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ORIGINAL ARTICLE

Pathology of stent implantation in internal mammary artery

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Abstract
The internal mammary artery (IMA) is the most durable conduit for bypassing the left anterior descending (LAD) coronary artery in patients undergoing coronary artery bypass graft surgery (CABG). However, little is known about how the IMA reacts histologically to stent implantation. From CVPath stent registry (1048 lesions, 614 cases), we obtained 4 stent lesions (2 bare metal stents, 2 drug-eluting stents) involving IMA grafts. The mean age of our patients was 63 years and the duration of stent implantation in the IMA ranged from 5 days to 5 years. Stented arteries were dissected from hearts and embedded in plastic, segmented at 3 mm intervals, sectioned at 4–6 microns and stained with H&E and Movat pentachrome stains. Histological observations were performed. Majority of stents (3 of 4) were implanted in anastomosis between IMA and LAD while 1 stent was implanted in IMA body. One stent with duration of 5 days showed stent thrombosis while others were all patent with fully coverage by varying degrees of neointima. Foamy macrophage, lipid pool and calcification in neointima were observed in 1 stent with duration of 5 years but it was limited only to the distal LAD part within the stented segment. Overall, in this small pathologic series, the majorities of stents were implanted in IMA-to-LAD anastomosis site and demonstrated acceptable pathologic responses.

Keywords Coronary artery bypass graft · Internal mammary artery · Graft failure · Stent · Anastomosis

Introduction

The internal mammary artery (IMA) is the most durable conduit for bypassing the left anterior descending (LAD) coronary artery in patients undergoing coronary artery bypass graft surgery (CABG) [1]. The IMA-to-LAD graft has greater longevity than other arterial or venous grafts for the treatment of LAD diseases with patency rates > 90% at 5-year follow-up [2, 3]. Owing to the high patency rate of IMA graft following CABG, it has also been shown to be superior to revascularization of LAD by percutaneous coronary intervention (PCI) performed with bare metal stents (BMS), or drug-eluting stents (DES) [4–7]. However, the pathological causes of IMA graft failure and the response of the IMA to stenting remain understudied.

We reviewed CVPath Registry of human stents to determine the pathological response following stent implantation in subjects with IMA grafts.

Methods

From CVPath stent registry ($n = 614$ patients, $n = 1048$ lesions), we identified 4 cases where stenting of the IMA had been performed. The mean age of our patients was 63 years and the duration of stent implant in the IMA ranged from 5 days to 5 years (Tables 1 and 2). All available clinical records were reviewed for patient history, duration of implantation, coronary risk factors and cause of death. Following an autopsy, stented IMA segments were embedded in plastic (methyl methacrylate). Histological sectioning was performed at 3 mm intervals and sections stained with Hematoxylin and Eosin, and modified Movat Pentachrome.

To understand differences in the histological response to stenting between the IMA and the native LAD, pathological assessment was separated into stent response in the IMA and distal LAD when a stent was implanted at

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the anastomosis. Underlying plaque was assessed based on modified American Heart Association (AHA) classification [8]. Lesion calcification was graded as none, mild, moderate or severe based on radiograph in each segments [9]. Inflammatory reaction to stent was graded as none, mild, moderate or severe. Nature of neointima was assessed as "smooth muscle cell (SMC) > collagen (CLG)", "SMC = CLG", or "SMC < CLG" based on the composition as previously described [10]. Components of in-stent atherosclerotic change (neoatherosclerosis) such as lipid pool, foamy macrophage, necrotic core or neointimal calcification were also described [11].

Morphometric measurements were accomplished with image analysis software (Zen2, blue edition, Carl Zeiss, Oberkochen, Germany) after digital scanning, which was separated into IMA, anastomosis and distal LAD. Internal elastic lamina area, stent area, underlying plaque area and neointimal area (only for cases with duration > 30 days) were included.

Results

Patient demographics and lesion characteristics are shown in Tables 1 and 2. Pathological features and morphometric analysis are summarized in Tables 3 and 4. These details are also described within the pathological finding described in the following paragraphs.

Case 1

A 68-year-old male with history of severe coronary artery disease (CAD), congestive heart failure (CHF), hypertension, hyperlipidemia, diabetes mellitus, hypothyroidism, and cerebrovascular accident with residual right-sided weakness

Table 1 Patient characteristics

	Case 1	Case 2	Case 3	Case 4
Age	68	76	66	43
Gender	Male	Male	Male	Male
Cause of death	Stent thrombosis	Aortic stenosis	Cardiac arrhythmia	Heart failure

Table 2 Lesion characteristics

	Case 1	Case 2	Case 3	Case 4
Duration	5 days	1 year	2 year	5 year
Lesion location	Anastomosis	Body	Anastomosis	Anastomosis
Stent type	Taxus	AVE	Taxus	Bx Velocity
Stent size	2.75 × 20 mm	4 × 15 mm	3 × 18 mm	2.5 × 15 mm
Stent fracture	None	None	None	None
Stent outcome	Subacute thrombosis	Patent	Patent	Patent

Table 3 Pathological feature

	IMA/LAD	Case 1	Case 2	Case 3	Case 4
Underlying plaque type	IMA	Normal	Normal	Normal	Normal
	LAD	Fibroatheroma	NA	Fibrocalcific plaque	Pathological intimal thickening
Underlying calcification	IMA	None	None	None	None
	LAD	Mild	NA	Moderate	None
Inflammation	IMA	Mild	Mild	Mild	Mild
	LAD	Mild	NA	Mild	Mild
Neointimal character	IMA	NA	SMC = CLG	SMC = CLG	SMC = CLG
	LAD	NA	SMC < CLG	SMC < CLG	SMC < CLG
Neoatherosclerosis	IMA	NA	None	None	None
	LAD	NA	NA	None	Foamy macrophage
					Lipid pool microcalcification

IMA internal mammary artery, *LAD* left anterior descending artery, *NA* not applicable, *SMC* smooth muscle cells, *CLG* collagen

Table 4 Morphometric analysis

	IMA/Anastomosis/LAD	Case 1	Case 2	Case 3	Case 4
Internal elastic lamina area (mm^2)	IMA	7.3	9.4	4.7	3.0
	Anastomosis	10.9	NA	8.4	6.0
	LAD	8.3	NA	5.1	4.5
Stent area (mm^2)	IMA	6.7	9.2	4.5	2.5
	Anastomosis	6.1	NA	4.6	3.1
	LAD	4.9	NA	4.1	3.3
Underlying plaque area (mm^2)	IMA	0.6	0.2	0.2	0.5
	Anastomosis	4.7	NA	3.8	2.9
	LAD	3.4	NA	1.0	1.2
Neointimal area (mm^2)	IMA	NA	2.6	1.3	1.8
	Anastomosis	NA	NA	1.9	2.1
	LAD	NA	NA	2.6	1.8
Luminal narrowing (%)	IMA	NA	28%	28%	74%
	Anastomosis	NA	NA	41%	67%
	LAD	NA	NA	63%	56%

Abbreviation as Table 3

had undergone multivessel coronary artery bypass surgery 19 years earlier. The left internal mammary artery (LIMA) had been anastomosed in the LAD, and saphenous vein (SV) grafts had been utilized to bypass the right coronary artery (RCA,) obtuse marginal and the left diagonal. The patient had also had frequent percutaneous coronary interventions (PCI) since his bypass for symptomatic CAD.

He had presented to the emergency room with chest pain and a diagnosis of inferior ST elevation myocardial infarction was established, and he underwent cardiac catheterization. Native coronary angiography revealed 75% stenosis in left main coronary artery, total occlusions in the LAD ostium, proximal RCA and mid left circumflex (LCX). All saphenous vein bypass grafts were occluded and there was severe stenosis at the LIMA-LAD anastomosis. The patient had a poor left ventricular function with an ejection fraction (EF) of 30%. A Taxus stent (2.75 × 20 mm, Boston Scientific, MA) was implanted in the LIMA-LAD anastomosis and an intra-aortic balloon pump was placed. Hospital course was complicated by pneumonia with respiratory failure, acute renal failure secondary to contrast nephropathy and hyperbilirubinemia secondary to congestive heart failure. Five days after admission, he developed hypotension, requiring intravenous infusions of dopamine, vasopressin, and dobutamine. Despite maximal support, he died of cardiac arrest.

In autopsy, his heart showed mild left ventricular hypertrophy (heart weight 450 g), and acute and old multiple myocardial infarcts. Histologic assessment of the stent in LIMA-LAD anastomosis showed an occlusive platelet-rich thrombus (Fig. 1). Minimal peri-strut fibrin deposition, and acute and chronic inflammation were observed. Underlying plaque at anastomosis site was a fibroatheroma with calcification and a fibrous cap disrupted during stenting, which was the contributing factor for the stent thrombosis. Underlying plaque was minimal in IMA.

Case 2

A 76-year-old male with history of CAD, aortic stenosis, hypertension, and end-stage renal disease had been treated with CABG (LIMA-LAD, vein graft anastomosis to distal RCA, and ramus intermedius). AVE stent (4 × 15 mm, Medtronic, CA) had been placed in the body of the LIMA 1 year prior due to recurrent symptomatic CAD. The patient had a fall and sustained abrasions and laceration to the arms and legs, respectively, along with bruises to the chest. Three days later, he complained of chest pain followed by 2 syncopal episodes. He presented to emergency department with hypotension and left bundle branch block, became unresponsive, and died.

In autopsy, the heart revealed cardiomegaly (650 g) with concentric left ventricular hypertrophy, no gross myocardial fibrosis or necrosis. The aortic valve was moderately calcified. The native coronary circulation was right dominant and showed severe calcification in main arteries. All bypass grafts were patent. In Fig. 2, histologic sections of stent in LIMA showed minimal neointimal formation and focal peri-strut fibrin with mild peri-strut chronic inflammation and neoangiogenesis. There was almost no underlying plaque in the IMA implying perhaps the presence of spasm.

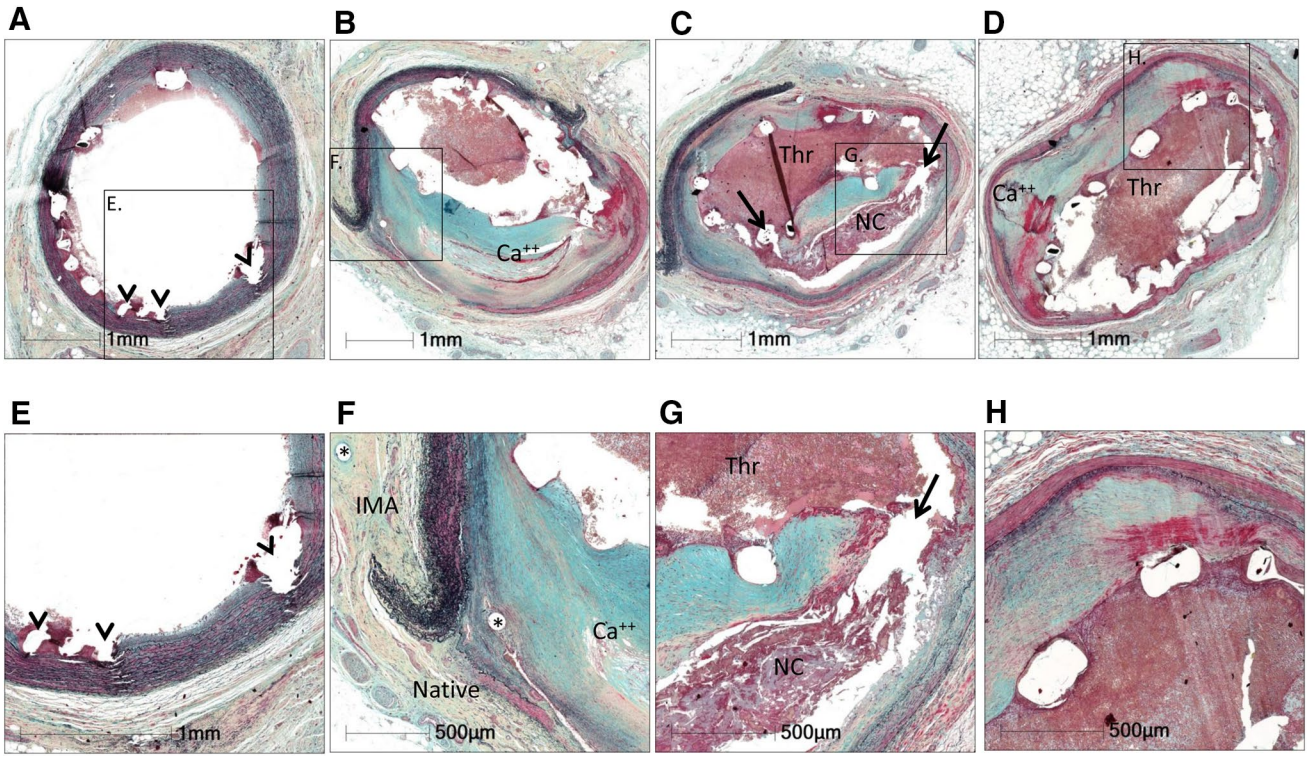

Fig. 1 **a**–**d** Histologic proximal to distal sections of the LIMA-LAD anastomosis from case 1. **e**–**h** High power images from boxed area from **a** to **d**. Occlusive thrombus (Thr) is observed in **c**, **d**. Arrowheads in **a** and **e** show mild medial disruption in the proximal IMA. **b**, **c** The anastomosis site, which is further highlighted in **f**. Half of the arterial wall is IMA (upper) and the other half (lower) is native coronary artery (**b** and **c**). Asterisk (*) in **f** indicates surgical suture. Calcification (Ca^{++}) is observed in underling plaque only in the native coronary artery at the anastomosis in **b** and **f**. Necrotic core (NC) with disrupted fibrous cap (arrows) was observed only in the native coronary artery at the site of the anastomosis in **c** and **g**. Occlusive thrombus with underlying calcification is observed in native distal LAD (**d** and **h**)

Case 3

A 66-year-old male with history of severe CAD, hypertension, and smoking had been treated with CABG 10 years earlier (LIMA-LAD, SV-distal RCA, SV-obtuse marginal and SV-left diagonal). A paclitaxel-eluting stent (3 × 18 mm, Boston Scientific, MA) had been placed in LIMA-LAD anastomosis 2 years prior, due to symptomatic CAD. The patient collapsed suddenly, paramedics found the patient in ventricular fibrillation but could not resuscitate him.

In autopsy, the heart showed cardiomegaly (690 g) with healed transmural infarctions in anterior and lateral left ventricle. Native coronary arteries showed severe luminal narrowing > 95% of the LAD, LCX and RCA. LIMA had been grafted to the LAD and a saphenous vein bypass grafts to the distal RCA was patent, while grafts to the obtuse marginal and the left diagonal were occluded. In Fig. 3, histologic sections of the stent in LIMA-LAD anastomosis showed mild neointimal proliferation with fibrin around stent struts and peri-strut chronic inflammation. Underlying plaque was fibrocalcific plaque in native coronary artery at the site of anastomosis, while there was almost no plaque in IMA.

Case 4

A 43-year-old male with history of severe CAD, CHF, smoking and pulmonary hypertension had been treated with coronary artery bypass graft 10 years earlier with LIMA to the LAD and saphenous vein graft to the left diagonal. He had a Bx Velocity stent (2.5 × 15 mm, Johnson&Johnson, NJ) implanted in the IMA 5 years earlier for symptomatic CAD. His CHF had been treated with cardiac resynchronized therapy with defibrillator and a left ventricular partitioning device. He was in severe heart failure with an LVEF of 14%, and was admitted to the hospital 11 days prior to death. Initially, the patient was managed with pressors and diuretics, but had complications of left upper extremity deep vein thrombosis and suffered acute cardiopulmonary arrest. He was resuscitated, required intubation and extracorporeal membrane oxygenation (ECMO). His condition further deteriorated with multi-organ failure (MOF), anoxic brain injury, and death.

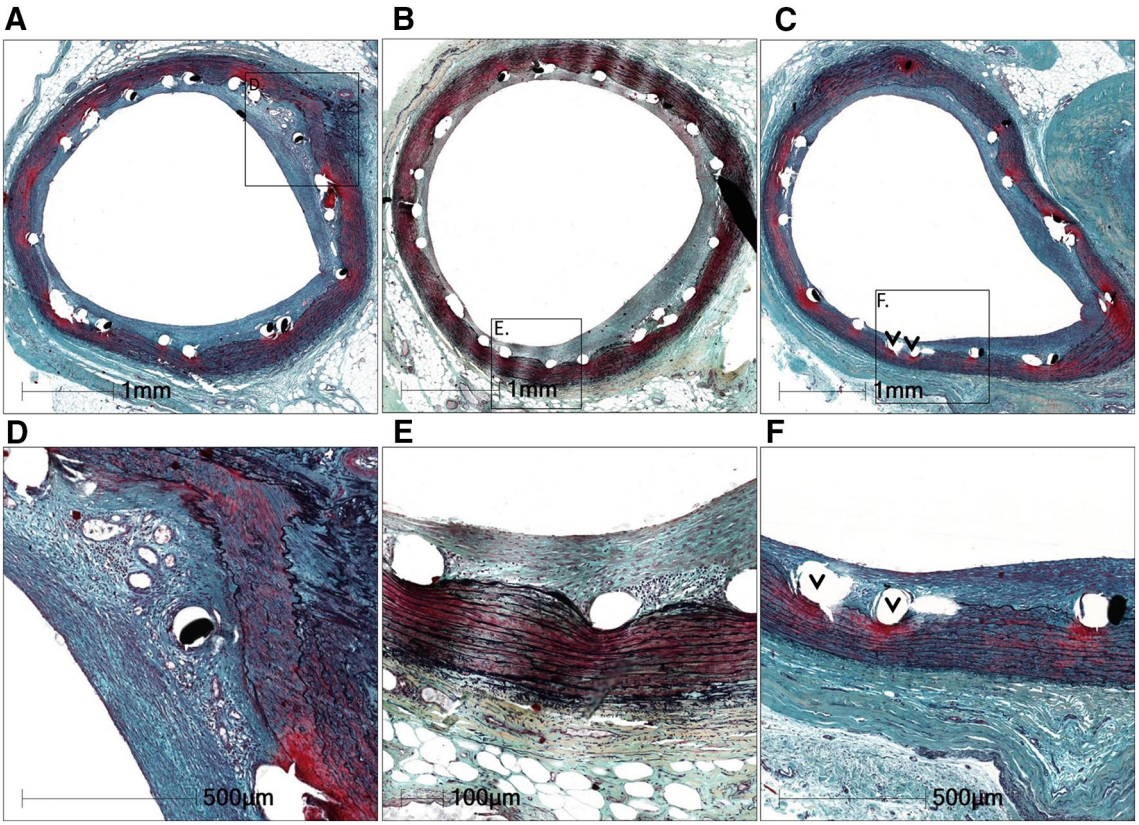

Fig. 2 **a**–**c** Proximal to distal histologic sections of stent in LIMA body from case 2. **d**–**f** High power images from boxed area in **a**–**c**. Focal inflammation with or without neovascularization is observed in **d** and **e**. There is mild medial disruption (arrow head) in **c** and **f**

In autopsy, his heart weighed 846 g, there was severe left ventricular hypertrophy, and aneurysmal dilatation with calcification of the anteroseptal wall. The myocardium showed healed myocardial infarctions involving the anteroseptal, lateral and posterior left ventricular walls. Histologically, an inflammatory infiltrate was observed consisting of eosinophilic infiltrate, macrophages, and giant cells surrounding an eosinophilic material consistent with hypersensitivity myocarditis. His native coronary artery exhibited total occlusion in LAD, 70% stenosis in LCX and 70% stenosis in the RCA. The vein graft anastomosed to the left diagonal was occluded while the LIMA to the LAD was patent. Histologic sections of the stent in LIMA-LAD anastomosis showed moderate neointimal proliferation with smooth muscle cell-rich neointima leading to a 68% area stenosis of the lumen (Fig. 4). Medial disruption by the stent struts was observed and there was focal chronic inflammation with giant cells and moderate angiogenesis around struts. Underlying native plaque showed pathological intimal thickening with minimal calcification at the anastomotic site, while it was scarcely observed in IMA.

Overall pathological response in IMA and LAD

Overall, there was almost no underlying plaque as well as calcification in IMA while these were mainly observed in distal LAD. Neointimal smooth muscle cells were relatively rich in IMA compared to distal LAD. Neoatherosclerotic change was observed only in one case but it was limited in distal LAD part of stented segment.

Discussion

To the best of our knowledge, this is the first study to report the pathologic findings of stent implantation in the IMA. Majority of lesions (3 of 4) were located at the anastomosis site while one lesion was located in IMA body. Underlying plaque mainly existed in distal LAD while it was minimally observed in IMA. Neointimal smooth muscle cells were relatively rich in IMA than in distal LAD. Neoatherosclerosis was observed only in distal LAD part of stented segment while it was not observed in IMA part.

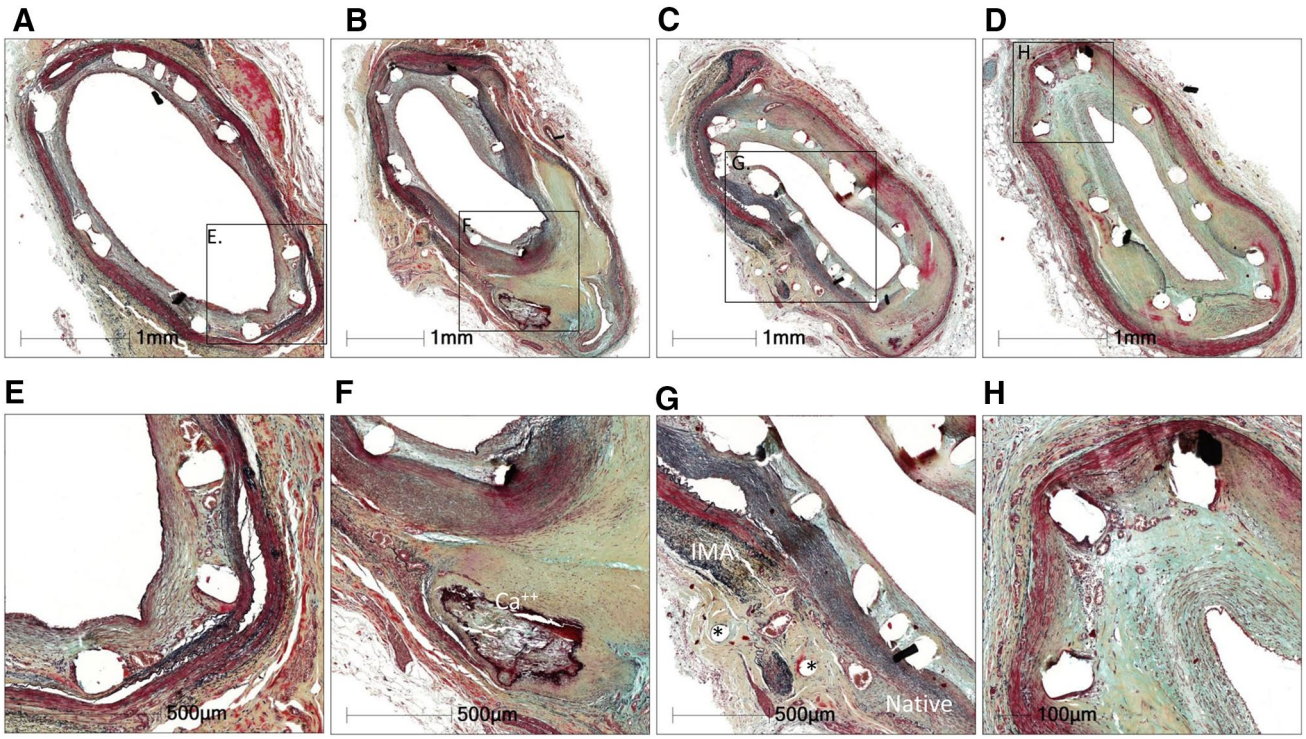

Fig. 3 **a**–**d** Proximal to distal histologic sections of stent in LIMA-LAD anastomosis from Case 3. **e**–**h** High power image from boxed area in **a**–**d**. **a** Proximal IMA and boxed area (**e**) shows some inflammation with neovascularization. **b**, **c** The anastomosis site. Underlying plaque was fibrocalcific plaque mainly in the native coronary artery at the site of the anastomosis (**f**). **g** High power image of anastomosis site with surgical suture (*). **d** Distal native LAD. **h** Mild inflammation with neovascularization

Feasibility of IMA Stenting

IMA stenting appeared to be feasible with satisfactory pathologic responses in this study. The IMA is a protected artery because of its ability to withstand surgical trauma, its musculoelastic nature, and its smaller diameter which is better matched to the native LAD. There are several biological reasons why IMA stenting is feasible. One of the reasons we believe is that the endothelium of the IMA has fewer fenestrations and lower intercellular junction permeability, which likely prevents lipoproteins from entering the subendothelial space [12]. Also, the endothelial cells of the IMA are rich in heparin sulfate and endothelial nitric oxide synthase compared to veins, which leads to antithrombotic properties allowing endothelial homeostasis, conferring greater protection from atherosclerosis [12, 13]. Moreover, the expression of endothelial nitric oxide synthase produces low concentrations of nitric oxide, which preserves endothelial function and integrity, and allows arterial remodeling (enlargement of arterial lumen or limited intimal thickening and less vessel lumen loss). We believe that these unique features prevent atherosclerosis development in IMA as well as in neointima following IMA stenting as shown in our cases.

Clinical feature of IMA graft failure

Frequency of IMA graft failure is not high (< 10%) and was reported to be 8.6% at 12–18 month angiographic follow-up in a recent study by Harksamp et al. [14]. However, the prognosis of patients with IMA graft failure is significantly worse than that of patients without IMA graft failure. In the same study [14], the event rate for the composite of death, myocardial infarction, or repeat revascularization over 4 years was reported to be 32.0% for the patients with IMA graft failure (as detected by angiographic follow-up) versus 16.5% for the patients without IMA graft failure ($p < 0.0001$). Therefore, it is important to understand why IMA grafts fail and how best to treat these failed conduits.

Shiono et al. reported impact of functional focal versus diffuse CAD on bypass graft patency [15]. Using the pressure wire pull back data, the LAD lesions were classified into functional focal disease (abrupt pressure step-up) or functional diffuse disease (gradual pressure increase) [15]. In follow-up computed tomography angiography within 1 year, occlusion or string sign of the IMA graft to LAD was more frequently observed in functional diffuse disease group versus in the functional focal disease group (26 vs. 7%, $p = 0.02$) [15]. Our cases with anastomosis stenting

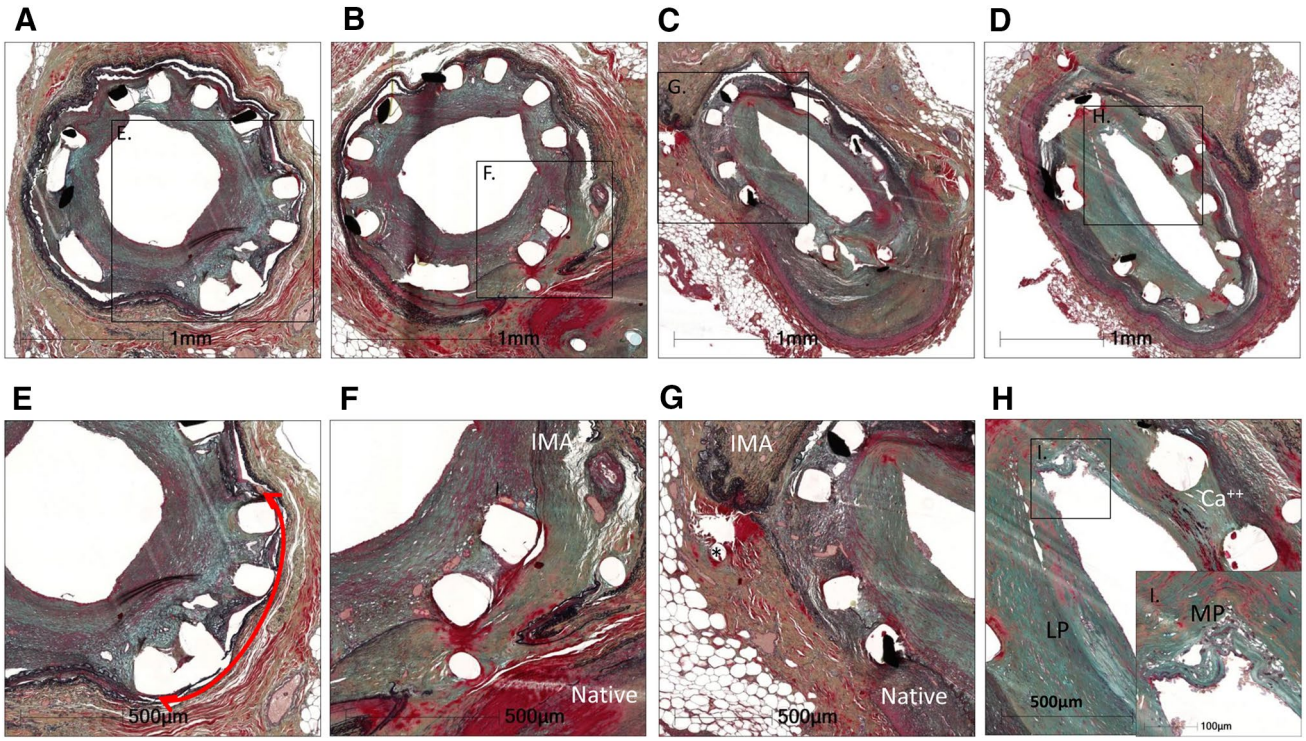

Fig. 4 **a**–**d** Proximal to distal histologic sections of stent in LIMA-LAD anastomosis from case 4. **e**–**h** High power images from boxed area in **a**–**d**. **i** (boxed inset in **h**) Even higher power image from **h**. Struts were fully covered with relatively thick neointima in **a**–**d**. **a** Medial disruption of proximal IMA which is highlighted in red double headed arrow in **e**. **b**, **c** (high power images in **f** and **g**) Anastomosis site with surgical suture (*). Underlying plaque was pathological intimal thickening mainly in native coronary artery at the site of LIMA anastomosis (**c**). Foamy macrophage (MP in **i**), Lipid pool (LP in **h**) and microcalcification (Ca^{++} in **h**) are observed in neointima of distal LAD (**d**, **h** and **i**)

are likely to be functionally diffuse disease because substantial amount of underlying plaque was observed in distal LAD. Another thing that may be worth commenting on is that anastomosis site in case one was not ideal because of underlying fibroatheroma. If intravascular imaging could be applied to determine the anastomosis site during surgery, such issues might be avoidable.

Pathologic comparisons of LIMA and vein graft interventions

It is well known that graft failure is more common in SV vs. IMA. Aggarawal et al. reported safety and effectiveness of DES versus BMS in SV bypass graft PCI [16]. In this large study using Veterans Affairs integrated health system in USA, PCIs in SV grafts accounted for 8–10% of all PCIs from 2008 to 2011 [16]. Lesion location was most frequent in body followed by aorto-ostial location and anastomosis (65, 21, 11%) in SV graft failure, which was very different from IMA as lesion location was most frequent at the anastomosis followed by body and proximal for IMA graft failure (81, 13, 6%) [16–18]. Death during SV graft PCI procedure was as low as 0.04% (1 of 2471) [16]. No reflow or slow flow was observed in 3.3% at final angiography [16]. Long-term mortality at 2 years was better in DES than in BMS (15, 21%, $p < 0.01$), which is worse than that reported for IMA [16, 18].

The healing process following stent implantation in IMA seems better compared to VG because evidence of accelerated neoatherosclerosis in vein grafts has been reported to be as high as 39% (33% in BMS and 44% in DES) at 1 year [19]. In current study, only 1 case showed the evidence of neoatherosclerosis. However, it was limited in distal LAD part within stented segment. This can be due to the underlying disease in vein graft which is friable atherosclerotic disease that is not only prone to distal emboli but also prolapse of necrotic core into the lumen. Also, the specific character of neointimal smooth muscle cell in IMA could potentially be protective for neoatherosclerosis although this is purely speculative as the mechanism of neoatherosclerosis is not fully elucidated [11].

Limitations

This is a small autopsy study involving a small number of complex cases, and therefore may not be applicable to everyday clinical practice. However, there are no other histologic studies of IMA stenting that been reported.

Conclusion

Overall, stent implantations in IMA including anastomosis site is feasible and the review of literature suggests the clinical results are better than repeat CABG for both mortality and patency. The majority of cases shown here had satisfactory responses to stent implantation.

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Compliance with ethical standards

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Conflict of interest CVPath Institute has research Grants from Abbott Vascular, Atrium Medical, Boston Scientific, Biosensors International, Cordis-Johnson&Johnson, Medtronic CardioVascular, OrbusNeich Medical and Terumo Corporation. H. M has received honorarium from Abbott Vascular Japan, Goodman and Terumo Corporation. S. T has received honorarium from Abbott Vascular Japan, Terumo Corporation and SUNRISE lab. R. V has speaking engagements with Merck; receives honoraria from Abbott Vascular, Boston Scientific, Lutonix, Medtronic and Terumo Corporation and is a consultant for 480 Biomedical, Abbott Vascular, Medtronic and W. L. Gore. A. V. Finn has sponsored research agreements with Boston Scientific and Medtronic CardioVascular and is an advisory board member to Medtronic CardioVascular. The other authors report no conflict.

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