozzani A, Arici V, Tavazzi G, et al. Acute Thrombosis of lower limbs arteries in the acute phase and after recovery from COVID19. Ann Surg 2021;273:e159–60.
54. Bozzani A, Arici V, Tavazzi G, et al. Acute arterial and deep venous thromboembolism in COVID-19 patients: risk factors and personalized therapy. Surgery 2020;168:987–92.