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A Case series of Gel Foam Embolization for Distal Coronary Artery Perforation

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Abstract

Coronary artery perforation is a rare but potentially catastrophic complication of percutaneous coronary intervention (PCI), often associated with complex lesions such as chronic total occlusions. This case series presents two distinct clinical scenarios of distal coronary perforation managed successfully with gel foam embolization. The first case involved a 49-year-old male with in-stent restenosis of the right coronary artery, who developed an Ellis Class II perforation during PCI, promptly sealed with gel foam via a microcatheter. The second case featured a 64-year-old male with a long-segment RCA CTO who developed delayed cardiac tamponade due to distal PDA perforation, requiring prolonged pericardial drainage before definitive embolization. In both cases, gel foam proved to be an effective, accessible, and economical alternative to coils or covered stents, particularly in distal vessel injuries. These cases underscore the importance of guidewire technique, vigilant post-procedural monitoring, and timely intervention. They highlight gel foam embolization as a practical option in managing select cases of coronary perforation, especially in resource-limited settings.

Keywords: Coronary artery perforation, Percutaneous coronary intervention, Distal vessel perforation, Gel foam embolization, Cardiac tamponade, Chronic total occlusion

Introduction

A rare but possibly fatal side effect of percutaneous coronary intervention (PCI) is coronary perforation. It is typified by iatrogenic damage and the subsequent rupture of the coronary artery wall, which leads to blood either draining into a neighbouring heart chamber or accumulating outside the vessel usually in the pericardial region.(1, 2) Percutaneous coronary intervention is increasingly being employed to manage complex and calcified coronary lesions, which are inherently associated with a higher risk of procedural complications, including coronary perforation. The incidence of perforation during PCI varies across different patient populations and clinical settings.(3) However, a meta-analysis involving a pooled cohort of 197,061 patients estimated the overall incidence of coronary perforation to be approximately 0.43%, reflecting the rarity but significant clinical impact of this complication.(4) Prolonged balloon inflation, gel foam embolization, or thrombogenic metallic coil embolization are the primary methods used to seal small distal vessel perforations. The use of covered stents or prolonged balloon inflation are the current recommendations for treating perforated coronary arteries. The diameter of covered stents ranges from 2 to 2.25 mm. When a vessel less than 2 mm perforates, there is a danger of late tamponade after 3–6 hours, which presents a significant problem. In order to establish haemostasis and stop bleeding, autologous clot or fat, gel foam,

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fibrin glue, microcoils, and polyvinyl alcohol embolization are used to treat coronary perforation in tiny vessels with a diameter of less than 2 mm.(5, 6) This case series highlights two unique clinical presentations of coronary artery perforation, both effectively managed using gel foam embolization as a definitive treatment strategy.

Case presentation

Case 1: A 49-year-old male with a prior history of ischemic heart disease and single-vessel right coronary artery (RCA) disease, who had undergone PCI with a 3.00 × 32 mm stent in 2018, presented with effort intolerance (NYHA class III) for the past two months. On examination, his vital signs were stable. ECG showed sinus rhythm with Q waves in leads II, III, and aVF, and echocardiography revealed hypokinesia of the basal and mid-inferior wall with preserved left ventricular systolic function (ejection fraction 55%). Coronary angiography demonstrated a normal left main coronary artery (LMCA); the left anterior descending (LAD) artery was a Type III vessel with an 80% mid-segment lesion and provided collateral supply to the RCA, while the non-dominant left circumflex (LCX) artery also contributed retrograde collaterals to the RCA. The RCA was dominant and showed mid-segment in-stent restenosis with distal TIMI I flow; the posterior descending artery (PDA) was totally occluded and was supplied by collaterals from both the LAD and LCX. A decision was made to proceed with PCI to the RCA (DEB) and LAD, and a JR 3.5 guiding catheter with a Fielder-XT guidewire was used to cross the lesion; however, during guidewire manipulation in the RCA, distal coronary perforation was noted. Angiography classified this as an Ellis Class II perforation—pericardial or myocardial blush without a ≥1 mm exit hole (8). Immediate echocardiography showed no pericardial effusion, but given that distal perforations are prone to delayed pericardial effusion and tamponade, the team decided to seal the perforation; after considering covered stent, coil embolization, autologous blood clot or fat particle embolization, and gel foam embolization, gel foam was selected and delivered through a 4F Carvel microcatheter. The patient remained hemodynamically stable post-intervention, and on Day 3, repeat RCA angiography showed no residual leak. Subsequently, successful PCI with drug-eluting stent (DES) placement to the mid-LAD was performed. This case highlights that although coronary perforation is a feared catheterization laboratory complication, prompt recognition and tailored management using readily available materials like gel foam can lead to favourable outcomes, and it underscores the importance of meticulous wire manipulation, particularly in chronic total occlusion (CTO) and other complex lesions, in both preventing and managing such complications.

Case 2: A 64-year-old male with known hypertension on regular medication for four years presented with chest pain persisting for 10 days. His electrocardiogram showed an evolved anterior wall myocardial infarction, and echocardiography revealed regional wall motion abnormalities in both the anterior and inferior walls with preserved left ventricular systolic function (ejection fraction 50%). Coronary angiography demonstrated a critical 90% ostioproximal lesion in the left anterior descending (LAD) artery, an 80% lesion in the distal left circumflex (LCX) artery, and a long-segment chronic total occlusion (CTO) of the right coronary artery (RCA) with distal filling via both homo-collateral and contralateral circulation. Although coronary artery bypass grafting (CABG) was advised, the patient refused surgery; therefore, PCI to both the LAD and RCA was performed. Four hours after the procedure, he developed hypotension and was found to have cardiac tamponade, prompting emergency pericardiocentesis that aspirated 240 ml of fresh blood. Repeat angiography of the RCA revealed perforation in a small distal branch of the posterior descending artery (PDA), while

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

LAD angiography was unremarkable. Despite initial drainage of 150 ml that accumulated gradually within 24 hours, there was gradual reaccumulation of pericardial effusion over the next 3–4 days with daily aspirations of 120–160 ml, and there were no signs of spontaneous closure of the leak; in view of ongoing haemorrhagic effusion, ticagrelor was switched to clopidogrel. After three days, the team decided to wait a few more days with close monitoring, but as the same reaccumulation pattern persisted, super-selective contrast injection into the PDA through a Progreat microcatheter was performed, confirming a persistent distal leak. Considering the available options to seal the perforation—covered stent, coils, autologous blood/fat particle embolization, and gel foam embolization—the team opted for gel foam, and embolization was performed in the mid and distal PDA. Following the procedure, no further reaccumulation of pericardial fluid occurred, the pigtail catheter was removed 48 hours later, and the patient was discharged in stable condition. This case underscores the importance of close hemodynamic surveillance after PCI, particularly in complex CTO cases, as delayed small distal coronary perforations may appear benign yet lead to life-threatening tamponade, and it highlights gel foam embolization as a minimally invasive, cost-effective approach that successfully sealed the perforation without the need for surgical intervention or high-cost devices.

Case 3: A 62-year-old male with chronic kidney disease on maintenance haemodialysis and a prior history of coronary artery bypass grafting (CABG) performed two years earlier presented with chest pain for one week suggestive of myocardial ischemia. Transthoracic echocardiography showed mild concentric left ventricular hypertrophy with regional wall motion abnormalities involving the basal and mid anterior wall and anterolateral wall, mildly reduced left ventricular systolic function (ejection fraction 48%), Grade I left ventricular diastolic dysfunction, mild mitral regurgitation, and mild tricuspid regurgitation with a pulmonary artery systolic pressure of 26 mmHg; the aortic valve was sclerotic without stenosis or regurgitation, the pericardium was normal, no intracardiac thrombus or vegetation was identified, and the inferior vena cava measured 12 mm with normal inspiratory collapse. Coronary angiography was performed using a 6F sheath with standard diagnostic catheters (6F JR and 6F JL) and revealed a normal left main coronary artery; the left anterior descending artery (LAD) was a type III vessel with an ostioproximal chronic total occlusion and TIMI II distal flow; the non-dominant left circumflex artery (LCX) had a critical 99% tight ostioproximal lesion with mild diffuse disease in the distal LCX; the obtuse marginal branch showed a long proximal-to-mid segment lesion with maximum severity of approximately 80%; the dominant right coronary artery (RCA) had a proximal chronic total occlusion; and the left internal mammary artery (LIMA) graft was patent. The angiographic impression was triple-vessel coronary artery disease in a post-CABG patient, and given persistent ischemia, percutaneous coronary intervention was planned. During guidewire manipulation, angiography demonstrated a coronary artery perforation localized to a small distal coronary vessel, classified as an Ellis class II perforation characterized by myocardial or pericardial blush without a frank contrast jet or a ≥1 mm exit hole. Immediate transthoracic echocardiography showed no pericardial effusion; however, considering the patient's high-risk profile (chronic kidney disease, post-CABG status, and risk of delayed pericardial effusion and cardiac tamponade), prompt definitive management was undertaken. Therapeutic options including covered stent placement, coil embolization, and autologous clot embolization were considered, and due to the distal location and small vessel caliber, gel foam embolization was selected; gel foam was delivered through a microcatheter, successfully sealing the perforation with complete resolution of contrast extravasation on repeat angiography. The patient remained hemodynamically stable throughout the procedure and post-intervention, serial echocardiography showed no development of pericardial effusion, and after embolization he

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

had no recurrence of chest pain or electrocardiographic changes; serial transthoracic echocardiography performed immediately after the procedure and during subsequent monitoring showed no evidence of pericardial effusion or cardiac tamponade. A repeat coronary angiogram after stabilization confirmed complete sealing of the perforation with no residual contrast leak and preserved distal flow in the treated vessel. Given the favourable angiographic result and clinical stability, further PCI was planned and performed as indicated, the remainder of the hospital course was uneventful, the patient remained stable on optimal medical therapy, and no delayed complications related to the coronary perforation or embolization were observed. This case underscores that although coronary artery perforation is a potentially catastrophic complication, early recognition, immediate imaging, and definitive management using gel foam embolization can result in excellent procedural and clinical outcomes even in complex, high-risk post-CABG patients with chronic kidney disease, and the remainder of the hospital course was uneventful.

Discussion

The incidence of coronary artery perforation, a rare PCI event, ranges between 0.3% to 3%.(7, 8) Chronic complete occlusion, angulated lesions, tiny arteries, aging, calcified vessels, and postcoronary artery bypass grafting (CABG) are some of the risk factors that raise the chance of perforation.(9) GELFOAM Sterile Powder, saturated with sterile sodium chloride solution, is indicated in surgical procedures, including those involving cancellous bone bleeding, as a haemostatic device, when control of capillary, venous, and arteriolar bleeding by pressure, ligature, and other conventional procedures is either ineffective or impractical. Gel foam preparation steps: 1. Open the envelope of GELFOAM Sterile Powder. 2. Pour the contents (1 gram) carefully into a sterile beaker. 3. Add approximately 3-4 mL of sterile saline or thrombin solution to prepare a putty-like paste. 4. If less viscosity is desired, add 7-10 mL of sterile saline or thrombin solution. 5. Compress the mixture with gloved fingers into the bottom of the beaker (to avoid dispersion of the powder). 6. Knead into the desired consistency. 7. Smear or press the doughy paste against the bleeding surface. 8. Remove the excess paste once haemostasis is achieved. The contraindications of GELFOAM include: should not be used in closure of skin incisions because it may interfere with healing of the skin edges. This is due to mechanical interposition of gelatin and is not secondary to intrinsic interference with wound healing. Do not use GELFOAM Sterile Powder in patients with known allergies to porcine collagen

Our case series and the case report by Das et al.(6) demonstrate the critical utility of GELFOAM embolization in managing distal coronary artery perforations, particularly in challenging anatomical and clinical settings. In the present case series, two elderly male patients presented with ischemic heart disease, one with in-stent restenosis and the other with CTO of the RCA, both of whom developed distal coronary perforations—one acutely during PCI and the other in a delayed fashion with hemodynamic compromise due to tamponade. At the same time, Das et al.(6) described a single, younger 43-year-old diabetic patient undergoing primary PCI for anterior wall STEMI with a heavily calcified Type C lesion in the mid-LAD. The perforation in Das et al.(6) occurred in a small distal diagonal branch following balloon dilation and was complicated by persistent bradycardia, possibly triggered by a Bezold–Jarisch reflex due to pericardial irritation. Despite multiple balloon tamponade attempts and heparin reversal, the leak persisted, necessitating precise Gelfoam embolization using a Caravel microcatheter to sacrifice the distal diagonal branch.(10)

Similarly, Ford et al.(11) reported a mean patient age of 69 years, with patients suffering Ellis III/IIIB perforations being older (mean 71 vs. 67 years, P=0.027), while renal dysfunction and

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

diabetes were both predictors of worse outcome in the GNOCCI study. While the COPIT analysis by Mikhail et al.(10) flagged female sex, hypertension, CKD, and prior CABG as statistical predictors of CAP, the present study patients were both males without CKD or prior CABG, but with other known risk factors such as hypertension and complex coronary anatomy (CTO, ISR).

However, Agarwal et al.(7) comprises six cases of coronary artery perforation including multiple Ellis Class III perforations involving major epicardial arteries like the mid-LAD and RCA, predominantly managed with covered stents, balloon tamponade, and surgical drainage where necessary. Comparatively, the COPIT study by Mikhail et al.(10) determined an overall coronary perforation rate of 0.39%, with 21.1% leading to cardiac tamponade and Ellis III perforations accounting for 43%, and in the GNOCCI study by Ford et al.,(11) the overall incidence of coronary perforation was 0.37% across 43,343 coronary procedures over 18 years, with Ellis I/II types accounting for 65% and Ellis III/IIIB types for 35% and identified a rising trend in perforation incidence ($P<0.001$), likely due to an increase in complex PCI techniques, whereas, the current case series involved two patients with distal RCA/PDA perforations, one of which was an Ellis II perforation without tamponade, and the other evolved into delayed tamponade post-PCI. Also, Mikhail et al.(10) identified distal wire exit (37%) and balloon dilatation (28%) as the two most common causes of perforation. This mechanistic trend is reflected in our first case, where guidewire manipulation during RCA CTO PCI likely led to distal vessel injury. The second case might represent a combination of wire trauma and balloon-induced microperforation evolving over time.

In the present series, our first case involved a prophylactic use of Gelfoam soon after detecting an Ellis Class II perforation, without significant hemodynamic instability, and the second case involved a more prolonged clinical course with daily pericardial drainage for several days before definitive Gelfoam embolization was performed. Notably, both of our cases involved the RCA and PDA branches, and careful monitoring, staged decision-making, and avoidance of immediate vessel sacrifice allowed for favourable outcomes without major myocardial infarction. However, Das et al.(6) highlight the controlled and intentional occlusion of a distal vessel to arrest ongoing bleeding, accepting a small infarct as a trade-off. Furthermore, Das et al.(6) method of preparing and delivering Gelfoam—by manually shaving sponge particles, mixing with contrast, and injecting through a three-way cannula—is similar to our approach, reinforcing the practicality of this low-cost technique in resource-limited cath labs. However, covered stents were used in 25%, and 17% required emergency surgery in the Mikhail et al.(10) study, whereas in the GNOCCI study by Ford et al.,(11) covered stents were deployed in 63.2% of Ellis III/IIIB cases, achieving a 75% success rate, however, complications were notable such as device delivery failure, high thrombosis risk (9%), and 47% 24-hour mortality when haemostasis was not achieved in their research and was in contrary to our findings where we managed both cases using gel foam embolization via microcatheter, with successful haemostasis, no mortality, and no reintervention.

The above studies emphasize the importance of recognizing perforation early, understanding the limitations of covered stents or coils in small or distal vessels, and the value of Gelfoam as an effective embolic agent. However, our cases uniquely underscore the importance of post-PCI hemodynamic surveillance, judicious guidewire manipulation in CTO settings, and timing the intervention based on patient stability, whereas Das et al.(6) illustrate an acute procedural complication requiring immediate vessel sacrifice to prevent catastrophic tamponade. Despite different clinical trajectories, above studies underscore a unified message: Gelfoam is a viable, accessible, and effective tool for coronary perforation management, particularly in distal or small vessel perforations where conventional options are limited. Several authors, including

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

Thomas et al.,(12) have reported successful Gelfoam coil embolization of coronary perforations. While our approach did not involve coils, the standalone use of Gelfoam delivered via microcatheter mirrors the mechanism of distal flow occlusion described by Thomas and colleagues.(12) The shared outcome—immediate sealing of perforation with no recurrence and stable post-procedural course—supports Gelfoam as a mechanically effective embolic agent even in the absence of coil reinforcement.

Similarly, Deb et al.(13) highlighted the role of Gelfoam in collateral vessel perforation, which parallels the distal PDA perforation observed in our second case. In both reports, standard conservative approaches were initially used (balloon tamponade, drainage), but persistent bleeding necessitated selective Gelfoam embolization, which proved successful in halting the leak. This affirms the applicability of Gelfoam even in small-caliber collateral branches, where covered stents are technically impossible and surgical options are overly invasive. Hashemi et al.(14) documented coronary perforation closure using Spongostan, an ENT-sourced form of Gelfoam, demonstrating cross-disciplinary adaptation of embolization tools. This ingenuity echoes the resource-conscious approach in our practice, where standard microcatheters and readily available materials were used effectively, without requiring specialty embolic agents. It reinforces the idea that distal perforation management can leverage tools beyond the standard cardiology kit, provided their use is precise and clinically appropriate.

Additionally, Vakili et al.(15) emphasized Gelfoam use after guidewire-related distal perforations, which is strikingly similar to our first case, where Ellis II perforation occurred during manipulation in a chronically occluded RCA. In both cases, early detection and targeted embolization prevented progression to tamponade or hemodynamic instability, underscoring the critical value of timely diagnosis and intervention using Gelfoam. While Gelfoam has gained favour for its temporary occlusive properties and ease of delivery, other embolic materials have also been explored. For instance, Mishra et al.(16) described the use of cyanoacrylate glue to manage an Ellis III perforation in the LAD. While glue offers permanent vessel closure, it carries risks of non-target embolization and requires meticulous preparation and expertise. Our preference for Gelfoam reflects a balance between safety, efficacy, and technical simplicity, especially suited to low-pressure, distal perforations where temporary flow cessation suffices for vessel healing. Likewise, Lim et al.(17) reported the successful closure of a distal first diagonal artery perforation using <0.5 mm Gelfoam, further validating the idea that distal vessel size does not preclude embolization success. Our technique aligns with this principle, as his team opted for precise delivery of Gelfoam through 4F and Progreat microcatheters, which allowed safe embolization of small PDA branches without extending to adjacent myocardial territories.

Dash et al.(18) provided a broader context in CTO PCI, suggesting that Gelfoam, autologous clots, thrombin, and PVA are valid bailout tools when balloon occlusion fails. This sentiment is reflected in our second case, where the patient developed delayed tamponade after complex RCA CTO PCI, and conservative measures failed to prevent reaccumulation. Gelfoam was deployed effectively after a 3-day observational window, demonstrating both clinical patience and procedural confidence—traits emphasized by Dash et al.(18) for managing slow-leaking or delayed perforations in CTO settings.

Conclusion

Coronary perforation remains one of the most feared and potentially life-threatening complications during percutaneous coronary intervention, particularly in complex lesions and

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

chronic total occlusions. These two cases highlight distinct scenarios—one with an acute distal RCA perforation managed promptly in the cath lab, and the other with a delayed, slow-leaking distal PDA perforation resulting in recurrent pericardial effusion and tamponade. In both cases, gel foam embolization emerged as an effective, safe, and low-cost treatment modality. Its accessibility and ease of use make it a valuable option, especially in distal vessel perforations where covered stents or coils may not be feasible. These experiences underscore the critical importance of careful guidewire selection and manipulation, vigilant post-procedural monitoring, and readiness to employ innovative, resource-appropriate solutions during cath lab emergencies. While prevention remains the best strategy, prompt recognition and appropriate management can lead to favourable outcomes even in the face of a cath lab catastrophe.

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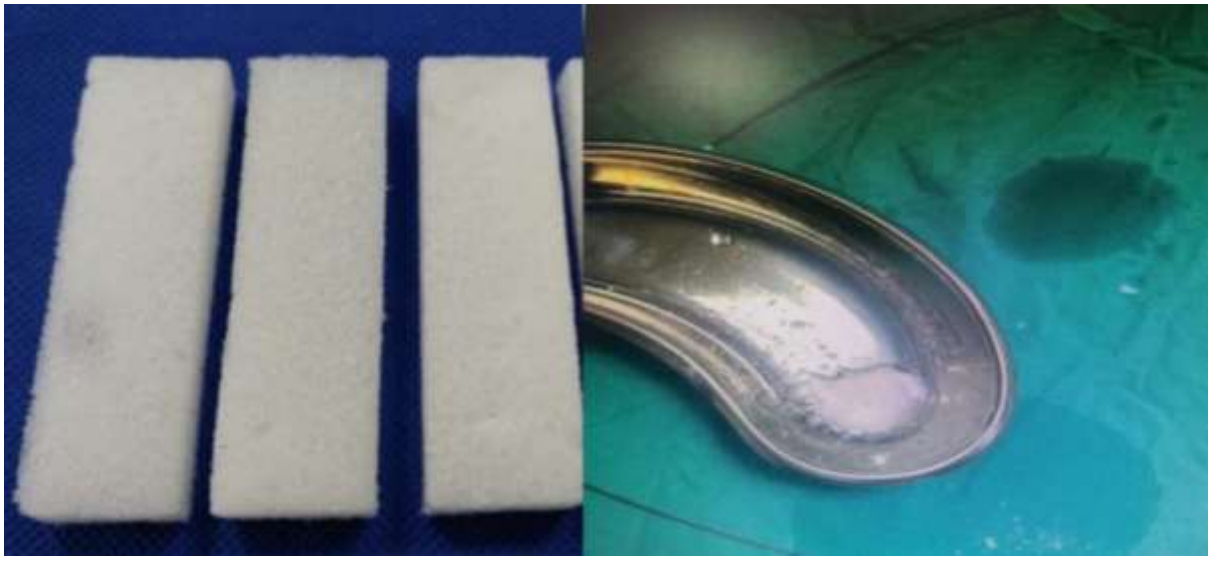

Figure 1: Gelfoam

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

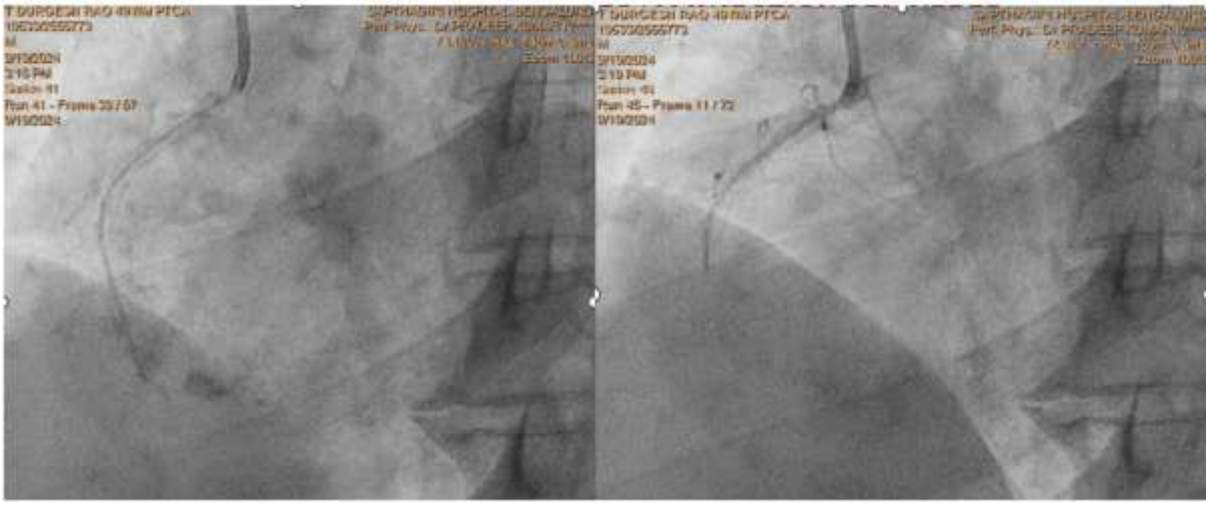

Figure 2: (Case 1) Ellis Class II coronary perforation on angiography before gel foam embolization and after gel foam embolization

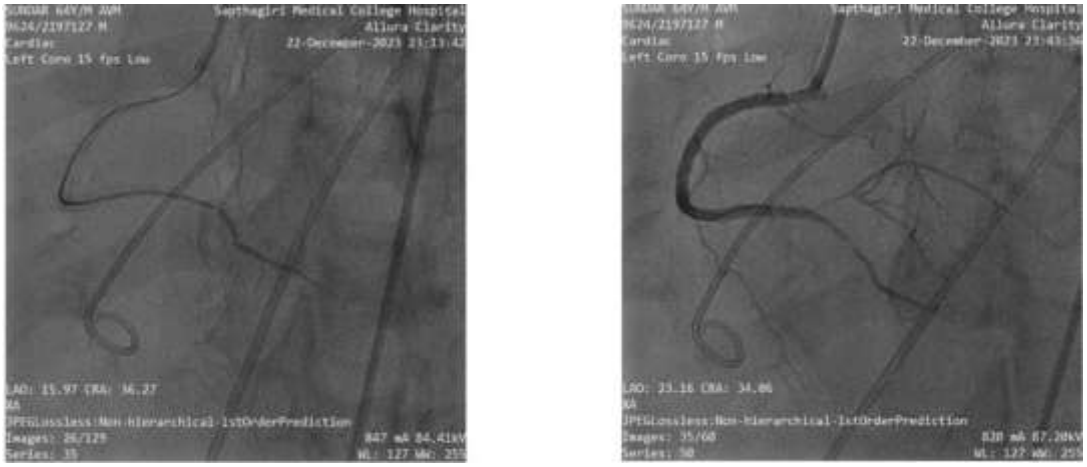

Figure 3: (Case 2) Gel embolization was done at mid and distal PDA

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

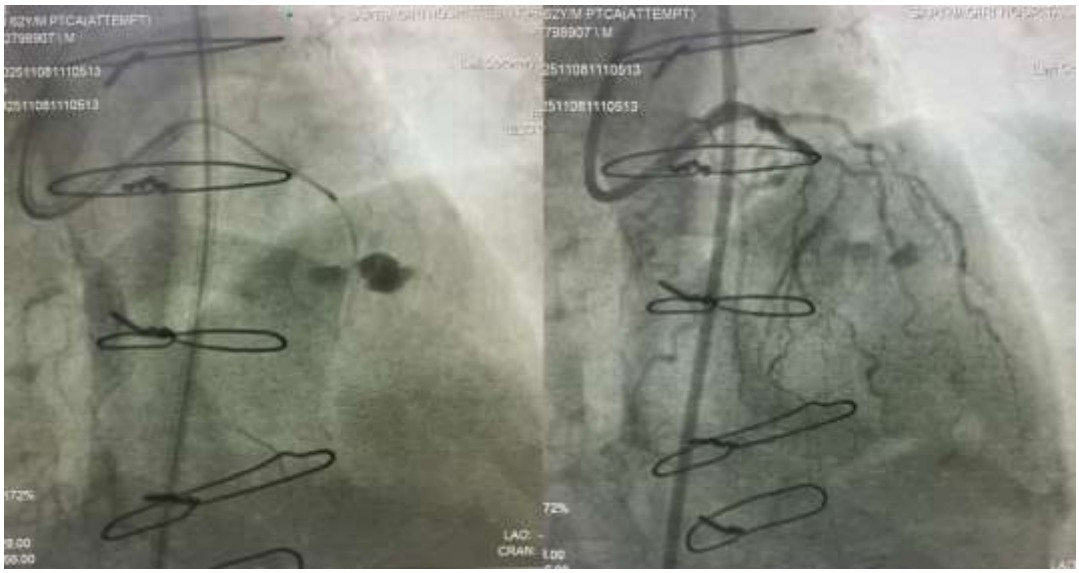

Figure 4: (Case 3) Before and after gel embolization