Case Report
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A Case of Endoleaks from an Endovascular Prosthesis for Aortic Aneurysm

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Abstract

Background: Many chronic conditions, as diabetes (DM) and cardiovascular Diseases suffer Major Adverse Cardiac Events (MACE): i myocarditis, congestive heart failure (CHF), Ventricular Tachycardia (VT), Ventricular Fibrillation (VF), Acute Coronary Syndromes [ACSs], and Sudden Cardiac Death (SCD) Acute infections, like COVID-19 also involve oxidative stress, leading to increased Sympathetic tone (S) and decreased Parasympathetic tone (P), increasing Sympathovagal Balance (SB) and MACE. The antioxidant (r)alpha lipoic acid (ALA) improves SB. The anti-anginal Ranolazine (RAN), also an antioxidant is an anti-arrhythmic. Our studies of their effects on MACE in DM, and non-DM patients with CHF, ventricular arrhythmias and SCD are reviewed herein, as our findings may apply to acute diseases, such as COVID-19.

Methods: In a case-control study, 109 CHF patients, 54 were given adjunctive off-label RAN added to ACC/AHA Guideline therapy (RANCHF). MACE and SB were compared with 55 NORANCHF patients; mean f/u 23.7 mo. 59 adults with triggered premature ventricular contractions (PVCs), bigeminy, and VT were given off-label RAN. Pre and post-RAN Holters were compared; mean f/u 3.1mo. 133 DM II with cardiac diabetic autonomic neuropathy were offered (r)ALA; 83 accepted; 50 refused. P&S were followed a mean of 6.31yrs, and SCDs recorded.

Results: (1) 70% of RANCHF patients increased LVEF 11.3 EFUs (pâ‰¤0.003), SCD reduced 56%; VT/VF therapies decreased 53%. (2) 95% of patients responded: VT decreased 91% (p<0.001). (3) SCD was reduced 43% in DM II patients taking (r)ALA (p=0.0076). Conclusions: RAN, (r) ALA treat CHF, VT, and prevent SCD. Trials in COVID-19 are needed.

Keywords: Endoleaks • Cardiovascular • Aneurysm • Endovascular • Prosthesis

Introduction

One of the conditions that require surgery on the aorta is the thoracic aortic aneurysm. It is a loss of parallelism between the walls of the aorta causing dilation. The risk of rupture becomes significant when this expansion reaches a certain threshold generally defined by a diameter ≥ 5.5 cm, and/or a growth rate ≥ 1 cm/year [1].

The thoracic aorta is divided into three segments: the ascending aorta, the descending aorta and the aortic arch. The most often affected part is usually the ascending aorta which is the cause of the majority of operations for an aortic aneurysm [2].

The etiology of the aortic aneurysm can be divided in two main parties [3]: Congenital diseases like Bicuspidy of the aortic valve, Marfan syndrome or Ehlers-Danlos syndrome, while Acquired diseases are mainly due to Atherosclerosis, arteritis, infections or traumatic events.

The dilation is most often found by chance in asymptomatic patients. It is found during a chest x-ray, CT or MRI scan, echocardiogram or aortography [4]. Severe chest pain may be a sign of rupture or cracking of the aneurysm. The patient must be operated on as a matter of extreme urgency in order to stop or prevent the bleeding. The aneurysm can also sometimes release clot debris which will then occlude the arteries in the legs. More rarely, it can compress adjacent structures such as the intestine, urinary tract or iliac veins.

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The main treatment for aortic aneurysm is surgery [5]. The different operating techniques are:

- Replacement of the aortic valve (with a mechanical or biological prosthesis) associated with the replacement of the entire ascending aorta and re-implantation of the coronaries
- Replacement of the entire ascending aorta and re-implantation of the coronaries with preservation of the native aortic valve
- Replacement of the ascending supra-coronary aorta associated with replacement of the aortic valve
- Replacement of the ascending supra-coronary aorta
- Replacement of the aortic arch.

Endovascular technique can also be considered [5], this technique involves inserting a prosthesis (a stent) into the aneurysmal aorta through the interior of the femoral arteries. Only two punctures or small incisions are needed at the folds of the groin to insert the stent. This technique has the advantage of being minimally invasive and of presenting a lower risk of mortality and complications than short-term surgery and is usually preferred in elderly patients with comorbidities.

Here we report a case encountered in our service of cardiac surgery concerning an elderly patient treated with an endovascular stent on a pre-existing aortic arch prosthesis for endoleak and massive hematic pleural effusion.

Case Report

We present the case of an 88-year-old male presenting to the emergency department for orthopnea and fatigue associated with a cervical pain exacerbated on the extension of the neck. On his arrival, the patient noted several episodes of hypotension reaching 70mmHg of Systolic arterial pressure.

On admission, vital signs were Pulse rate: 129 BPM; SAP: 94 mmHg; DAP: 61 mmHg; SaO_2: 95%; Temperature: 36.7

The patient is known for having an atrial fibrillation treated with Apixaban 2.5 mg per day, Obstructive Sleep apnea treated with CPAP, benign hypertrophic prostate treated by Trans urethral resection of prostate, Chronic renal failure Stage V with dialysis 3 times per week since 2016 and Myelodysplasia. We also note that the patient had an endovascular stent in 2009 for an aortic aneurysm in the ascending and descending aorta.

The procedure done in 2009 was performed at two times:

1. Ascending Aorta and aortic arch prosthesis with Brachiocephalic truncus and left primitive carotid implantation with CABG of LCA, LDA and LMA
2. Endovascular prosthesis of the descending aorta

In Figure 1, we show the Contrast enhanced computed tomography performed after the surgical intervention in 2009 as part of the medical history of our patient.

In the CT- Scan report, we can notice a normal aspect of the ascending aorta where we find the surgical prosthesis beginning at 1 cm after the sino-tubular junction. It continues with an endoprosthesis placed percutaneously and covering the descending aorta up to the thoraco-abdominal junction. These are two units with overlap. We note a good coaptation of the endoprosthesis without periprosthetic leakage. The pleural effusion is reduced to 50%.

Abdominal, cardiac, pulmonary and neurological examinations were done systematically, showing a complete abolition of the breath sounds on the left lung, irregular heartbeats with a systolic murmur at the left sternal board. The rest of the examination was normal.

An EKG was performed by the intern showing a rapid Atrial fibrillation at 120 beats per minute but well tolerated by the patient.

A series of examinations was conducted including a complete blood analysis as shown in the Table 1, and an ABG as shown in Table 2.

A CXR performed on the 16th of April showed a complete left pleural effusion as shown in Figure 2. An injected thoracic CT Scan was then required to elucidate the origin of the pleural effusion, the CT scan is shown in Figure 3.

On the Scan report, the imagery shows the presence of several periprosthetic leak sites; the first of type I is located postero-superiorly at the level of the arch of the aorta starting a few centimeters from the ostium of the left sub clavicular artery, a second of type III located on the anterior wall of the descending aorta and a third probably independent of the previous one but of the same type, lower located at the level of the descending aorta near a prosthetic plicature at this level.

Due to all these findings, an intervention is required via radiologic intervention by implementing an endovascular stent, followed by an evacuation of the pleural effusion with a chest tube.

The intervention was successful with no major complications during the procedure. The chest tube evacuated 3000ml on day 0, 700ml on day 1, 550ml on day 2, 570ml on day 3, 530ml on day 4 and then the chest tube was removed. Hemodynamics of the patient was stabilized in the cardiac intensive care unit.

Discussion

Owing to high rates of immediate success and low morbidity, endovascular stenting has become an alternative to conventional surgery in the treatment of (pseudo) thoracic and/or abdominal aortic aneurysms [5]. Endoleaks, one of the main causes of failure of endovascular procedures [6], are defined as the presence of blood flow outside the prosthesis and within the aneurysm sac. Their rate of occurrence is estimated at 5 to 20% of cases of thoracic and/or abdominal aortic stent grafting [7].

A recent classification similar to the WHITE classification [8] commonly used for infra-renal aortic aneurysms, defines 4 types of endoleaks. The classification is shown in Table 3. Endoleaks can also be classified chronologically: primary endoleaks being discovered within the first 30 post-operative days and secondary endoleaks discovered later.

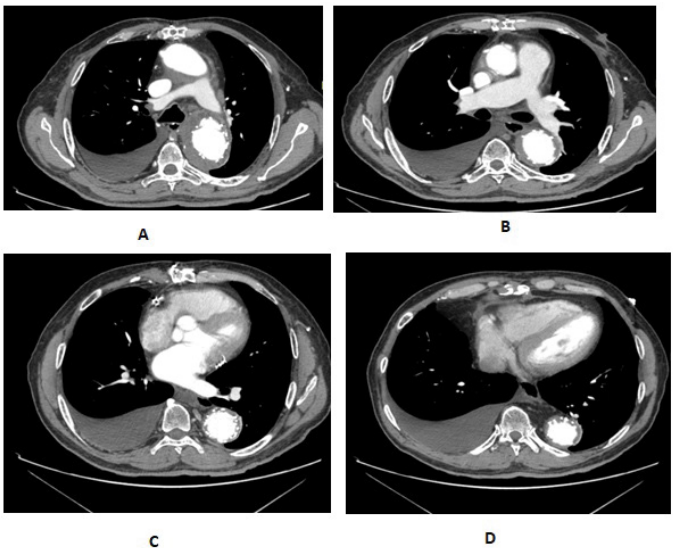

Figure 1. Contrast enhanced computed tomography performed after the surgical intervention.

Table 1. Showing results of blood analysis (Hematology, biochemistry, immunology) dated 16-04-2021.

Blood Analysis	Results	Normal value
White blood cells	5600	4000-10000/mm^3
Red blood cells	3.41	4.3-5.7 × 10^6/mm^3
Hemoglobin	10.4	14-17.5 g/dl
Hematocrit	32.8	41.5-50.4%
MCV	96	80-98 fl
MCHC	31.8	32-36 g/dl
MCH	30.6	27.5-33.2 pg
RDW	19.3	10-15%
Platelets	80000	150000-400000/mm^3
Neutrophils (%)	75	43-70%
Lymphocytes (%)	18	38-42%
Monocytes (%)	6	5-10%
Eosinophils (%)	1	0-5%
Basophils (%)	0	0-1%
Urea	16	2.5-7.5 mmol/L
Creatinine	532	46-92 micromol/L
Na	138	135-145 meq/L
K	4.1	3.5-4.5 meq/L
Cl	98	98-107 meq/L
Alkaline reserve	31	24-30 meq/L
PT*	53	70-100%
TCA*	55	37sec

*We note that the patient is under Apixaban (Eliquis)

Table 2. Showing the ABG results.

PH	7.47	7.35-7.45
PCO^2	44	35-48 mmHg
PaO^2	75	83-100 mmHg
HCO^3	32	*20-24 mmol/L*
SaO^2	96	96-100%
Base excess	7.5	2-3 *mmol/L*

Treatment of endoleaks

- Type I and III endoleaks present a high risk of breakage due to the absence of exclusion of the aneurysm sac from the systemic

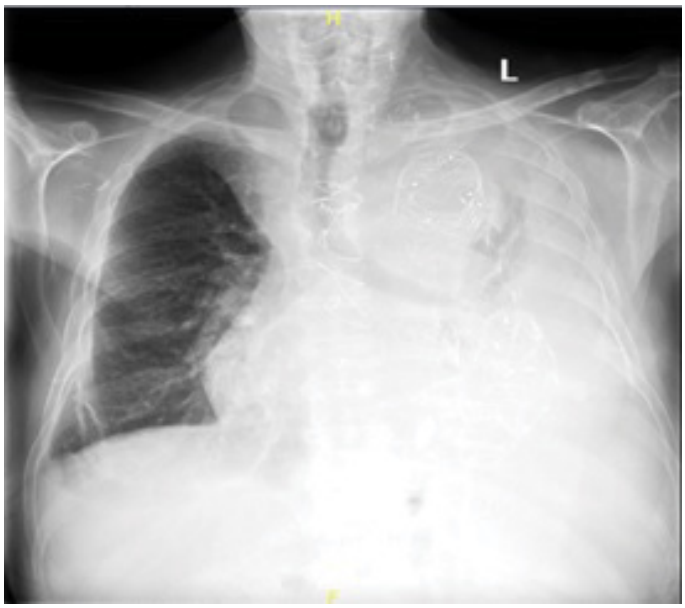

Figure 2. Chest X-ray performed on 16-04-2021.

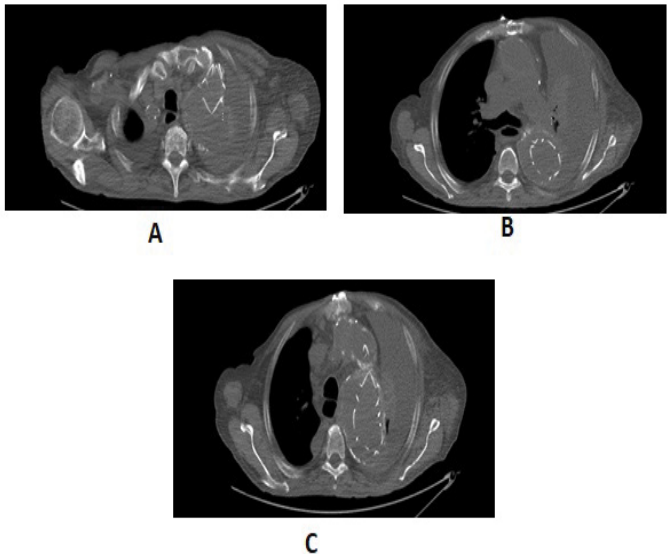

Figure 3. Injected thoracic CT Scan.

circulation. Therefore, the general consensus is to actively treat them as soon as they are identified with either new endovascular procedure or the placement of a covered prosthetic extension (=cuff) or a new endoprosthesis [9].

- Type IV endoleaks, which have become rare with new generations of stent grafts [10], will be treated if they persist beyond one month, as they are then considered to be type III endoleaks.
- There is currently no therapeutic consensus for type II endoleaks [11]. Many authors are increasingly in favor of radical treatment once the diagnosis has been established, because of the participation of these endoleaks in a risk of progress towards rupture. Several therapeutic alternatives are currently proposed [11]. One of them consists of endovascular embolization, that is to say the obliteration of the collateral responsible by the establishment of millimetric metallic foreign bodies (coils), causing thrombosis of the lumen arterial. Another therapeutic option could be laparoscopic vascular surgery, allowing easy control and radical treatment of type II endoleaks using clip, while remaining less invasive than conventional surgery [12]. However, in most cases Type II endoleaks will be treated conservatively by close monitoring, unless expansion continues during monitoring.

Table 3. Classification of endoleaks [8].

A. Type I endoleak
TL based
• Type I (TL)a → proximal seal failure with persistent flow through the primary entry tear and antegrade FL filling
• Type I (TL)b → Distal seal failure with persistent retrograde Fl flow through distal entry tear (s)
FL based
• Type I (FL)a → Antegrade proximal FL filling due to incomplete coverage of the primary tear and/or additional proximal entry tears
• Type I (FL)b1 → Retrograde distal FL flow through one of more major distal entry tears
• Type I (FL)b2 → Persistent FL flow through multiple minor secondary entry tears
• Type I (FL)d → dSINE
• Type I (FL)r → RAAD
B. Type II endoleak
• Type II (FL)a → Retrograde flow into FL via multiple small posterior branches
• Type II (FL)x → Retrograde flow into FL via at least one large aortic branch or at least one visceral branch
C. Type III endoleak
• Type IIIa → component disjunction in modular repairs
• Type IIIb → structural defect in the graft
• Type IIIc → Poor branch stent fenestration seal
• Type IIIg → Gutter leak
D. Complex endoleak
• Various combinations of the above subtypes
TL: True Lumen; FL: False Lumen; Dsine : Distal Stent Induced New Entry; RAAD: Retrograde Ascending Aortic Dissection

In our case, the endoleak is classified as secondary type I and type III endoleaks causing a leakage into the left pleural cavity through the pleural incision done in the Coronary artery bypass Graft, which led to a complete left hemothorax. The patient presented to the ED with hemodynamic instability upon his arrival with a SAP below 70mmHg requiring an immediate intervention.

We preferred not to evacuate the pleural effusion caused by the endoleaks to prevent massive blood loss and extreme hemodynamic instability. Therefore, the patient was driven to the OR with the presence of a cardiothoracic surgeon, an interventional radiologist and an anesthetist.

The endovascular stent was inserted via femoral artery and placed in the aortic arch with close monitoring via aortography. The intervention was successful followed by a chest tube placement. The patient was then monitored in the Intensive care department.

Conclusion

1. We can learn that endovascular approach for endoleaks is safe in elderly patients; a larger data collection is needed in further studies to assess the tolerance and efficiency of this technique in old patients with comorbidities.
2. The diagnosis should be quick with no time to lose, and an immediate intervention is needed to stabilize the patient and preserve his life.
3. Chest Tube must be indicated after the intervention to prevent massive hemodynamic outcomes.
4. Aging is a risk factor and a possible etiology for endoleaks. We suggest in our case that the patient had a displaced stent due to his axial aging which led us to a large questioning about aneurysm stents in patients with a life expectancy that exceeds 10 years from the date of the intervention.

5. Large studies with sufficient data are still required to verify our hypothesis, respond to our concerns and elucidate the outcomes of invasive interventions in elderly patients.

Informed Consent

Written informed consent for the publication of the article was obtained from the patient.

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