Chapter 7

Treatment of the Progressive Endoleak Type 2 After EVAR

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Additional information is available at the end of the chapter

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Abstract

An endoleak type 2 (EL2) is a relatively frequent event after an EVAR but 30–35% of EL2 can become progressive, which can cause a loss in the important sealing zone of the stent graft. Diagnosis is made by three-phase CT angiogram or by contrast-enhanced duplex scan. EL2 should be treated if the aortic sac grows more than 5 mm in 6 months time. The first suitable treatment is the endovascular approach with embolization of the inferior mesenteric artery (IMA) or lumbar arteries. Paravertebral puncture, under CT navigation to embolize the lumbar artery or a part of the aortic sac with the EL2, is another alternative. If the endovascular treatment is not successful in 2–3 times, we should consider a surgical approach. The operative approach can be a laparoscopic or an open operation: the laparoscopic approach allows us to clip the IMA and lumbar arteries. The open surgery involves laparotomy, ligation of the IMA, and endoaneurysmorrhaphy (suture of lumbar artery origins from inside) and then the suture of the aortic sac tightly around the stent graft in situ. The aortic occlusion balloon should be inserted below the renal arteries prior to open surgery. The surgical procedures have good outcomes and should be considered when the endovascular treatment is unsuccessful.

Keywords: endoleak type 2, AAA, EVAR, sac embolization, post-EVAR intervention

1. Introduction

Endoleak type 2 (EL2) is described as a refilling of the aortic sac via branches such as lumbar arteries (LAs), inferior mesenteric artery (IMA), median sacral artery, or accessory renal arteries after endovascular aneurysm repair (EVAR). Endoleak through the internal iliac artery should be classified as endoleak type 1c (**Figure 1**). We could imagine the EL2 as a type of

a false aneurysm in the thrombus around the stent-graft (SG) where we have inflow and outflow via sac branches.

We can distinguish certain types of EL2s:

Early—occurs within 30 days after EVAR

Late—occurs within 12 months after EVAR

Persistent—long lasting for more than 6 months

The progressive type of the EL2 causes the aortic sac to grow ≥5 mm/6 months with a risk of other complications. It plays an important role in the pressure gradient between the sac branches (mostly IMA, LAs, etc.), the position of the SG, and the pulsating volume between visceral branches through the sac space. It forms a cavity that clots around the SG, which has blood flow and causes EL2 (**Figure 1**) [1–4]. We have to take into account arterial hypertension and atherosclerotic changes of the aortic branches; their peripheral resistance is also an important factor. Visceral branches of abdominal aorta have low peripheral resistance physiologically, but due to atherosclerotic changes (calcifications) they lose flexibility and the resistance can be increased. Due to the pressure gradient between LAs and the IMA with artery wall calcification, EL2 can be persistent for a long time. Low flow is difficult to detect,

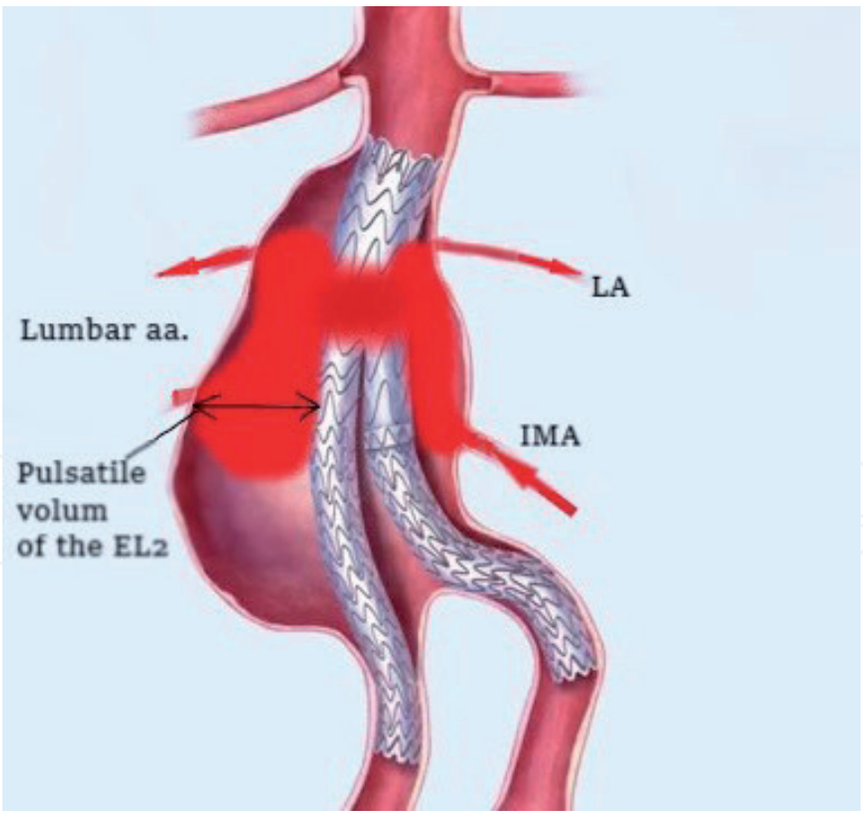

Figure 1. Example of the endoleak type 2 (EL2) flowing into a sac and image of the pulsatile volume behind/around the stent-graft (SG) with inferior mesenteric artery inflow (red arrow) and lumbar artery outflow. The black arrow shows the distance between the SG and the sac wall where part of the SG can work as a piston to pump the blood as a pulsatile volume and keep the EL2 persistent and progressive.

therefore it is important to provide a precise three-phase computed tomography angiogram (CTA). An alternative investigation is a contrast-enhanced Duplex scan (CEDS). It is well known that EL2 is a relatively frequent finding after EVAR in 25–30% of cases [5–11], but it can be resolved spontaneously (80%) [6, 9–13]. It can be persistent for a longer period of time without further aortic sac dilatation, but when progressive growth is accompanied by increasing diameter of an aneurysm, then the finding requires immediate treatment to avoid severe complications.

2. Diagnosis

We can obtain initial information regarding EL2 directly from the final angiogram promptly after EVAR; this is routine and it can show clear initial evidence of EL2. Thanks to regular ultrasound monitoring we can pick up enlargement of the sac diameter or detect flow in the sac by Doppler ultrasound scan (**Figure 2**). Diagnosis is mostly confirmed by three-phase CTA (arterial, venous, and late venous phases). An ideal alternative to providing a dynamic scan is CEDS (**Figure 3**) [14, 15]. If aortic sac growth is slow we can use an aortic angiogram to confirm the endoleak and at the same time we can use this approach for endovascular treatment. A new method of endoleak diagnosis could be 4D magnetic resonance but this technique is not a routine method at present [16]. A CTA with good arterial and venous phases is the gold standard (two- or three-phase CTA) for diagnosis, and together with software projections can provide an image for potential treatment. Dynamic feeling of the aneurysmal sac can confirm the importance of a solution and explain the progressivity of the endoleak. Visualization of the sac collaterals and connection with the superior mesenteric artery (SMA), renal arteries, or other important sac arteries can be the best option for endovascular treatment to reach the branch for embolization. Not every EL2 is indicated for intervention.

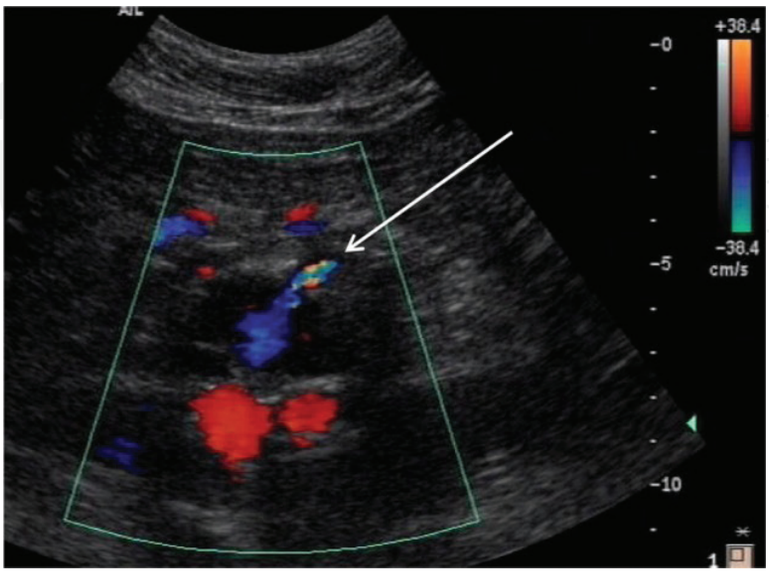

Figure 2. Duplex scan with color Doppler with endoleak type 2 via inferior mesenteric artery, marked by arrow.

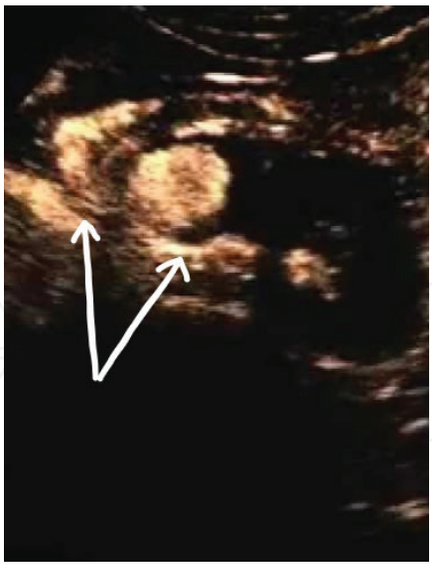

Figure 3. Contrast enhanced Duplex scan of endoleak type 2 via lumbar arteries, marked by arrows.

Characteristics, especially progressivity (growth ≥0.5 cm/6 months, ≥1 cm/1 year), play an important role in the decision to treat EL2 by intervention to stop its progress and avoid further complications.

3. Prevention of EL2

Intra-procedural embolization of IMA, LAs, and other sac branches prior to proper EVAR is necessary if their diameters are more than 2 mm [8, 17, 18]. Embolization of the accessory renal artery can inflict regional renal necrosis and a hypertension episode, therefore it must be evaluated individually to minimize patient risk. An individual approach has to involve considerations of age, main organs, functions, and reserves together with comorbidity. The best type of SG is an important factor as well (it should minimize inflow into the sac through branches and reach the maximum of the sealing zone) to decrease EL2 formation as a sac branches flow connection. The endovascular aneurysm sealing technique [19] is an option to reduce flow into the aneurysmal sac via visceral branches. The technique used to fill the sac with a polymer can reduce backflow from the sac branches and their communication within the sac can decrease the potential formation of EL2. This technique can have side effects regarding sac and neck dilatation, and expansion in the sac can lead to misplacement of the SG and further complications [20]. Aneurysm sac embolization [21, 22] during the EVAR procedure using coils and glue can decrease the rise of endoleak, reduce sac volume, and improve its shrinkage. This method was described with a lower rate of reintervention due to EL2, but statistically, there was no clear outcome to confirm this technique as the best solution for prevention. From a clinical perspective, selective embolization of the sac branches with greater lumen diameters (≥2–3 mm) is more efficient to decrease the potential risk of EL2 formation. We can use fewer coils and glue together with lower irradiation time with our comparison with the sac embolization only. It is helpful to use so-called 3D fusion to navigate cannulation of the sac branches.

Stopping backflow and outflow via sac branches (to break communications between the sac branches, namely IMA and LAs) becomes more efficient when we compare sac embolization with a potential risk of formation of small roads between coils and glue mass as a restoration of communication between the sac branches. The coils and glue in the sac cause further artifacts on CTA, and to visualize the endoleaks may be difficult and involve visibility of the treated EL2. High blood pressure treatment [23], statins [24], and regulation of anticoagulation therapy [25] are also important parts of prevention with the possibility of decreasing the risk of EL2 development. The decreased pressure gradient between sac branches as described means that sac communication can prevent EL2 and help to reduce EL2 flow. Statins can stabilize sclerotic plaques and the arterial wall, which leads to rapid shrinkage of the sac after EVAR. Reduction of the space around the SG can minimize the flow between sac branches and improve contact between the SG and the sac, the so-called sealing zone.

4. Treatment

EL2 should be treated if the aortic sac grows more than 5 mm in 6 months. Conservative treatment is the first method for EL2 treatment, which is occluded spontaneously in 80–90% of cases [26]. Conservative treatment is not the only method to watch and wait but require active treatment of arterial hypertension to decrease pressure gradient into the sac and sac branches. Statins can support the stabilization process of atherosclerotic plaque and avoid further sac dilatation [1, 27, 28]. Surveillance by ultrasound to measure the aneurysm diameter is an important method of dynamic monitoring when considering a plan for interventional treatment if conservative treatment is unsuccessful and the EL2 is becoming progressive.

4.1. Endovascular

EL2 can be persistent over a long time, with a stable aneurysm diameter, but in 3–5% of cases it can become progressive with dilatation of the aortic sac, which can cause loss of the important sealing zone of the SG. This process can lead to the development of endoleak type 1 and the higher risk of aneurysmal rupture. Enlargement of the sac size can be one of the important signs. The first choice for interventional treatment is the endovascular approach with embolization of the IMA or LAs. CTA can show a good connection between the SMA and IMA and is a suitable approach to reach the IMA through a connection with the SMA and to proceed with coil insertion [12]. Sometimes there can be a good connection into another sac branch together with the LA. Embolization of the IMA can be successful but the EL2 can remain due to another patent LA, which can continue with the outflow instead of the IMA. We could also use a technique to reach the sac by a transperitoneal approach [29]. Risks of endovascular treatment are the dissection of the important artery (SMA, renal artery, etc.) and embolization into SMA/IMA branches with intestinal ischemia. If there is an early and large EL2, which requires rapid solution, we can try to reach the sac by the endovascular technique behind the SG because the SG is not fully adherent (the connection between the SG and the arterial wall is not rigid). This way provides an embolization of LAs or other important branches directly from the sac, respectively from the pulsating volume in the thrombus around the SG. We

can use this technique as well if there is a combination of EL2 with endoleak 1b (distal end of the SG endoleak) or 3b (the SG limbs are disconnected) to complete the endoleak treatment. Another approach to endovascular treatment can be a paravertebral puncture of the aneurysm sac under CT navigation to provide embolization of a biological/histoacryl glue alone or with a combination of coils to embolize the LA origins and the part of the aortic sac with the endoleak flow where the main route of endoleak refilling can be halted [30]. We can use a peritoneal approach to reach the sac and provide the embolization [31]. Consistency and adherence of the glue are important characteristics, which play a role in the outcome and ensure the right place for installation. The disadvantage is the rigidity and relatively fast adherence of the glue, which is contrary to our aim of filling the whole volume of the EL2 around the SG or reducing the flow as much as possible, which leads to spontaneous occlusion. The post-procedure portion of the glue in the sac causes certain artifacts in the CTA, which could cover small EL2s. Gelatin foam and alcohol as sclerosing agents are not often used due to their reduced efficiency and potential risk of complications (namely, peripheral embolization). The main risk is infection of the SG (a severe life-threatening complication) and bleeding from the sac area, therefore constant repetition of this technique is not recommended. If endovascular treatment is not successful after two to three attempts we should consider a surgical approach. Indications for operation must be considered precisely due to important patient background, therefore pre-assessment involving function tests such as spirometry and stress ECHO together with Duplex scan of carotid arteries have become standard procedures before any open or laparoscopic intervention.

4.2. Laparoscopic treatment

The operative approach can be a laparoscopic or open operation. The laparoscopic approach is indicated for patients where the pneumoperitoneum is not contraindicated. This approach allows us to clip the IMA close to the sac and an initial CTA should guide us to the reachable segment of the IMA to avoid duodenum injury. Thanks to a camera (angulated optic) the laparoscopic procedure allows very good visibility of the LAs for clipping [32, 33]. The left side of the aorta and aneurysm is easily approached but the right side is difficult to access due to the tight, adherent position of the inferior vena cava. The retroperitoneal approach with the same endoscopic technique is a very suitable alternative, which requires a good overview from each side to reach the LAs for clipping; however, for the IMA it is important to have a clear standard laparoscopic approach to combine these techniques. The retroperitoneal endoscopic approach is the same as thelumbar sympathectomy approach. The laparoscopic approach is relatively safe. When an approach from the right side of the aorta is too risky, then we can clip and stop the flow in one of the side LAs to stop the EL2 because we can reduce the inflow or outflow with good results. We have to check the efficiency of our procedure by CEDS or by a table angiogram. With the results, we can evaluate our efficiency at the same time and conclude if our procedure has been successful. It is the opinion of the author that CEDS is the best combination to detect the EL2 preoperatively and guide us to find the main blood vessels to fix it. Only deflation and inflation of the operation space is time consuming, especially when the scan has to be repeated a few times. The main risk to endoscopic treatment involves peritoneal and retroperitoneal bleeding but the incidence is small [32, 33].

4.3. Open transperitoneal approach

Open surgery involves laparotomy and ligation of the IMA (**Figure 4**). The main approach is transperitoneal, which is exposure of the retroperitoneal space and aortic sac involving the neck of an aneurysm and aortic bifurcation. The important part is preparing the small space from both sides of the neck for an emergency aortic clamp or compression above the planned sac incision. A very good alternative is to have an occlusion balloon in the aorta close to the origin of the renal arteries to be ready for the eventual leak from the neck area. The main step of the procedure is an incision of the aortic sac and endoaneurysmorrhaphy (suture of LA origins from inside the sac), then the suture of the aortic sac is tightened (in two layers is a good option) around the SG in situ [34, 35] (**Figures 5** and **6**). It is very important to protect the SG against a misplacement. Protection involves keeping the distance from the neck area at around 5 cm below and not opening an aneurysm too close to the neck because there is a risk of losing a proximal sealing zone. To protect the position of the SG it should be kept very close to the dorsal wall, respectively the spine column when we are removing the thrombus around the SG. The moderate pressure applied to the SG against the dorsal wall will help keep the SG in position and decrease back bleed from the LAs. The aortic occlusion balloon, which

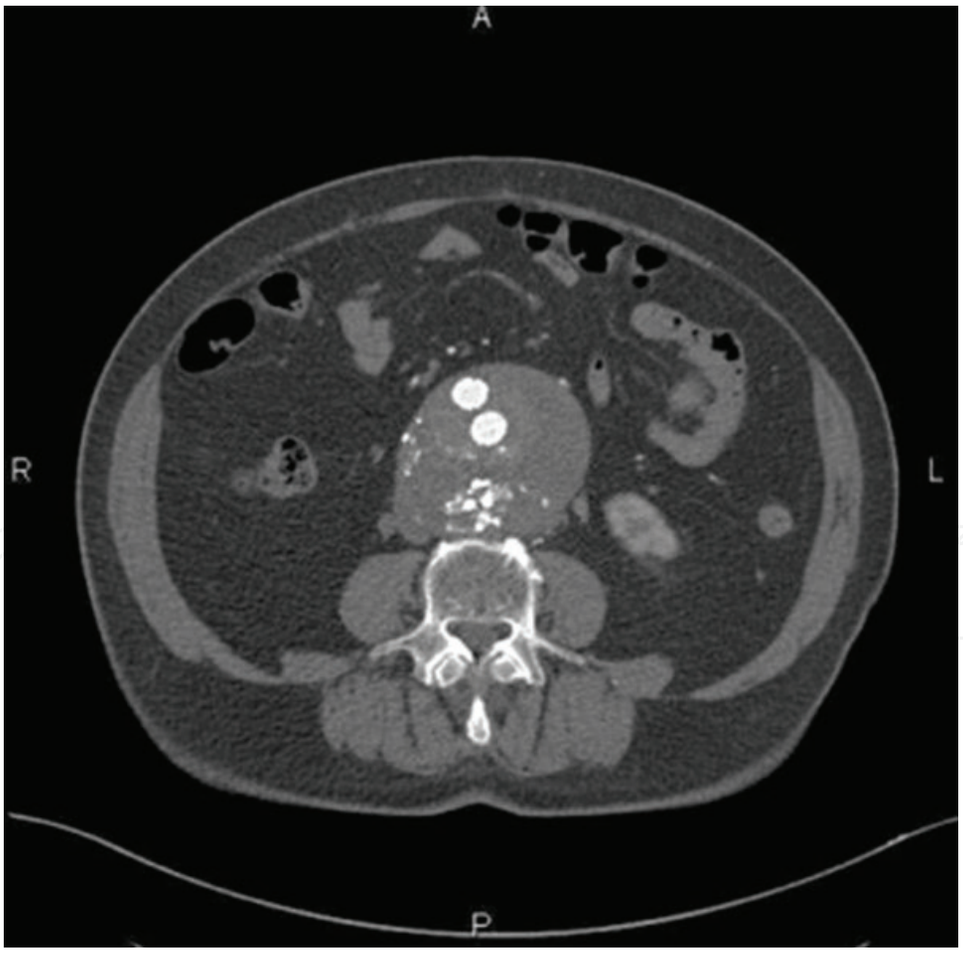

Figure 4. Computed tomography angiogram of the persistent endoleak type 2 via lumbar arteries after paravertebral puncture of the sac and unsuccessful glue instillation/embolization.

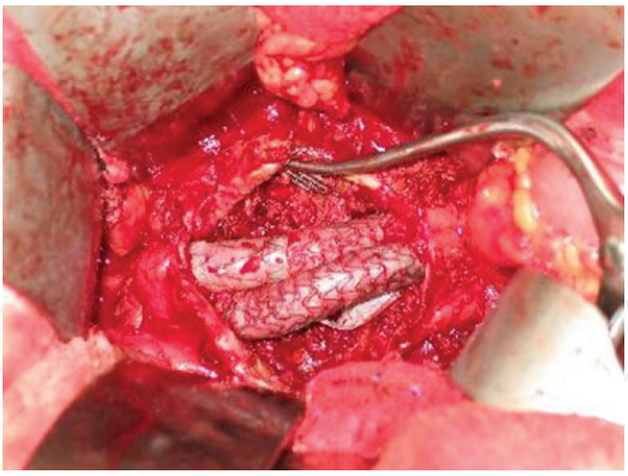

Figure 5. Open sac view with stent-graft in situ after thrombus removal and suture of lumbar artery origins.

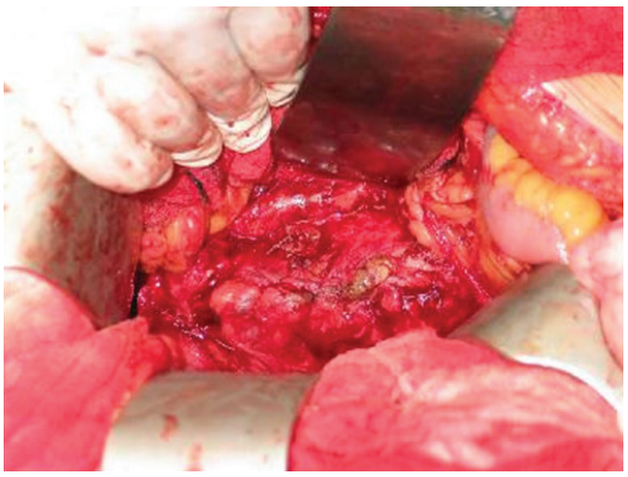

Figure 6. Dual layer suture of the aortic sac after endoaneurysmorrhaphy.

should be inserted below the renal arteries prior to open surgery to provide aortic occlusion in case of bleeding, is a better option than aortic clamping of the neck area with a risk of aortic wall injury. In case of bleeding it is important to suture the SG in position and provide a package by the aortic sac rather than to explant the SG and replace the whole SG. This type of the operation has severe complications, namely high lethality for patients (initially not suitable for any open abdominal aortic aneurysm (AAA) repair primarily) [30]. Postoperative high dependency unit/intensive care unit monitoring is important to check blood pressure but mostly uneventful laparoscopic/open procedures are well tolerated because blood loss is minimal and blood transfusion is not required. Fast recovery is due to an operation without any aortic clamp. Mostly, the patient can be discharged within 2–5 days. There is a clear advantage of laparoscopy versus laparotomy because the minimally invasive approach does not cause paralytic ileus and small wounds are well tolerated, therefore to discharge a patient within 2 days is manageable without high risks. To check the efficiency of surgical treatment by CTA is recommended within 7–10 days and thereafter standard post-EVAR monitoring can be performed (**Figure 7**). We should wait for 1–3 months for complete occlusion of the EL2,

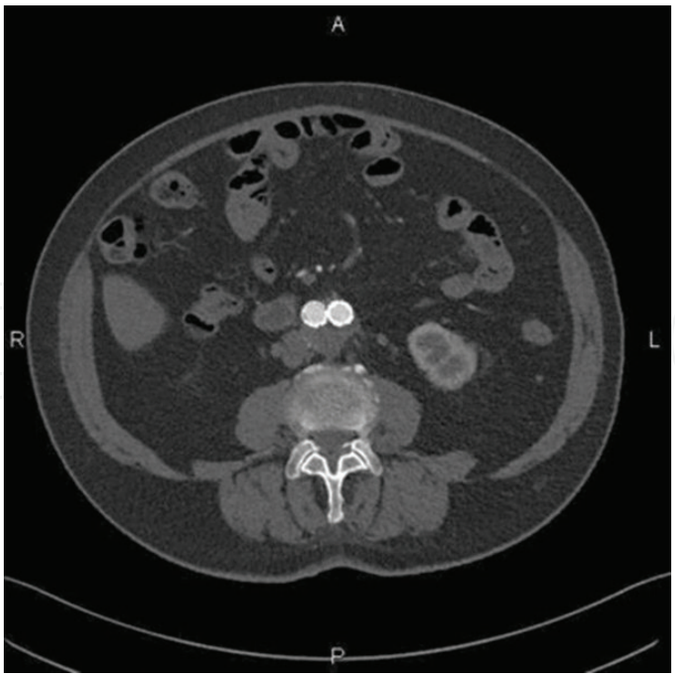

Figure 7. Computed tomography angiogram after endoaneurysmorrhaphy without endoleak type 2 and the stent-graft tightly covered by the sutured sac in two layers.

especially when stopping only one side LAs together with IMA flow. Shrinkage of the sac can be clearly visible later on within a few months. CEDS is a good choice to reduce the number of CTAs and contrast solutions.

5. Discussion

EL2 may be a relatively passive diagnosis, and may be active only for a short period of time, but if there is significant growth, then it will become a potential problem with risks such as rupture. It is well known that the risk of rupture is relatively low but we have to continue with treatment to avoid refilling of the aortic sac, especially when we have a good position for the SG without sac dilatation, which can lead to loss of sealing zones. The advantage is that progressive EL2 is not as aggressive as endoleak type 1 and we should have enough time to plan the best strategy for treatment. EL2 remains the important diagnosis, which requires precise monitoring and appropriate investigation as well as an individual plan for each patient involving conservative, laparoscopic, or open surgical treatment. Dynamic filling of the sac in an EL2 directly after SG deployment can predict the future of EL2 but we have to evaluate a number of factors, which play a role in the persistence and progressivity of EL2 [17].

These factors are:

- Systolic and diastolic blood pressure
- Peripheral resistance and calcification of the sac branches (its origins)
- Position of the SG

- Volume of a pulsatile cavity with arterial blood flow (in the sac and around the SG)
- Anticoagulation/antiaggregation therapy
- Numbers and diameters of patent LAs [36]
- Diameter of IMA [36]
- Warfarin [25]

Intra-operative predictors of the EL2 diameter of IMA and position of LAs play important roles as predictive factors of EL2, together with the diameter of the visceral branches (origins), their calcifications, and pressure gradients. The flow between aortic visceral branches is similar to the arteriovenous fistula. Pulsation cavity around the SG involving the origin of LAs or IMA, where the SG can work as a piston to pump blood between the LAs or IMA and another LA, respectively the sac branches. This physical mechanism can keep the patency of the EL2 and increase pressure in the sac, which can be a reason for the progressivity of the EL2 with growth of the sac. It is thought that 3D printing of the endoleaks as a real model of the CTA with important details could provide an ideal view of the pathology and a realistic overview of the best method for treatment. Anatomical risks regarding potential EL2s as accessory renal arteries, diameter, and position of LAs should also be considered. It is believed that the position of the SG plays an important role when we consider the pulsation cavity between the SG and the dorsal aneurysmal wall. Pulsation mass due to EL2 and SG pulsation can both work as a pump because visceral aortic branches have low peripheral resistance and could help to keep the EL2 persistent and growing (**Figure 1**). The pulsation cavity can potentiate the pressure gradient between LAs and between IMA and LAs as well, therefore systemic blood pressure and aortic branch calcifications (blood vessel rigidity) can play an important role regarding patency and endoleak flow. When the SG is close to LA origins its sealing effect can reduce the incidence of EL2 because a potential volume of EL2 is reduced. Slow progression of the sac dilatation due to EL2 can give us useful time to arrange our strategy of treatment. Monitoring is very important to check progression. Prevention involves selective embolization of the aneurysmal sac branches or use of the special type of SG as described for endovascular aneurysm sealing [19, 37]; however, together all effects of the device involve complications (endoleaks, SG misplacement). The preventive embolization of the sac patent branches (LAs, IMA, etc.), which can contribute to formation of EL2, leads to a reduction in EL2 incidence. When we compare the preventive sac embolization only (coils + glue) with selective sac branch embolization we can confirm that the selective approach is more efficient and has fewer artifacts and embolization materials. When we compare longer clinical outcomes, we can confirm there is no statistical evidence that sac preventive embolization has long-term efficiency as a technique to prevent EL2 [21, 22]. It is recommended to embolize IMA and LA before SG deployment when the lumen diameter is more than 2–3 mm or if the number of patent LAs is more than 3. The role can play a distance between origins of LA as well because their flow communication is with higher predisposition. Conservative treatment involves blood pressure treatment, statins, and the possibility of reducing antiplatelet or anticoagulation therapy for a short period to support spontaneous sealing of the EL2 together with shrinkage of the sac. We should consider timing for future plans if conservative treatment does not give results when ultrasound and three-phase CTA are used. The possibility of

treatment involves endovascular and surgical treatment. Minimally invasive surgical techniques are a safe option with good results. If endovascular treatment is not successful, then we can proceed with a laparoscopic approach to clip LAs and the IMA. The retroperitoneal approach reaches both sides of the aortic sac with good visibility of the LA origins. If the main body of the SG is too far from the dorsal aortic wall and there is a relatively large space behind the SG (such as a pulsatile volume), then we can consider an open approach with endoaneurysmorraphy to stabilize the SG position and minimize the space around the SG. The preassessment of any intervention, especially the surgical one, should involve patient fitness, reserves, and risks in the context of patient comorbidities. Patient quality of life is an important point in our decision to treat progressive EL2. The standard monitoring plan can be within 3–6 months in the first year after reintervention and then every half year or at 1-year intervals, eventually becoming more frequent as an individual plan due to potential restoration or previous high progressivity of EL2. We should consider a fresh CTA or a new method of scanning such as 4D magnetic resonance angiography [16], or maybe an angiogram with the possibility of endovascular reintervention. The importance of diagnosis and treatment of EL2 is to investigate hemodynamic characters of the EL2 to consider an individual plan and watch the dynamic changes in time to consider the timing of intervention. When renal functions are borderline and with a view to quicker progression, it would be better to prevent further sac growth due to EL2 and find a reason for quicker treatment to avoid issues of providing endovascular or surgical treatment. EL2 is not as aggressive as the endoleak type 1 and 60–80% of cases can be treated conservatively. The residual numbers of patients with the progressive type of EL2 require an intervention to avoid severe complications. Endovascular treatment is the first step in progressive EL2. It has good efficiency and the possibility of repeating the intervention. The laparoscopic procedure of clipping IMA and LAs is a relatively safe option from peritoneal or retroperitoneal points of view with good results, and is a minimally invasive approach, which indicates the best individual treatment for each patient to prevent complications after EVAR. Open endoaneurysmorrhaphy [34, 35] is another treatment for patients where the previously described treatment is not successful and there are difficulties using a laparoscopic method. The risks are low where the patient is relatively fit for the intervention and an experienced team can prevent misplacement of the SG to minimize blood loss without an aortic clamp. Explantation of the SG is not recommended because it is a very dangerous technique with very high lethality [30]. Haq IU et al. presented their results regarding the incidence and treatment of EL2 [6]. They describe 386 patients over 10 years with a 21% (81 patients) incidence of EL2, which was treated in 65% (53 patients) conservatively and in 35% (28 patients) by intervention, in addition to 60% (17 patients) endovascularly and 40% (11 patients) transarterially. They concluded that the incidence of progressive EL2 could be represented by an aggressive phenotype. Kumar et al. presented a cohort of 693 patients (2009–2013) with EVAR [38]. The team treated 225 patients due to EL2. The mean follow-up was 2 years. One hundred and thirty-three patients were resolved spontaneously, 37 were unresolved and untreated, 16 underwent an intervention, 3 had a AAA rupture due to EL2, and 2 patients were in the absence of the sac expansion ≥5 mm/6 months. The late type of EL2 occurred in 117 patients, of which 26 had sac expansion. They concluded that age and smoking were significant independent predictive factors for non-survival. They described the rupture of two patients after EVAR without sac enlargement ≥5 mm. Ultee and his colleagues described in a systematic review that the persistence of EL2 could be

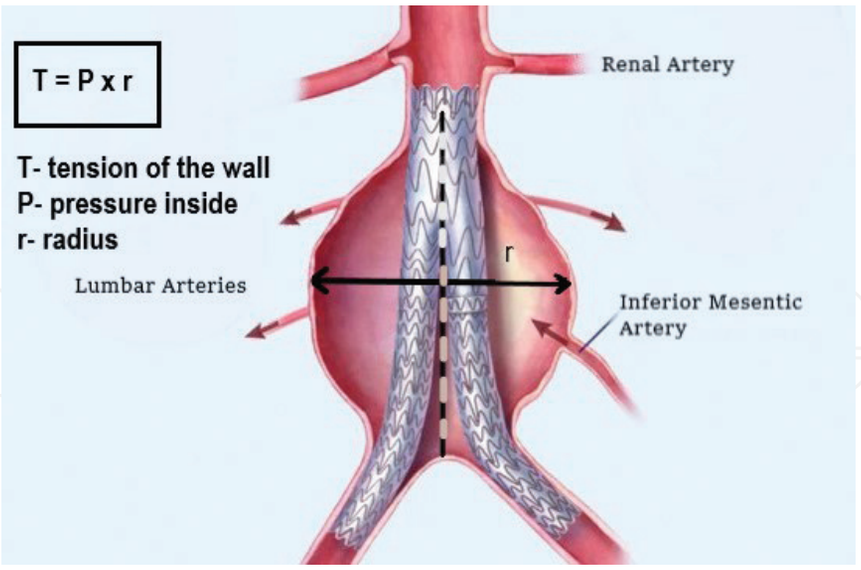

Figure 8. Laplace's law is described as tension (*T*) of the aneurysm wall, which is relative to pressure (*P*) in an aneurysm and the radius (*r*) of an aneurysm, $T = P \times r$.

accompanied by risk of rupture without an extensive growth up to 1.8% [12]. This is not common and very probably depends on the pressure in the sac and aortic wall, respectively sac wall endurance, where Laplace's principle explains the pathology (**Figure 8**). Statins are described as an important part of post-EVAR treatment to support sac shrinkage and decrease the incidence of endoleaks involving EL2. The theory is that the effect of statins helps to stabilize the arterial wall with atherosclerotic changes. The authors presented the shrinkage of an aneurysm within 12 and 24 months [1, 27, 28]. With the results we can expect to prevent endoleaks by prolonged statin administration together with a low-fat diet and regular control of serum lipids and lipoproteins. Patients using warfarin can have a higher predisposition for persistent or progressive EL2 [25]. We should consider omitting warfarin for a short period or using another type of anticoagulation (LMFW) if there are no further risks regarding their initial indication (ischemic heart disease, AF, DVT, PE, etc.). Another author suggested that antiplatelets such as salicylates and clopidogrel do not increase the risk of endoleaks after EVAR [39].

6. Conclusion

EL2 is a relatively non-aggressive diagnosis after EVAR but its growth can lead to severe complications. Monitoring keeps the situation under control and diagnoses the progression over time so that a strategy can be implemented when progressive EL2 requires treatment. We can summarize the clear process regarding progressive EL2 as follows [40–42].

Prevention:

Embolization of the large abdominal infra-aortic visceral branches prior to SG deployment.

Diagnosis:

Duplex scan (measurement of sac growth), contrast-enhanced Duplex scan, and three-phase CTA.

Treatment:

- Conservative (endoleak without extensive sac growth).
- Endovascular with embolization of IMA and reachable aortic sac branches.

Surgical:

- laparoscopic clipping of IMA and LA.
- endoaneurysmorrhaphy and a tight suture around the SG.

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