ANGIOLOGIA E CIRURGIA VASCULAR
PUBLICAÇÃO OFICIAL SPACV

REVIEW ARTICLE

Diagnosis and predictors of post-implantation syndrome following endovascular repair of aortic aneurysms – a narrative review

Tiago F. Ribeiro[a], **Rita Soares Ferreira**[a,b], **Rita Bento**[a], **Fabio Pais**[a], **Joana Cardoso**[a], **Frederico Bastos Gonçalves**[a,b,c], **Carlos Amaral**[a,b], **Maria Emília Ferreira**[a,b]

[a] Departamento Angiologia e Cirurgia Vascular, Hospital de Santa Marta, Centro Hospitalar Universitário Lisboa Central, Lisboa, Portuga; [b] NOVA Medical School|Faculdade de Ciências Médicas, NMS|FCM, Universidade Nova de Lisboa; Lisboa, Portugal; [c] Hospital CUF Tejo, Lisboa, Portugal.

Submitted: May 7, 2023; **Reviewed:** August 1, 2023; **Accepted:** September 3, 2023
Presented at: 22nd Annual Meeting of the Portuguese Society of Angiology and Vascular Surgery, 2023

ABSTRACT

INTRODUCTION: After endovascular aortic repair (EVAR), many patients develop a systemic inflammatory response called post-implantation syndrome (PIS). AAA and procedure-related characteristics have been linked with increased odds of developing this syndrome. Similarly, some short- and long-term consequences have been associated with PIS. This study aims to review the literature on the diagnosis and predictors of post-implantation after endovascular repair of aortic aneurysms.

RESULTS: A non-systematic review of the MEDLINE and Scopus databases was performed using the keywords "abdominal aortic aneurysm," "inflammation," and "endovascular techniques." No time or language limitations were imposed. Manuscripts were considered irrespective of study design. Articles of interest were analyzed, and the relevant information was organized in tables.

RESULTS: PIS is defined as a combination of constitutional symptoms, including fatigue and fever, and elevated inflammatory markers. There are several proposed diagnostic criteria, most including a combination of fever with leukocytosis and/or elevated C-reactive protein (CRP). These result in discrepant rates, as low as 2% and up to 100%. The typical evolution of this syndrome is spontaneous resolution, although pharmacologic measures for symptom relief may be needed. These symptoms often resolve within two weeks; no significant permanent complications remain. Most PIS cases will present up to the first 72 postoperative hours. Endograft material, particularly polyester-based stent grafts, has been consistently linked to increased odds of PIS, up to five-fold, compared to polytetrafluoroethylene (PTFE) grafts. Aneurysm thrombus load (both pre-existing and new-onset) has also been related to an increased odds of PIS. Bacterial translocation, contrast media, and other patient or procedure-related characteristics have not been linked to an increased risk of PIS.

CONCLUSION: PIS is a common finding after EVAR. Universal diagnostic criteria for diagnosis are required. Polyester-based stent grafts present the highest risk of developing this syndrome. Aneurysm thrombus load may also relate to this increased risk. The impact of other clinical or anatomical factors remains undetermined.

Keywords: Abdominal Aortic Aneurysms; Post-implantation syndrome.

Corresponding Author:
Tiago F. Ribeiro | ribeiro.tiago1193@gmail.com
Rua de Santa Marta, 50, 1169-024 Lisboa

Angiol Vasc Surg 2024;20(1):20-24
DOI: https://doi.org/10.48750/acv.569

INTRODUCTION

After EVAR, a significant number of patients develop a systemic inflammatory response named post-implantation syndrome (PIS). This pathology is characterized by constitutional symptoms and elevated inflammatory biomarkers in the early postoperative period.[1] AAA and procedure-related characteristics have been linked with an increased risk of developing this syndrome. Similarly, some short and long-term consequences have been associated with it.[1-7] This study aims to review the literature about diagnosis and predictors of post-implantation after endovascular repair of aortic aneurysms.

METHODS

MEDLINE and Scopus search using the terms "abdominal aortic aneurysm," "inflammation," and "endovascular techniques" was performed. No time or language limitations were imposed. Manuscripts were considered irrespective of study design. Additional articles of scientific interest for this non-systematic review were included by cross-referencing. Relevant articles were analyzed in detail, and relevant information was extracted and organized in tables.

RESULTS

Animal studies have shown a severe foreign-body response to endograft implantation. A study in which a group of sheep underwent endovascular implantation of Dacron stent grafts in the iliac arteries has shown significant peri-graft inflammatory response, vascular wall thickening, and adhesions.[8] This local response ultimately results in systemic symptoms and laboratory changes responsible for PIS.

Diagnosis:
PIS is defined as a combination of constitutional symptoms, including fatigue and fever, with elevated inflammatory markers. There are several proposed diagnostic criteria. These result in discrepant rates, as low as 2% and up to 100%.[9,10] The common finding is the inclusion of fever as the symptom/sign for diagnosis. Regarding inflammatory markers, they are more variable. Most reports have used the systemic inflammatory syndrome criterion (SIRS) with a combination of fever and leukocytosis.[1,3,4,5,6,12,13,14,15] Others have used a combination of fever coinciding with elevated C-reactive protein (CRP) levels.[2,7,16,17,18] Table 1 depicts diagnostic criteria used throughout the literature and corresponding PIS rates.

The first study reported was by Blum et al. in 1997. The authors prospectively analyzed the clinical outcomes after EVAR in 154 patients treated with polyester-covered nitinol stent grafts. Eighty-seven (56%) developed a fever (38.0–39.7°C) lasting 4–10 days. Most patients developed leukocytosis (range 9.800-29.500/μL) and CRP elevation (range 4 - 34.1 mg/dL). Diagnosis, however, was not based on specific markers.[19]

Two years later, Velazquez et al. described the use of specific markers for PIS diagnosis (leukocytosis and fever).[11] No cutoff values were reported. Within those with specified diagnostic criteria, incidence is similar (20-40%).

On the other hand, in the study by De la Motte et al.,[10] the authors performed a randomized, double-blind, placebo-controlled trial that aimed to analyze the effect of a single preoperative dose of 30 mg/kg of methylprednisolone or placebo administered 2 hours before EVAR. For diagnosis of PIS, they used SIRS criteria (presence of at least two of the following criteria: temperature > 38°C or < 36°C; leukocytes >12.000/l, <4.000/ or > 10% bands; heart rate > 90; respiratory rate > 20; PaCO2 < 32 mm Hg), except the criterion of leukocytosis. Instead of leukocytosis, the criterion was elevation of CRP to >75 mg/L. With a single preoperative dose, they obtained a reduction of PIS from 100% in the placebo group to 27% in the treatment group. The 100% PIS rate in the placebo group is due to the addition of other clinical criteria that are common findings in EVAR patients, which are often associated with other cardiopulmonary diseases.

Many other studies did not specify diagnostic criteria.[9,19-22] In those with pre-specified criteria, there is a significant degree of variance in incidence. A universal consensual definition of this syndrome is urgently needed.

Clinical manifestations:
Symptoms associated with PIS are described above. The typical evolution is spontaneous resolution, although pharmacologic measures for symptom relief may be needed. These symptoms often resolve within two weeks; no significant permanent complications remain. However, kidney injury and impaired quality of life may ensue occasionally.[3,20] Conversely, a reduced risk for type 2 endoleak rates has been reported.[16,24] The impact on cardiovascular events remains to be determined.[16,25] The diagnosis will be frequently suspected in those with fever and inflammation and no infection source. Leukocytosis usually will peak on the first postoperative day, and CRP and temperature will peak on the second postoperative day. Most PIS cases will present up to the first 72 postoperative hours. A higher suspicion for infection should occur if inflammation and fever occur after this time.

Table 1. Incidence of post-implant syndrome and utilized diagnostic criteria of studies included in the review.

Authors	Year	Design	N	Fever	Leucocyte	CRP	PIS Rate
Unclear Definition							
Blum et al.[19]	1997	Prospective	154	N/S	N/S	N/S	56%
Chang et al.[20]	2014	Prospective	38	N/S	N/S	N/S	47%
Georgiadis et al.[21]	2011	Prospective	77	N/S	N/S	N/S	36.4%
Mazzacaro et al.[22]	2016	Retrospective	10	N/S	N/S	N/S	30%
Melissano et al.[9]	2015	Retrospective	42	N/S	N/S	N/S	2%
Leukocytosis and Fever							
Arnaoutoglou et al.[1]	2010	Prospective	162	>38°C	>12.000/mL	N/A	30.2%
Dosluoglou et al.[12]	2014	Retrospective	79	>37.8°C	>12.000/mL	N/A	23%
Nano et al.[3]	2014	Retrospective	118	>38°C	>12.000/mL	N/A	20.3%
Kakisis et al.[14]	2014	Retrospective	87	>38°C	>12.000/mL	N/A	39%
Arnaoutoglou et al.[5]	2014	Prospective	214	>38°C	>12.000/mL	N/A	36%
Sartipy et al.[13]	2015	Prospective	45	>38°C	>12.000/mL	N/A	38%
Ferreira et al.[15]	2015	Retrospective	42	>38°C	>12.000/mL	N/A	21.2%
Kwon et al.[4]	2016	Retrospective	204	>38°C	>12.000/mL	N/A	31.4%
Arnaoutoglou et al.[6]	2016	Prospective	182	>38°C	>12.000/mL	N/A	35.7%
SIRS criteria with CRP instead of leucocytosis							
De la Motte et al.[10]	2014	RCT	76 (placebo)	>38°C	N/A	>75mg/L	100%
Fever, Leucocytosis, and CRP							
Chatzelas et al.[23]	2022	Retrospective	191	>38°C	□12.000/mL	>100mg/L	21.5%
Fever and CRP							
Voûte et al[2]	2012	Retrospective	149	>38°C	N/A	>100mg/L	38.9%
Gorla et al.[17]	2015	Retrospective	133	>38°C	N/A	>75mg/L	15.8%
Soares-Ferreira et al.[7]	2018	Retrospective	205	>38°C	N/A	>75mg/L	19%
Soares-Ferreira et al.[16]	2021	Retrospective	149	>38°C	N/A	>100mg/L	39%
Ribeiro et al.[18]	2023	Retrospective	253	>38°C	N/A	>75mg/L	23.7%

SIRS: systemic inflammatory response syndrome; **CRP:** C-reactive protein; **PIS:** post-implantation syndrome; **N/S:** not specified; **N/A:** not applicable.
Adapted and updated from Soares Ferreira R, Bastos Gonçalves F. Post-implantation syndrome after endovascular aneurysm repair. In: Koncar I, editor. Abdominal Aortic Aneurysm – From Basic Research to Clinical Practice. IntechOpen 2018, with permission from the authors.

Preditors of PIS:
The pathophysiology and etiology of PIS are partially clear. The mechanisms behind this condition seem multifactorial and mainly involve aneurysms and procedure-related characteristics.

1 – Stent graft material: Stent graft material is probably the most reliable predictor of PIS after EVAR. Multiple studies have compared the incidence of PIS and inflammatory response according to the implanted graft composition (polytetrafluoroethylene - PTFE vs Dacron), and the use of polyester-based grafts has been recurrently related to an increased odd of PIS, up to five-fold.[1-4,14] Vôute et al., in a retrospective analysis of 149 patients (82 polyester and 67 PTFE grafts), found a PIS rate of 56.1% vs 17.9%, P<.001 in polyester vs PTFE grafts, respectively. Almost every PIS case occurred during the first three postoperative days. The inflammatory response (temperature and CRP)

presented a significantly higher magnitude after Dacron graft implantation, highest temperature rise (1.6 vs. 0.9°C, P<.001) and CRP level rise (154.8 vs. 38.0 mg/L, P<.001). Finally, a multivariable logistic regression model was constructed, and a woven polyester graft was the sole predictor of PIS (HR 5.58, 95% CI 1.60-19.42, P=.007).[2] Kakisis et al., in a retrospective analysis of 87 patients, found similar results when testing for risk factors for PIS.[14] Chatzelas et al., in a prospective analysis of retrospectively collected data (191 patients), found that polyester graft composition was the single factor significantly associated with an increased PIS rate (HR 2.6, P=.043).[23]

Contrary to these, other studies could not confirm a difference in PIS rates when stratifying for stent graft composition.[26-28] Gerasimidis et al. prospectively compared the degree of inflammatory response between endovascular aneurysm repair with polyester (12 patients) and ePTFE devices (10 patients). One patient in each group had PIS, according to SIRS criteria. Three patients in the polyester group had a fever (>38°C), and just one in the PTFE group ($p < 0.005$). There were no statistically significant differences between groups for all endpoints, possibly due to the sample size.[26] In a cohort reported by Sartipy et al., there were significant differences between the two types of graft material concerning fever and CRP, but there were no significant differences in the number of PIS events. It could also be related to sample size, with 32 patients treated with polyester grafts but only 13 with ePTFE grafts. They performed a sensitivity analysis showing that the results would have reached significance if three more patients in the polyester group had developed PIS (or none instead of one patient in the PTFE group).[13] Lastly, Moulakakis et al. assessed the inflammatory and renal response after TEVAR, and no significant differences in inflammatory response between polyester and PTFE groups were noted. They attributed that to the small number of patients implanted with ePTFE stent grafts in their trial.[29]

2- Thrombus: The hypothesis that the amount of thrombus (pre-existing or new-onset) within the aneurysm sac could be related to PIS derives from the finding that mural thrombus of an aortic aneurysm contains high levels of interleukin-6.[27] It is hypothesized that thrombus manipulation during the procedure would be able to release this inflammatory mediator to the systemic circulation, contributing to the postoperative inflammatory response. Also, newly formed thrombus after aneurysm exclusion could be able to increase this response, ultimately resulting in PIS. Nano et al. reported an association between preoperative thrombus thickness and PIS with EVAR using the Anaconda® stent graft (P=0.001).[3] Kakisis et al. (87 patients) found a mean volume of chronic mural thrombus of 63.7 ± 73.6 mL, and a mean volume of new-onset thrombus was 55.1 ± 74.8 mL. A statistically significant correlation was found between the volume of new-onset thrombus and peak postoperative temperature (r=.379; P<0.01) as well as the increase in CRP level (r=.533; P<0.001) and IL-6 level (r=.380; P=0.01). On the contrary, the volume of chronic mural thrombus was not found to affect any parameter of PIS significantly.[14] The volume of chronic mural thrombus, as well as the volume of new-onset thrombus, was calculated using imaging software. The closed polygon tool was used to outline the perimeter of the aneurysm sac on every transverse slice of the CTA, starting from the level of the renal arteries and ending at the aortic bifurcation. With this input, the software automatically calculated the aneurysm sac volume. In 2012, Voute et al., other than studying the influence of graft material, aimed to analyze the association between new-onset thrombus and the inflammatory response. They report no significant association between new-onset thrombus and temperature or C-reactive protein elevation.[2] Arnaoutoglou et al., in a prospective evaluation of 214 EVAR patients, found no significant association between preoperative and newly formed thrombus and increased inflammatory response.5 Chatzelas et al., in a cohort of 191 patients, found that new-onset thrombus did not significantly relate to an increased chance of developing PIS (OR 1.29, P=0.10).[23] Literature reports are variable regarding this specific feature, and although most discuss the association between mural thrombus (chronic or new-onset) and PIS, the real impact remains to be determined. Finally, as polyester stent grafts are associated with increased intraprosthetic mural thrombus, this raises the question of whether stent graft material does not act as a significant confounder of new-onset thrombus.[27]

3 – Bacterial translocation: Transient bacterial translocation is another potential factor impacting the chance of developing PIS. This is thought to be due to the occlusion of a previously patent inferior mesenteric artery (IMA). The IMA is inevitably occluded during EVAR procedures, except in highly selected cases. This occlusion could result in transient colonic ischemia with the consequent risk of bacterial translocation, significantly contributing to PIS occurrence. Kakisis et al. analyzed the association between the patency of the IMA and the postoperative temperature and inflammatory markers. No significant correlation was found.[14] Akin et al. performed a prospective single-center trial involving 40 patients. They aimed to study the effects of antibiotic therapy in PIS after thoracic aortic stent placement. Two groups were created, one receiving cefuroxime 20mg/kg tid during the first 24h postoperative hours and the other receiving the same dosing scheme during the first seven days. There were no significant differences in clinical or laboratory findings between treatment strategies. Therefore, the hypothesis of bacterial translocation as a cause for PIS seems remote, and there is no evidence to support it.

4 – Contrast media: Videm et al. suggested that the contrast medium iohexol provokes neutrophil degranulation, which is greatly enhanced when combined with stent graft material, contributing to PIS occurrence.[30] Other studies analyzed inflammatory response after endovascular aneurysm repair but did not find any correlation between contrast use or dosage and PIS parameters.[1,3,14,29] As such, this theory remains to be demonstrated.

5 – Other factors: The type of procedure performed can be independently associated with different chances of PIS. Using

an additional iliac branch device or fenestrated graft for the visceral aorta results in additional procedural steps and stent graft material, increased branch manipulation, more lengthy operations, and more associated complications. These criteria have been scarcely studied, and fenestrated branched aortic grafts are frequent exclusion criteria throughout the literature. Ferreira et al., in a retrospective analysis of 52 patients, found a 0% PIS incidence after f/bEVAR. However, just four patients were included.[15] In addition, Ribeiro et al. found an increased rate of PIS after complex EVAR when compared to standard infrarenal repair (34.1% vs. 18.2%, P=0.005; aOR 2.833, P=0.009).[18] The impact of previous exposure to an aortic stent graft, particularly in cases of secondary interventions, has not been addressed. Other factors, such as age, gender, aneurysm diameter, length of operation, blood loss or blood product requirements, COPD, or heart disease, have been explored; however, no significant associations were noted.[1,3,13,20]

CONCLUSION

PIS is a common finding after EVAR, but universal diagnostic criteria are required. Polyester-based stent grafts present the highest risk of developing this syndrome, and Aneurysm thrombus load may also be associated with an increased incidence. Other specific predictors in higher-complexity procedures are currently unknown.

Acknowledgments: None

Conflicts of interest: None

Funding None

REFERENCES

1. Arnaoutoglou E, Papas N, Milionis H, Kouvelos G, Koulouras V, Matsagkas MI. Post-implantation syndrome after endovascular repair of aortic aneurysms: Need for postdischarge surveillance. Interact Cardiovasc Thorac Surg. 2010; 11:449-54

2. Voûte MT, Bastos Gonçalves FM, Van De Luijtgaarden KM, Klein Nulent CGA, Hoeks SE, Stolker RJ et al. Stent graft composition plays a material role in the post-implantation syndrome. J Vasc Surg. 2012; 56:1503–9.

3. Nano G, Occhiuto MT, Stegher S, Malacrida G, Cova M, Righini P et al. Post-implantation syndrome after endovascular aortic repair using the anacondaTM endograft. Ann Vasc Surg. 2014; 28:1409–15.

4. Kwon H, Ko GY, Kim MJ, Han Y, Noh M, Kwon TW, et al. Effects of post-implantation systemic inflammatory response on long-term clinical outcomes after endovascular aneurysm repair of an abdominal aortic aneurysm. Medicine (Baltimore). 2016; 95:4532

5. Arnaoutoglou E, Kouvelos G, Papa N, Kallinteri A, Milionis H, Koulouras V, et al. Prospective Evaluation of Post-implantation Inflammatory Response After EVAR for AAA : Influence on Patients' 30 Day Outcome. Eur J Vasc Endovasc Surg. 2015; 49:175-83

6. Arnaoutoglou E, Kouvelos G, Papa N, Gartzonika K, Milionis H, Koulouras V et al. Prospective evaluation of post-implantation syndrome evolution on patient outcomes after endovascular aneurysm repair for abdominal aortic aneurysm. J Vasc Surg. 2016; 63:1248–55

7. Soares Ferreira R, Bastos Gonçalves F, Abreu R, Camacho N, Catarino J, Correia R et al. Post-implantation syndrome after EVAR. Angiol Cir Vasc. 2018; 14:176-81

8. Schürmann K, Vorwerk D, Bücker A, Neuerburg J, Klosterhalfen BMG. Perigraft inflammation due to Dacron-covered stent grafts in sheep iliac arteries: Correlation of MR imaging and histopathologic findings. Radiology. 1997; 204:757-63.

9. Melissano G, Tshomba Y, Rinaldi E, Chiesa R. Initial clinical experience with a new low-profile thoracic endograft. Journal of Vascular Surgery. 2015; 62:336-42.

10. De La Motte L, Kehlet H, Vogt K, Nielsen CH, Groenvall JB, Nielsen HB, et al. Preoperative methylprednisolone enhances recovery after endovascular aortic repair: A randomized, double-blind, placebo-controlled clinical trial. Annals of Surgery. 2014; 260:540-9.

11. Velázquez OC, Carpenter JP, Baum RA, Barker CF, Golden M, Criado F et al. Perigraft air, fever, and leukocytosis after endovascular repair of abdominal aortic aneurysms. American Journal of Surgery. 1999; 178:185-9

12. Dosluoglu HH, Lall P, Blochle R, Harris LM, Dryjski ML. Ambulatory percutaneous endovascular abdominal aortic aneurysm repair. Journal of Vascular Surgery. 2014; 59:58-64

13. Sartipy F, Lindström D, Gillgren P, Ternhag A. The role of procalcitonin in post-implantation syndrome after EVAR: A pilot study. Annals of Vascular Surgery. 2014; 28:866-73;

14. Kakisis JD, Moulakakis KG, Antonopoulos CN, Mylonas SN, Giannakopoulos TG, Sfyroeras GS, et al. Volume of new-onset thrombus is associated with the development of post-implantation syndrome after endovascular aneurysm repair. Journal of Vascular Surgery. 2014; 60: 1140-5.

15. Ferreira V, Machado R, Martins J, Loureiro L, Loureiro T, Borges L et al. Post-implantation syndrome - retrospective analysis of 52 patients. Angiol Cir Vasc 2015; 11:204-8

16. Soares Ferreira R, Oliveira-Pinto J, Ultee K, Voûte MT, Oliveira NFG, Hoeks S et al. Long Term Outcomes of Post-Implantation Syndrome After Endovascular Aneurysm Repair. Eur J Vasc Endovasc Surg. 2021; 62:561-8

17. Gorla R, Erbel R, Kahlert P, Tsagakis K, Jakob H, Mahabadi AA, et al. Clinical features and prognostic value of stent graft-induced post-implantation syndrome after thoracic endovascular aortic repair in patients with type B acute aortic syndromes. Eur J Cardiothorac Surg. 2016; 49:1239-12

18. Ribeiro TF, Soares Ferreira R, Amaral C, Ferreira ME, Bastos Gonçalves F. Post-Implantation Syndrome Incidence is Higher After Complex Endovascular Aortic Procedures Than After Standard Infrarenal Repair. Eur J Vasc Endovasc Surg 2023; 66:804-12

19. Blum U, Voshage G, Lammer J, Beyersdorf F, Töllner D, Kretschmer G et al. Endoluminal stent–grafts for Infrarenal abdominal aortic aneurysms. New Eng J Med. 1997;336:13-20

20. Chang CK, Chuter TAM, Niemann CU, Shlipak MG, Cohen MJ, Reilly LM et al. Systemic inflammation, coagulopathy, and acute renal insufficiency following endovascular thoracoabdominal aortic aneurysm repair. J Vasc Surg. 2009;49:1140-6

21. Georgiadis GS, Trellopoulos G, Antoniou GA, Gallis K, Nikolopoulos ES, Kapoulas KC, et al. Early results of the Endurant endograft system in patients with friendly and hostile infrarenal abdominal aortic aneurysm anatomy. J Vasc Surg 2011; 54:616-27

22. Mazzaccaro D, Occhiuto MT, Stegher S, Righini P, Malacrida G, Nano G. Tips about the Cordis INCRAFT Endograft. Ann Vasc Surg. 2016; 30:205-10

23. Chatzelas DA, Pitoulias AG, Telakis ZC, Kalogirou TE, Tachtsi MD, Christopoulos DC et al. Incidence and risk factors of post-implantation syndrome after elective endovascular aortic aneurysm repair. Int Angiol 2022; 41:196-204.

24. Kwon H, Ko G-Y, Kim M-J, Han Y, Noh M, Kwon T-W, et al. Effects of post-implantation systemic inflammatory response on long-term clinical outcomes after endovascular aneurysm repair of an abdominal aortic aneurysm. Medicine (Baltimore). 2016; 95:4532

25. Sousa J, Vilares AT. Post-implantation Syndrome Is Not Associated with Myocardial Injury after Noncardiac Surgery after Endovascular Aneurysm Repair. Ann Vasc Surg 2020. 68:275-82

26. Soares Ferreira R, Bastos Gonçalves F. Post-implantation syndrome after endovascular aneurysm repair. In: Koncar I, editor. Abdominal Aortic Aneurysm – From Basic Research to Clinical Practice. IntechOpen; 2018.

27. Swartbol P, Truedsson LNL. Adverse reactions during endovascular treatment of aortic aneurysms may be triggered by interleukin 6 release from the thrombotic content. J Vasc Surg. 1998; 28:664-8

28. Perini P, Bianchini Massoni C, Azzarone M, Ucci A, Rossi G, Gallitto E et al. Significance and Risk Factors for Intraprosthetic Mural Thrombus in Abdominal Aortic Endografts: A Systematic Review and Meta-analysis. Ann Vasc Surg 2018. 53:234-42

29. Moulakakis KG, Sfyroeras GS, Papapetrou A, Antonopoulos CN, Mantas G, Kakisis J, et al. Inflammatory response and renal function following endovascular repair of the descending thoracic aorta. Journal of Endovascular Therapy. 2015;22:201-6

30. Videm V, Ødegård AMH. Iohexol-induced neutrophil myeloperoxidase release and activation upon contact with vascular stent graft material: A mechanism contributing to the post-implantation syndrome. Journal of Endovascular Therapy. 2003; 10:958-67