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CASE SERIES
Other

Combined thoracic endovascular aortic repair and endovascular aneurysm repair and the long-term consequences of altered cardiovascular haemodynamics on morbidity and mortality: case series and literature review

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Background Thoracic and abdominal aortic stent grafts are firmer and more rigid than the native aorta. Aortic implanted devices have been implicated in the development of acute systolic hypertension, elevated pulse pressure, and reduced coronary perfusion.

Case summary We report four cases of staged thoracic endovascular aortic repair (TEVAR) and then endovascular aneurysm repair (EVAR). All patients had TEVAR first for thoracic aortic aneurysm and later on developed infra-renal abdominal aortic aneurysm (AAA) that required EVAR. There were three males and one female with a median age of 74.5 years (range 67.5–78.5). None of the patients developed aortic-related major clinical adverse effects or required any aortic intervention during their follow-up. However, within 2 years, all patients developed symptomatic left ventricular hypertrophy with diastolic dysfunction. All patients had bilateral lower limb oedema, with on and off chest pain and shortness of breath (SOB), necessitating coronary angiograms, which showed no evidence of coronary artery disease. Three patients died from cardiovascular-related morbidities, and the fourth patient is still complaining of SOB despite a normal coronary angiogram.

Discussion Aortic-endograft compliance mismatch is an invisible enemy, with troubling consequences for the aorta proximal and distal to the endograft. Aortic stiffness due to vascular endograft could lead to cardiovascular adverse events, even in the absence of direct aortic-related complications. After combined TEVAR and EVAR, the compliance mismatch and elasticity loss are even more pronounced than with TEVAR alone, which necessitates patient monitoring for the development of cardiovascular complications.

Keywords Aortic aneurysm • Thoracoabdominal • Stents • Vascular capacitance • Cardiovascular complications • Case report

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Learning points

- Lining the aorta with metallic stent-grafts creates stress on the aortic wall with subsequent left ventricular hypertrophy, diastolic dysfunction, and systolic hypertension refractive to antihypertensive medication
- Non-occlusive coronary artery ischaemia can lead to chest pain post-thoracic endovascular aortic repair (TEVAR)/ endovascular aneurysm repair.
- Patients who have TEVAR must be monitored for the development of distal abdominal aortic aneurysm and congestive cardiac failure.

Introduction

Thoracic and abdominal aortic stent-grafts are firmer and more rigid than the native aorta. Even the best available aortic graft designs alter the function of the native aorta considerably, regardless of the underlying aortic disease.[1–3]

Thoracic endovascular aortic repair (TEVAR) and aortic endovascular aneurysm repair (EVAR) correlate with reduced early peri-operative morbidity and mortality compared to open surgical repair. However, this gain is diminished at long-term follow-up, primarily due to an increase in cardiovascular complications, the risk of development of which is enhanced by arterial stiffening, mainly related to the stent-graft materials.[1]

Aortic implanted devices have been implicated in the development of acute systolic hypertension, elevated pulse pressure, and reduced coronary perfusion.[2] However, there is no insight in the cardiovascular community about cardiac remodelling after aortic stenting, and interventionalists have focused on the endograft morphological adaptation to the aortic wall. Post-endovascular aortic surveillance is therefore concentrated on maintaining endograft position and avoidance of expansion of the aortic sac. These objectives are maintained even if that results in further intervention with implantation of additional endograft components, stents, and coils, which further increases aortic wall stress and harm cardiac, cerebral, renal, and mesenteric perfusion.[1–3]

This case series demonstrates some of the consequences of enhancing aortic wall stress post-TEVAR, with subsequent development of infrarenal abdominal aortic aneurysm (AAA), which, when managed by EVAR, induces further cardiac injury, with high cardiovascular morbidity and mortality.

Case series

Out of 18 791 aortic referrals to our tertiary referral centre, we performed 1480 aortic interventions over 20 years.

Ninety-six interventions were TEVAR/branched endovascular aortic repair (BEVAR), of which 19 were hybrid aortic repair (HAR); 910 EVAR ± Iliac Branch Device, of which 44 were HAR; 213 open aortic interventions, of which 51 were HAR; and 261 aorto-iliac revascularizations for severe aorto-iliac occlusive disease, of which 73 were HAR.

We report four cases of TEVAR, which subsequently required EVAR (*Timeline*, *Figures 1*, *2*, *3*, *4*, *5*, and *6*). All patients had TEVAR first for thoracic aortic aneurysm (TAA) and later on developed infra-renal AAA that required EVAR.

Two patients presented in close succession who developed AAA reaching the threshold for intervention within an exceptionally short time since their TEVAR. This forced us to audit our TEVAR cases and, in particular, identify if other TEVAR patients required subsequent intervention for AAA. We identified the two additional patients, which constituted the current series.

Cardiovascular risks factors are almost ubiquitous among patients with degenerative aneurysms and while cardiovascular risk factors are implicated in aneurysm progression in general, what distinguishes this series of patients is the rapid progression of the abdominal aortic disease subsequent to TEVAR implantation, which could not be explained by the underlying cardiovascular risk factors alone.

There were three males and one female patient with a median age of 74.5 years (range 67.5–78.5). None of the patients developed aortic-related major clinical adverse effects or required aortic intervention (i.e. rupture, dissection, endoleak, sac expansion, or device migration) directly related to their primary TEVAR during the follow-up.

For TEVAR, we utilized two Valiant thoracic aortic devices (Medtronic, Minneapolis, MN, USA) (*Figures 1 and 5*) and two cTAG devices (Gore Medical, Flagstaff, AZ, USA) (*Figures 3 and 6*). All TEVARs were executed using two pieces each.

For EVAR, we used three AFX endografts (Endologix, Irvine, CA, USA) (*Figures 1*, *5*, and *6*) and one Excluder (Gore Medical, Flagstaff, AZ, USA) (*Figure 3*).

Mean pre-operative D-Dimer was 5790 ng/mL (range 1198–9801), and it did not vary postoperatively. Pre-operative FEVI and predicted FEV1/FVC were all above 75%.

Median pre-operative antihypertensive tablets per patient were 1.75 (range 1–2) that increased to 4.25 (range 3–5) postoperatively. Median pre-operative eGFR was 75 mL/min/1.73 m^2 (range 57–84.5), while the post-operative median eGFR was 74 mL/min/1.73 m^2 (range 46.4–85). All our patients had a reasonable pre-operative echocardiogram (ECHO) (normal ventricular size and function, left ventricular ejection fraction >55%, no atrial dilatation, and no valvular disease) with median pro-BNP of 401 pg/mL (range 206.5–717.85), however, post-operative median pro-BNP had risen to 3053 pg/mL (range 1426.5–5686.5).

Median pre-operative troponin was 2 ng/mL (range 1–6.5) that increased to 46.5 ng/mL (range 16.5–106.5) following the procedure. All patients were hypertensive, and postoperatively all of them developed wide pulse pressure with sustained high systolic pressure (above 160 mmHg) and low diastolic pressure (below 55 mmHg).

Patients' cardiac function was classified according to the New York Heart Association (NYHA) Functional Classification. All had pre-operative functional capacity II and objective assessment B. Post-operatively, 50% of patients moved up one category, while the other 50% moved to functional capacity IV with objective assessment D. All patients developed prolonged Q-T interval with resultant new-onset atrial fibrillation during follow-up.

Timeline

Case	Age/Sex	Figures	Co-morbidities	Thoracic endovascular aortic repair(TEVAR)			Endovascular aneurysm repair (EVAR)			Remarks
				Date	Indication	Stent-graft employed	Date	Abdominal aortic aneurysm (AAA) size	Stent-graft employed	
One	61/M	*1A–D; 2A,B*	Hypertension (HTN), hyperlipidaemia, chronic obstructive pulmonary disease (COPD). Smoker (20 pack years)	2007	6 cm descending thoracic aortic aneurysm (TAA) (No evidence of infrarenal abdominal aneurysm)	Valiant thoracic aortic devices (Medtronic, Minneapolis, MN, USA) (2 pieces)	2009	5.5 cm (infrarenal)	AFX endografts (Endologix, Irvine, CA, USA) (3 pieces)	Death due to fatal cardiac arrhythmia 9 years following TEVAR, 7 years following EVAR Q-T interval prolongation with atrial fibrillation (AF) with normal coronary angiogram
Two	76/M	*3A–C; 4A,B*	HTN, COPD, asthma, hypothyroidism. Smoker (35 pack years)	2016	Type B aortic dissection (TBAD) + Extent I 7.3 cm TAA (No evidence of infrarenal abdominal aneurysm)	cTAG devices (Gore Medical, Flagstaff, AZ, USA) (2 pieces)	2017	5.8 cm (Infrarenal, ? sequelae of HTN or stent induced new entry)	Excluder (Gore Medical, Flagstaff, AZ, USA) (1 piece)	Death due to congestive cardiac failure and myocardial ischaemia 4 years following TEVAR Q-T interval prolongation with AF with normal coronary angiogram
Three	73/M	*5A–D*	HTN, chronic kidney disease. Smoker (25 pack years)	2010	Proximal TBAD + 6.6 cm aneurysmal false lumen	Valiant Thoracic Aortic devices (Medtronic, Minneapolis, MN, USA) (2 pieces)	2010	5.5 cm (Infrarenal)	AFX endografts (Endologix, Irvine, CA, USA) (3 pieces)	Death due to congestive cardiac failure and myocardial ischaemia 2 years following TEVAR Q-T interval prolongation with AF with normal coronary angiogram
Four	81/F	*6A–C*	HTN, hyperlipidaemia, right superficial femoral artery (SFA)/popliteal/tibial angioplasty and SFA stenting. Smoker (30 pack years)	2015	Saccular descending TAA	cTAG devices (Gore Medical, Flagstaff, AZ, USA) (2 pieces)	2018	4.9 cm (Infrarenal, saccular AAA)	AFX endografts (Endologix, Irvine, CA, USA) distally (3 pieces) and Gore cTAG (Gore Medical, Flagstaff, AZ,USA) proximally (1 piece)	Patient is alive; however, she is suffering from cardiovascular complications with shortness of breath. Q_T interval prolongation with AF with normal coronary angiogram

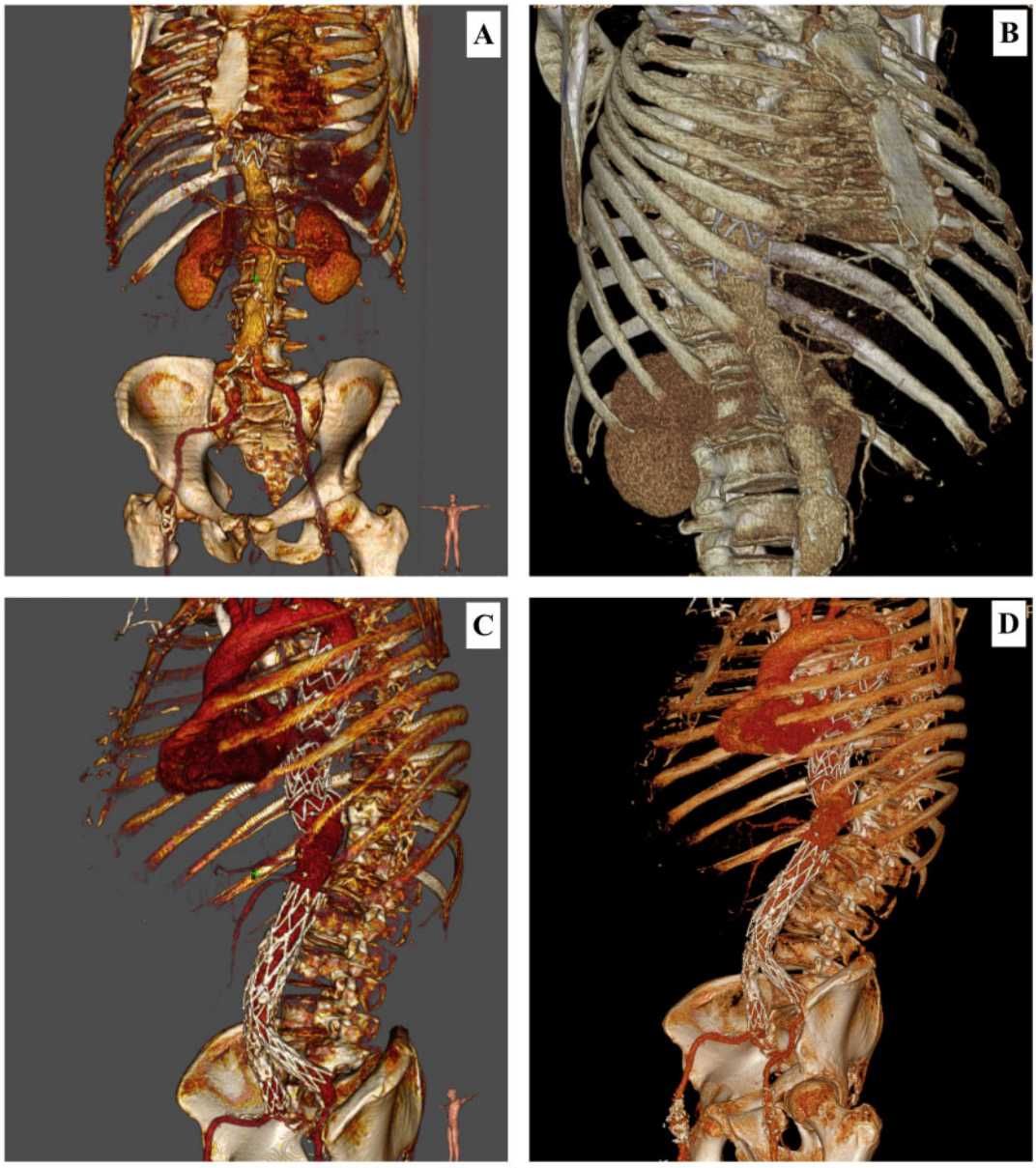

Figure 1 A 61-year-old male presented with a 6cm descending thoracic aortic aneurysm (TAA) and underwent thoracic endovascular aortic repair (TEVAR) in 2007. Within 21 months, he developed an abdominal aortic aneurysm (AAA) of 55 mm with the right common iliac artery aneurysm. Endovascular aneurysm repair (EVAR) was performed in 2009 with an uneventful post-operative course. His past medical history included hypertension, hyperlipidemia, chronic obstructive pulmonary disease (COPD) and smoking (20 pack-years). He died from cardiovascular-related complications nine years after his first TEVAR/EVAR. (*A*) A 3D-computed tomography angiography (CTA) follow-up of TEVAR at six weeks, showing 29 mm aorta. (*B*) A 3D-CTA at 29 months depicts 55 mm infrarenal AAA. (*C*) A 3D-CTA follow-ups at five years with two Valiant (Medtronic, Minneapolis, MN) endografts proximally and three pieces of AFX (Endologix, Irvine, CA) in the abdomen (main body 28mm with two 34mm proximal extensions). (*D*) A 3D-CTA follow-ups at eight years, showing no evidence of aneurysmal related endograft problems.

All patients who had TEVAR as the first step did not have an infrarenal aneurysm at the index procedure but developed AAA after TEVAR in a median of 9 months (range 4.5–24).

Within 2 years, all patients had developed symptomatic left ventricular hypertrophy with diastolic dysfunction as documented on ECHO. All patients had bilateral lower limb oedema, with on and off chest pain and shortness of breath (SOB), necessitating coronary angiograms (*Figures 2*, *4*, *5*, and *6*), which showed no evidence of coronary artery disease (CAD).

Three patients died from cardiovascular-related causes over 15 years of follow-up (*Timeline*). One patient had a fatal cardiac arrhythmia, and two died following progressive cardiac failure and

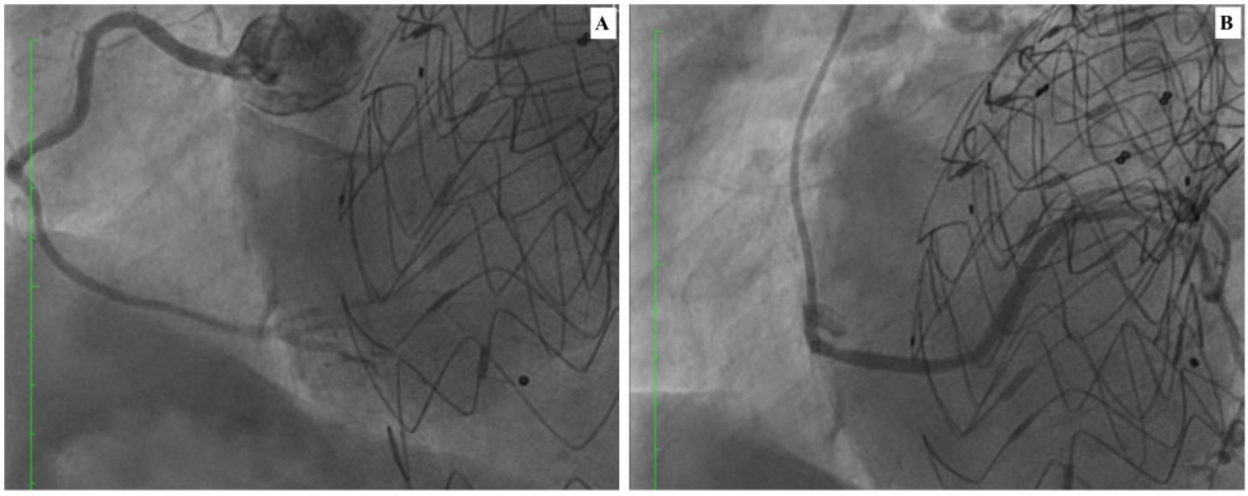

Figure 2 Coronary angiogram of the right (*A*) and left (*B*) main coronary arteries showing pristine coronaries without evidence of plaques.

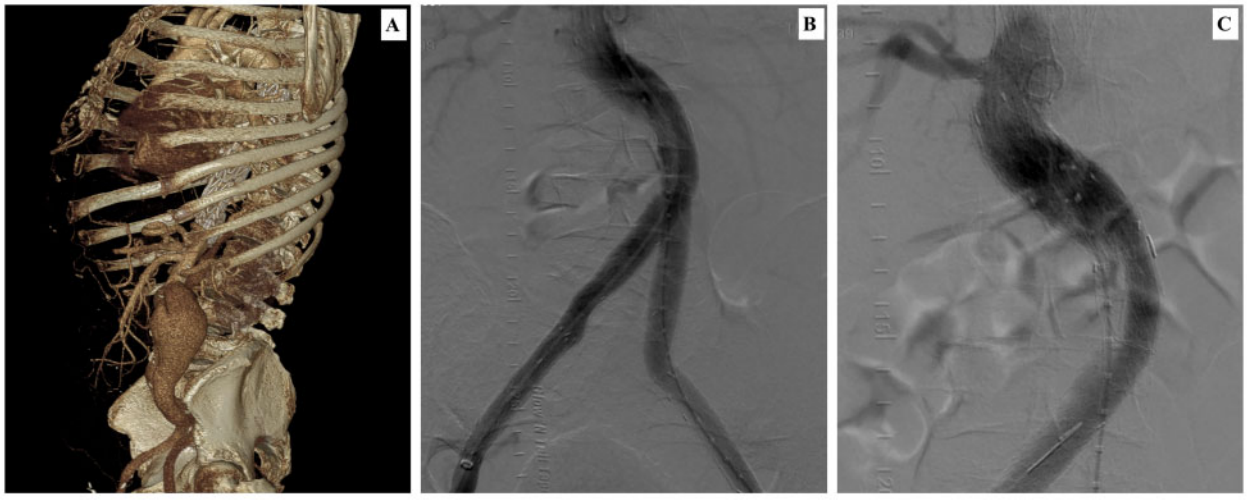

Figure 3 A 76-year-old man presented with acute type-B aortic dissection that stopped above the coeliac axis with an associated Extent I thoracic aortic aneurysm of 73 mm treated conservatively by strict blood pressure control for 2 weeks and thoracic endovascular aortic repair on 12th July 2016 with a GORE cTAG (Gore Medical, Flagstaff, AZ, USA) device. He required endovascular aneurysm repair for a 58 mm abdominal aortic aneurysm on 13th September 2017 using a GORE Excluder (Gore Medical, Flagstaff, AZ, USA) with Ballerina technique. He had a past medical history of hypertension, chronic obstructive pulmonary disease, asthma, hypothyroidism, and smoking (35 pack years). He died 4 years post- thoracic endovascular aortic repair/endovascular aneurysm repair from cardiovascular-related complications. (*A*) Within 14 months of thoracic endovascular aortic repair, the patient developed a 58 mm abdominal aortic aneurysm in a previously normal abdominal aorta. A 3D-CTA reconstruction demonstrated cTAG in position and the expanding abdominal aortic aneurysm. (*B*) Digital subtraction angiography (DSA) of a Gore excluder graft with proximal type Ia endoleak. (*C*) DSA of a giant Sinus XL sealing the proximal type Ia endoleak and bridging thoracic endovascular aortic repair and endovascular aneurysm repair connection.

myocardial ischaemia. The fourth patient is still complaining of SOB despite a normal coronary angiogram. The patient has been fully investigated, however, the patient has normal pulmonary function tests and haemoglobin, and no other cause of SOB has been identified.

Discussion

The best designed aortic graft is four times less compliant than the native aorta.[1,2] The failure of synthetic grafts to emulate the elasto-mechanical qualities of the native aorta is due to insufficient

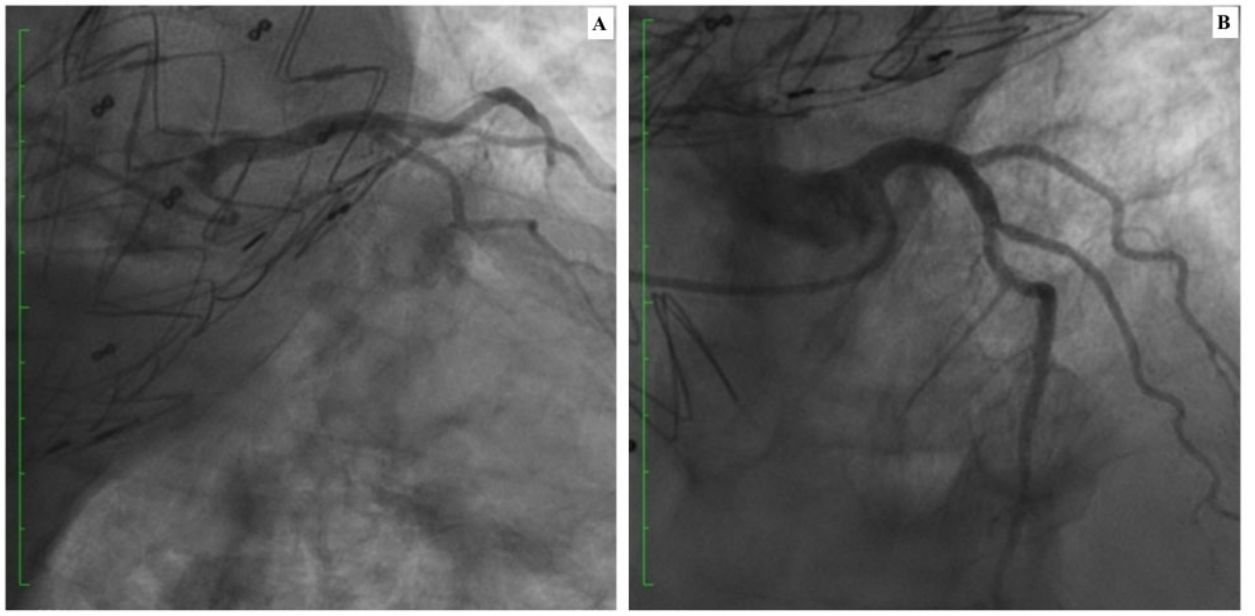

Figure 4 Coronary angiogram of the right (*A*) and left (*B*) main coronary arteries showing no signs of any obstructive coronary artery disease despite intermittent chest pain and troponin rise.

compliance, which results in a surge in haemodynamic and biological shifts that disturb the cardiovascular homeostasis.[3]

A 4D strategy for the management of complex aortic pathology is necessary for comprehensive therapy. Management of aortic pathologies is not limited to morphological adjustment and should consider the contribution of natural body forces that alter the internal haemodynamic milieu. These haemodynamic forces produce negative effects on wall shear stress and generate a milliard of adverse effects on blood flow. These adverse effects include flow displacement, turbulence, pathological vortex generation, reduction in helical blood flow, pressure gradients, and increased blood viscosity. All of these flow disturbances negatively influence the long-term management of the underlying aortic disease pathology.[4]

Rong *et al.*[5] applied intra-operative transoesophageal echocardiography to investigate the effect of synthetic graft replacement of the ascending aorta on the downstream descending thoracic aorta (DTA). They demonstrated immediate adverse effects on the DTA with increased circumferential strain and parallel increases in distensibility without adaptation in systemic haemodynamics or left ventricular stroke work. They found that replacing the native ascending aorta with synthetic graft amplifies pulse pressure and augments energy transfer to the distal aorta. They postulated that this is a potential mechanism for progressive distal aortic dilation and/or dissection. These results inform us about the intricacies of early and late graft failure and pathophysiological sequelae for other aortic segments.[1–5] This also provides a theoretical explanation for the resistant systolic hypertension that our patients developed with a subsequent SOB and intermittent chest pain. Our patients moved up at least one category in New York heart failure classification, whether functional or objective.

Aortic-endograft compliance mismatch has injurious consequences for the aorta proximal and distal to the endograft (*Figure 7A,B*). The aorta is a reservoir for 50% of the left ventricular stroke volume during systole. In diastole, the stored elastic forces of the aorta continuously thrust blood forward to the peripheral circulation. The interface between the emitted blood volume and the aorta's compliance is referred to as the Windkessel effect (*Figure 7A,B*).

The Windkessel theory essentially analogizes the distensibility of the aorta to a reservoir, which, if existing in an electrical circuit would act as a capacitor that stores energy. The walls of the aorta and other large elastic arteries contain elastin fibres which allow these arteries to distend when the blood pressure rises during systole and recoil when the blood pressure falls during diastole. Due to peripheral resistance, the rate of blood entering these elastic arteries exceeds that of leaving them. Therefore, there is net storage of blood in the aorta and large arteries during systole, which discharges during diastole.

The functional role of the aorta in the circulatory system, particularly its consequent effect on left ventricular function, has been studied in basic science,[6] animal,[7] and human clinical[8] studies. Evidence from these studies supports the Windkessel theory and relates aortic capacitance to ventricular size and function.

With TEVAR, Fenestrated EVAR, BEVAR, ChEVAR (EVAR with Chimney stents), and complex EVAR, the stented portion of the aorta loses its elasticity, and there is a consistent failure of the Windkessel effect.

The loss of the Windkessel effect and alteration of the pulse wave propagation, and its reflection, translate into a substantial workload for the left ventricle and has implications for the aortic valve's functioning. This ultimately results in adaptative hypertrophy due to failure in ventricular-arterial coupling.[9–11]

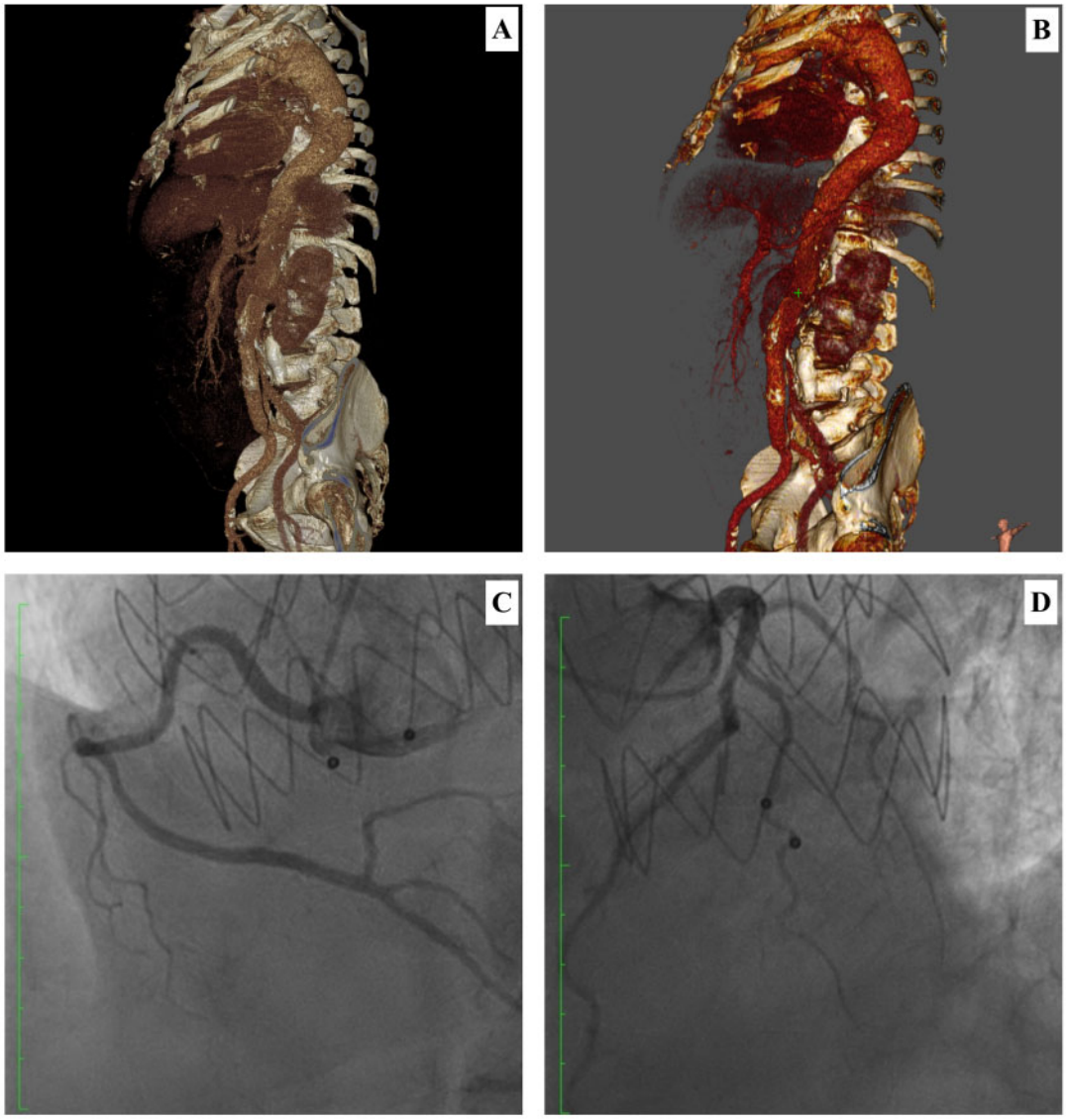

Figure 5 A 73-year-old man with proximal type B aortic dissection extending to the right common iliac artery. The false lumen was aneurysmal, 66 mm at the proximal descending thoracic aorta and 55 mm in the infrarenal abdominal aorta. He had a past medical history of hypertension, chronic kidney disease, atrial fibrillation, and smoking (25 pack years). He underwent thoracic endovascular aortic repair and subsequently endovascular aneurysm repair 6 months later. His carotids and ABI were normal. (*A*) A 3D-CTA, showing aortic dissection extending from the mid-thoracic aorta to both external iliac arteries. (*B*) Six months later, he complained of abdominal pain with left leg rest pain. A 3D-CTA demonstrated enlargement of infrarenal abdominal aortic aneurysm with compression of the left true lumen. (*C*) Coronary angiogram of the right coronary artery 12 months post-thoracic endovascular aortic repair showing no evidence of coronary artery disease, despite unstable angina and frequent chest pain with elevated troponin. (*D*) Coronary angiogram of the left coronary artery 12 months post-thoracic endovascular aortic repair showing no evidence of coronary artery disease.

Arterial stiffening yields higher systolic blood pressure and lower diastolic blood pressure, ensuring an increase in left ventricle afterload with coronary mal-perfusion. These changes also result in fatigue of arterial wall tissues. [9–16]

The resultant decline in ventricular pump effectiveness, due to negative impedance at the point of transition from the native aorta to endograft, leads to a loss of diastolic systemic blood pressure augmentation with reduced coronary flow leading to myocardial ischemia. Hence myocardial ischemia occurs even in the absence of coronary artery stenosis.[9,12]

Cardiac dysfunction was evident on the postoperative echocardiograms of our four cases, which showed mild to moderate left ventricular hypertrophy with a degree of diastolic dysfunction. The significant rise in proBNP indicates increased secretion of the hormone by cardiomyocytes in the ventricles of the heart in response to stretching caused by increased ventricular blood

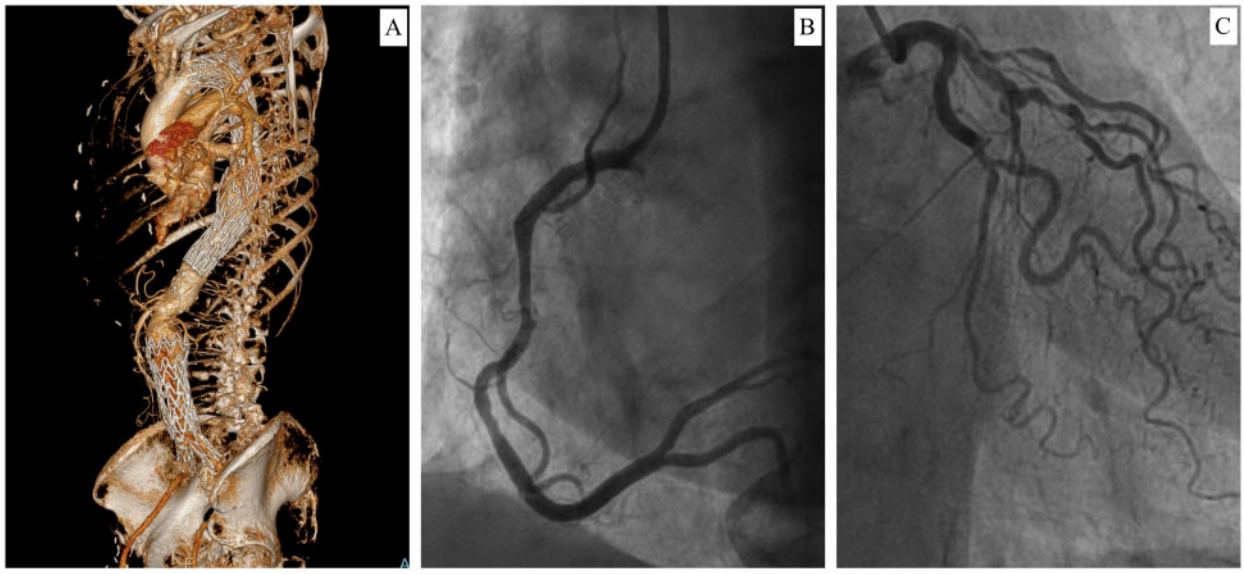

Figure 6 An 81-year-old female underwent thoracic endovascular aortic repair for a saccular descending thoracic aortic aneurysm in July 2015 using a Gore cTAG (Gore Medical, Flagstaff, AZ, USA) thoracic stent graft. She had an endovascular aneurysm repair in May 2018 for a 49 mm infrarenal saccular abdominal aortic aneurysm, which was not evident in 2015. She has a past medical history of hypertension, hyperlipidaemia, right superficial femoral artery (SFA)/popliteal/tibial angioplasty and SFA stent, and smoking (30 pack years). (*A*) A 3D-CTA reconstruction of thoracic endovascular aortic repair cTAG GORE (Gore Medical, Flagstaff, AZ, USA) using two pieces (40 mm by 20 cm) for a thoracic abdominal aortic aneurysm. Subsequently, she had an infrarenal abdominal aortic aneurysm managed by a 28 mm AFX device (Endologix, Irvine, CA, USA) distally and a 37 mm Gore cTAG (Gore Medical, Flagstaff, AZ, USA) proximally. There was no evidence of endoleak with total modulation of the aorta. The patient complained of on and off chest pain on minimal exertion and shortness of breath. (*B*) Coronary angiogram of the right main coronary artery showing no evidence of coronary occlusive disease. (*C*) Coronary angiogram of the left main coronary artery showing no evidence of coronary occlusive disease.

volume. It is, therefore, a reflection of increased ventricular workload.

The significant elevation in troponin indicates cardiac muscle ischaemia due to reduced coronary blood flow. In these patients, the reduction in blood flow was not due to coronary artery stenosis, as confirmed by coronary angiography, but rather due to under perfusion of the coronary vessels due to reduced diastole pressure.

The integrity of the aorta has been shown to adversely affect cardiovascular outcomes. Late rehospitalization following discharge in acute aortic syndrome is attributed to cardiovascular complications in about one-third of the patients.[17,18] Weiss *et al.*[18] have shown a two- to three-fold higher risk of non-aortic cardiovascular death, including the new occurrence of non-fatal cardiovascular event and heart failure in patients with aortic dissection, intramural haematoma, and penetrating aortic ulcer. These results emphasize the need for long-term cardiovascular follow-up and management.

After combined TEVAR and EVAR, the compliance mismatch and loss of elasticity are even more pronounced than with TEVAR alone. A compensatory mechanism results, whereby, the endograft adapts by altering its fabric's yarn architecture. This compensation has been demonstrated following open surgical repair, after which graft material adaptations result in gradual dilatation at 3.2% per year post-implantation.[13,14] This is accredited to the constant strain and the excessive stress on the suture lines of the noncompliant grafts, with subsequent development of pseudoaneurysms.[9,12] None of our cases developed pseudoaneurysm post-TEVAR, but they did develop infrarenal AAA that required repair with EVAR.

Pulse wave velocity (PWV) correlates with arterial stiffness; the higher the PWV, the greater the arterial stiffness. Pulse wave velocity < 9.4 m/s is associated with a routine cardiovascular risk, whereas a PWV of 9.4–12 m/s has a five-fold increased risk, and PWV above 12 m/s has a six-fold increased risk of cardiovascular morbidity and mortality. The increase in PWV can occur within a few hours of implanting a TEVAR or EVAR.[15,16,19] Blacher *et al.*[15] acknowledged that each PWV increase of 1 m/s double the rate of all-cause mortality. TEVAR increases the PWV 2–5 m/s, EVAR increases the PWV by 1–3 m/s, whereas a combined TEVAR and EVAR will increase PWV by 3–8 m/s, which has a negative determinant effect on cardiovascular morbidity and mortality.[15,16,19] All of our patients developed adaptive left ventricular hypertrophy with diastolic dysfunction, manifested clinically with bilateral lower limb oedema, shortness of breath, and chest pain. Although one would expect cardiac dysfunction to relate to CAD and be a consequence of the risk factor these patients have which predispose them to CAD, all of these patients had a coronary angiogram which demonstrated normal coronary arteries.

Endovascular aneurysm repair for AAA differs from TEVAR vis-à-vis the immediate increase in aortic stiffness.[20] The Windkessel effect is attenuated by TEVAR combined with EVAR, as the elastic

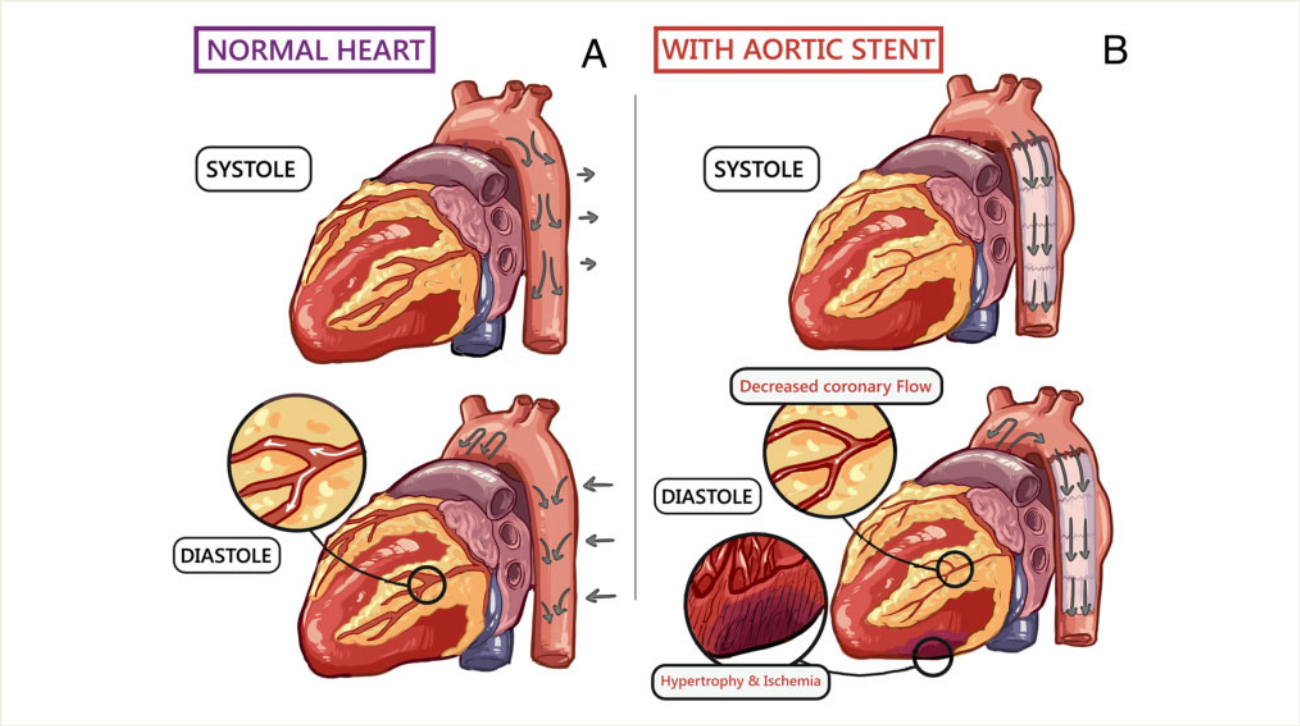

Figure 7 A schematic diagram of the heart showing: (*A*) Normal ventricular coupling and easy filling of the coronaries during diastole. (*B*) Post-thoracic endovascular aortic repair in descending aorta, which induces immediate increases in arterial stiffness and systolic hypertension, resulting in left ventricular hypertrophy, and compromises filling with an increase in end-diastolic pressure, resulting in an isolated left ventricular diastolic dysfunction, with the clinical features of heart failure post-implantation. Patients will develop non-occlusive coronary ischemia with major cardiovascular consequences due to a decrease in coronary flow.

properties are removed from both arterial segments. Currently, all commercially available endografts are noncompliant.[6,21] Three of our patients developed a AAA after having TEVAR in infrarenal aortas, which were not aneurysmal before TEVAR implantation (*Figures 1A*, *3A*, *5B*, and *6A*). Abdominal aortic aneurysm development is likely a consequence of amplified aortic wall stress and the activation of aortic cell molecular dysfunction.

The Liapis group[22] demonstrated that polyester-based aortic endografts cause a threefold increase in PWV compared to PTFE-based platforms. All four patients in the current series had PTFE-based EVAR technologies (one Gore Excluder and three Powerlink Pro/AFX Endologix) to prevent further increase in PWV.

This is an isolated series of four patients and while the rate at which they developed abdominal aortic disease progression was alarming, worsening hypertension, and late cardiovascular complications could be due to their underlying cardiovascular risk factors. To substantiate the theory that TEVAR/EVAR causes hypertension and cardiovascular complications late after placement, future studies will need to be undertaken which specifically address cardiovascular complications. It is likely that evidence already exists within large-scale registries, which could be interrogated with propensity matched-controls, for example.

Conclusion

All of our four cases developed cardiac dysfunction following TEVAR/EVAR. This vicious circle of events could be attributed to the adverse haemodynamic effects of the aorta's excessive metallic lining. The consequent aortic stiffness leads to cardiovascular adverse events, even in the absence of direct aortic-related complications.

Lead author biography

Prof. Sherif Sultan is a pioneering vascular/endovascular surgeon and the founder of the Western Vascular Institute, Ireland, a charitable research foundation committed to vascular research, technical innovation, and education. He is the current president of the International Society of Vascular Surgery and the American Society of Angiology, Irish chapter. He had pioneered the techniques of sub-intimal angioplasty, DRESS technique for complex EVAR, Kinetic elephant trunk for pan aortic dissection,

TIGER protocol for infra-diaphragmatic aortic dissection, triple neuroprotection for patients with acute vascular stroke post the 24 h opportunity window, and Art-assist for critical limb ischaemia patients.

Supplementary material

Supplementary material is available at *European Heart Journal—Case Reports* online.

Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report, including images and associated text, has been obtained from the patients in line with COPE guidance.

Conflict of interest: None declared.

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