Textbook of Interventional Cardiology

A Global Perspective

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Dedication

To our families for their selfless support.
To our mentors for invaluable life lessons.
To our institutions for successful partnership.

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Foreword

When I was a fellow learning to do coronary angioplasty from Richard Myler and Simon Stertzer, I had the privilege of meeting Andreas Gruentzig. By 1984, when we got to have a chat at one of his Emory University teaching courses, he was already regarded as the father of interventional cardiology. It took considerable courage in 1977 for him to be the first doctor to thread a balloon into the coronary artery to open the channel and relieve the patient's angina—and it worked. But those were the early days of the field, when all we had were rigid balloons, we had no idea of the right drugs (and doses) to have on board, and there were many patients who suffered significant complications. In spite of that, Gruentzig was an inspirational force with extraordinary charisma and infectious enthusiasm. His persona certainly helped propel the field forward.

Fast forward to the 1990s when I moved from the University of Michigan, running the catheter lab, to Cleveland Clinic, and the opposite happened to me. Over the ensuing years I had the rarified opportunity to work with so many über-talented fellows—and especially closely with the interventional fellows for we were connected by caring for patients together, doing procedures, and engaged in research projects. This was a heady and exciting time when we were testing new drugs like abciximab, bivalirudin, clopidogrel—and new devices including stents and atherectomy. Everything was seemingly new; we had so much to learn. The group of fellows took it all on with unmatched eagerness and work ethic. Each one demonstrated early in their careers that they truly fulfilled the proverbial "triple threat" of excellence in patient care, education (teaching each other and me) and research. Every fellow wrote and published multiple papers, and many of these were in leading journals with significant impact for how interventional cardiology would subsequently be practiced. They are now leaders in the field in many countries all over the world, including Australia, New Zealand, Canada, Israel, Italy, Argentina, Korea, Switzerland, and throughout the United States.

The fellows did more than all these things. They were the pivotal inspirational force to me. Throughout our years and the chance to work together, they provided intellectual stimulation because their curiosity was limitless. And their desire to make an impact in the field that they had chosen to be their life work was unbridled. All of this was admixed with humor and fun, although it seems hard now to remember how we were somehow able to weave that in.

So it was no surprise that many years later this group of former fellows—now leaders—would come to me with the plan to write and edit a new textbook on interventional cardiology. It would be one that would feature a global perspective, since collectively this group surely had that. Once again it surfaced: the interest and willingness to take time from their busy daily professional lives and families to work assiduously on developing this information resource.

I am immensely proud of this "fellow" group, now representing many of the go to gurus in the field, for having put this wonderful textbook together along with so many other experts in the field from all over the world. This is a group of some of the finest individuals, doctors, educators, and researchers I have ever had the privilege of working with in my career. We have lasting friendships and I always relish the chance to get to visit with them, not just to reminisce about a rarified time when there was such unique and accelerated progress in our field, but also to exchange ideas on how it can still advance. When we worked together interventional coronary procedures were quite unpredictable, with not infrequent dissections, thrombosis, and abrupt closures. Hardly were there any noncoronary structural cardiac interventions at the time, but now there are so many diverse ones with left-sided valves and beyond. So interventional cardiology grew up when we had the chance to work together. It is hardly recognizable from what it was in the 1990s.

Predictably, this textbook captures *all* of the ways it works now. And the father of the interventional cardiology—Andreas—would be thrilled to know that four decades later he spawned multiple generations of exemplary physician leaders. They will be relentless in striving to make the field better for patients. Their work to put this remarkable resource together exemplifies the same passion and spirit as when we all worked together—without question, a time I shall never forget.

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Preface

The field of interventional cardiology is growing rapidly across the globe, with advances in devices, techniques, imaging, and collaboration with surgical subspecialties. Technological advances and their adoption is quite variable across different continents. We, all editors of this textbook, although we practice in different continents, bring a unifying perspective from training at the Cleveland Clinic in the United States. We thought that bringing thought leaders from different continents to write a textbook would provide a unique textbook that can be practical and applicable all around the globe.

Interventional cardiology has three major foci for clinical practice and research, including coronary, peripheral, and structural interventions. The book is organized to address important disease states and interventions in each of these fronts. Each chapter is clinically focused and provides important technical pearls where necessary. Devices and their use are discussed including those available in different parts of the world. Despite being clinically oriented, the book is evidence-based, with tables and figures to provide organized presentation of available data. Each chapter also highlights important gaps in our understanding for future investigations. Case studies are provided with chapters to organize thought processes and procedural steps for practical guidance.

Authors who are world-renowned thought leaders in their fields and editors with their diverse but unifying background provide depth and consistency in the book that is unique. The production of the book with tables and illustrations of exquisite quality makes the book easy to read and comprehend. Tremendous effort has gone behind the scenes to make this project a success with outstanding contributions from all authors. Perspective on history and evolution of each field also provides a unique insight from stalwarts in the field who have lived through the advances.

We are hopeful that the book will be a great resource for fellows in training, physicians in practice eager to stay updated, and for researchers interested in understanding clinically important knowledge gaps.

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Video 71.31: Selective right coronary angiogram repeated in different angiographic projections to be certain there is no coronary artery compression. The device was then released.

Videos 71.32A and B: (A) Selective right coronary angiogram after releasing the device demonstrates ideal position of the device in the proximal portion of the fistula without compression of the proximal right coronary artery. There is trivial flow through the device into the distal part of the fistula. (B) Same angiogram in straight lateral projection.

Video 71.33: Selective left coronary angiogram in a patient with recurrent coronary artery fistula after surgical ligation of a distal fistula from the circumflex coronary artery shows a severely dilated proximal left main coronary artery continuing into a long tunnel-like fistula, which opens into the left ventricle underneath the mitral valve annulus. The fistula has proximal and distal stenosis. The left anterior descending coronary artery originates proximal to the fistula.

Video 71.34: Selective left coronary artery angiogram in the same patient as Video 71.33 with temporary occlusion of the fistula with a 12 mm low-pressure balloon inflated across the distal stenotic segment. This angiogram was performed to evaluate for the presence of normal coronary artery branches coming off the fistulous segment prior to closure. Small collateral vessels from the left anterior descending coronary artery supplying the posterolateral segment of the left ventricle were seen not connected to the fistula. Hemodynamics, electrocardiogram and regional wall motion by transesophageal echocardiogram were monitored during balloon occlusion with no ischemic changes documented.

Video 71.35: A 10-mm Amplatzer Septal Occluder (ASO) device was used to occlude the distal portion of this fistula. Selective left coronary artery angiogram during ASO device deployment shows the device in proper position with some residual flow across the device.

Video 71.36: Because of the risk of thrombus propagating into the left main coronary artery, closure at the origin of the fistula was also performed. This selective coronary angiogram was performed during deployment of an Amplatzer Vascular Plug in the proximal fistula after distal closure with the Amplatzer Septal Occluder device.

Videos 71.37A and B: (A) Final selective left coronary artery angiogram after deployment of both devices reveals minimal flow across the proximal segment into the distal portion of the fistula. The left anterior descending coronary artery flow is not compromised. (B) Lateral projection of the same angiogram.

Video 78.1: Right upper pulmonary vein angiogram demonstrating large left-to-right shunting at the atrial level.

Video 78.2: Second defect noted anteriorly while sizing balloon is inflated.

Video 78.3: 8 and 11 French Amplatzer TorqVue delivery systems positioned across the posterior and anterior atrial septal defects, respectively.

Video 78.4: Deployment of first device (18 mm ASO) in the posteriorly located defect.

Video 78.5: Deployment of left atrial disk.

Video 78.6: Deployment of device across septum.

Video 78.7: Deployment of second device (28 mm ASO) in anterior defect.

Video 78.8: Cobra deformity corrected with device manipulation.

Video 78.9: Deployment of left atrial disk.

Video 78.10: Deployment of device across septum.

Video 78.11: Devices released.

Video 78.12: Devices released.

Video 78.13: RA angiogram demonstrating no residual shunting across the atrial septum.

Video 78.14: Moderate-severe paravalvular leak in anterolateral location.

Video 78.15: Defect diameter measuring 3.5 mm (arrow).

Video 78.16: Transapical access using 7 French MUOM sheath. Left ventriculogram showing moderate-severe MR (+3) with left atrial dilatation.

Video 78.17: Paravalvular defect crossed using 0.35" Terumo wire and Glidecath.
Video 78.18: Deployment of 6 mm Amplatzer muscular VSD device across defect.
Video 78.19: Deployment of 6 mm Amplatzer muscular VSD device across defect.
Video 78.20: Left ventriculogram post-intervention with trivial residual leak across mitral prosthesis.
Video 78.21: Trivial residual leak across mitral prosthesis.
Video 78.22: 6 mm Amplatzer muscular VSD device (arrow).
Video 78.23: Two-dimensional video of TTE.
Video 78.24: Color Doppler video of TTE.
Video 78.25: Short axis view of transthoracic echocardiography.
Video 78.26: Modified parasternal view of transthoracic echocardiography.
Video 78.27: Visualization of a 15 × 19 mm ostium secundum ASD with adequate tissue rims around the defect.
Video 78.28: TEE image showing the 18-mm Amplatzer Septal Occluder device successfully implanted in the ostium secundum atrial septal defect.
Video 78.29: TEE images in short axis view, preclosure.
Video 78.30: TEE images in modified parasternal view, preclosure.
Video 78.31: TEE images in short axis view, 3 months postclosure.
Video 78.32: TEE images in modified parasternal view, 3 months postclosure.
Video 78.33: TEE images in four-chamber view, preclosure.
Video 78.34: TEE images in four-chamber view, preclosure.
Video 78.35: TEE images in four-chamber view, 3 months postclosure.
Video 78.36: TEE images in four-chamber view, 3 months postclosure.
Video 78.37: Superior rim (arrow) in TEE.
Video 78.38: Four-chamber view showing transthoracic echocardiography at three months.
Video 78.39: Angiographic confirmation of adequate balloon positioning.
Video 78.40: Injection of diluted angiographic contrast in the target septal branch through the central lumen of the balloon catheter to evaluate its myocardial distribution and confirm seal.
Video 78.41: Injection of diluted echographic contrast in the first septal branch through the central lumen of the balloon catheter with enhancement of the septo-basal area at the point of proximal mitral-septal contact.
Video 78.42: Final angiographic result after injection of 2mL of ethanol in the target vessel: note the occluded first septal perforator artery and the normal anterograde flow in the LAD (final angiography performed after a 10-minute observation period before balloon deflation).
Video 78.43: Left coronary artery in RAO with caudal angulation.
Video 78.44: Left coronary artery in RAO with cranial angulation.
Video 78.45: Coronary angiography: RCA.
Video 78.46: Angiographic confirmation of adequate balloon positioning.
Video 78.47: Diluted radiographic contrast injection in the target septal branch to evaluate its myocardial distribution and adequate balloon seal.
Video 78.48: Contrast enhancement of the basal septum.
Video 78.49: Alcohol septal ablation: alcohol injection. Note the hyperechogenic aspect of ethanol in the basal septum.
Video 78.50: Transesophageal echocardiography.
Video 78.51: 3D echo view of wire traversing the posteriorly located perimitral prosthetic defect.
Video 78.52: Placement of two AVPII plugs.
Video 78.53: Trivial residual paravalvular regurgitation.
Video 78.54: Transthoracic echocardiography.
Video 78.55: Transthoracic echocardiography.
Video 78.56: RAO view of stiff wire crossing the anteriorly located aortic defect below the sinotubular junction.
Video 78.57: 4F Glide catheter following wire across defect.
Video 78.58: 4 mm AVP 4 device was placed, significantly reducing degree of regurgitation.
Video 78.59: Outcome: one 4 mm AVP4 plug was placed.
Video 78.60: Outcome: only trivial residual paravalvular regurgitation was left.

Video 78.61: Transseptal puncture in inferior and posterior septal position. Transseptal access (8 Fr Medtronic system) and visualization of LAA with 4Fr pigtail injecting 9 cc of contrast at 9 cc/second.

Video 78.62: Exchange over Amplatz Extra Stiff wire for Watchman sheath. TEE 23 mm and Fluoro 24 mm means choice of Watchman 27 mm occluder.

Video 78.63: Outcome is adequate compression, no significant leaks. Device was released. Dual antiplatelet therapy, aspirin for 6 months, and clopidogrel for 3 months. TEE follow up at 1, 6, and 12 months. No embolic events at 2 years.

Video 78.64: RAO cranial.

Video 78.65: RAO caudal.

Video 78.66: Choice of 22 mm Amplatzer Cardiac Plug (St. Jude Medical) device based on 18.5 mm measurement of landing zone by TEE (requires a 10 Fr sheath).

Video 78.67: Lobe placement and mild compression without "strawberry" oversizing.

Video 78.68: Final release position.

Video 78.69: TEE at 60 degree view shows good result with no peri-device flow. Device released with no safety issues. Discharged on aspirin for 6 months and clopidogrel for 3 months. TEE follow up at 1, 6 months, and annually. No evidence of TIA/stroke/thromboembolism at 2-year follow-up.

Video 78.70: Echo confirmed severe aortic stenosis.

Video 78.71: Echo confirmed severe aortic stenosis.

Video 78.72: Angiographic check of the femoral access before remove the sheath shows iliac rupture.

Video 78.73: Angiographic check of the femoral access before remove the sheath shows iliac rupture.

Video 78.74: Endovascular repair of the iliac rupture.

Video 78.75: Echo confirmed severe aortic stenosis.

Video 78.76: Echo confirmed severe aortic stenosis.

CHAPTER 88

Clinical Cases

EDITORIAL COMMENT

This chapter provides clinical cases to help illustrate the previous chapters. The cases are contributed by chapter authors and presented in a concise manner with angiograms. The value of these chapters best realized with careful review of video clips of different angiograms. Clinical learning points are clearly outlined for each case. In reality the learning is even more expansive as these are real cases presented by thought leaders who are expert physicians taking care of these patients.

CASE 1: TREATMENT OF LESION IN RCA WITH A DRUG-ELUTING STENT

Georgios Christodoulidis, Usman Baber, Roxana Mehran

History

- A 78-year-old male with known history of coronary artery disease presented to our center with substernal chest pain and associated dyspnea on minimal exertion over the last few days.
- Symptoms were relieved with rest and by the use of sublingual nitroglycerin.
- *Past medical history*: Dyslipidemia, insulin dependent diabetes, chronic kidney disease, known coronary artery disease, and systolic heart failure with an ejection fraction of 30%.
- *Surgical history*: Biventricular Pacing Implantable Defibrillator.
- *Medications*: Insulin, simvastatin, aspirin, enalapril, carvedilol, furosemide.
- *Family history*: No premature coronary disease.
- *Social history*: No smoking, no alcohol use.

Basic Investigations

- Physical examination was unremarkable.
- Admission EKG showed biventricular pacing.
- CBC was remarkable for anemia with hemoglobin of 9.6 g/dL (normal range 11.7–15.0 g/dL).
- Metabolic panel was remarkable for elevated creatinine of 2.8 mg/dL (normal range 0.6–1.4 mg/dL).
- Cardiac enzymes were mildly elevated with a peak troponin of 2.4 ng/mL (normal range 0.0–0.5 ng/mL).
- Patient was diagnosed with non ST segment elevation myocardial infarction and coronary angiography was planned.

Risk Stratification

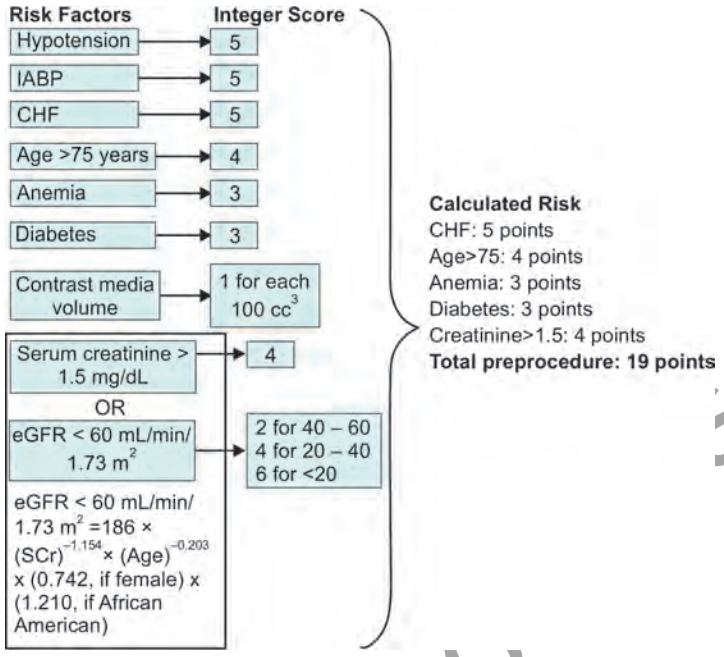

Fig. 88.1: Risk stratification.
Source: Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. J Am Coll Cardiol. 2004;44:1393-9.

Intervention (Management)

- Projected risk for contrast-induced nephropathy (CIN) was very high at 57% and accordingly the risk for dialysis exceeded 12%.
- Considering the high risk-features of the patient as well as his risk for overhydration, the decision was made to use the investigational device RenalGuard.
- A Foley catheter was placed, and 1 hour prior to the procedure, a 250 mL bolus of 0.9% normal saline (NS) was given, and concomitantly, furosemide at an intravenous bolus dose of 0.5 mg/kg was administered.
- Additional doses of furosemide were given whenever urine output fell below 300 mL/h.
- Contrast media given during the procedure was as low as possible (125 cc of iopamidol).
- RenalGuard therapy continued throughout the procedure, and for 4 hours afterward.

Outcomes

- Coronary angiography revealed a 95% lesion in the right coronary artery (RCA) that was treated successfully with a drug-eluting stent.
- Follow-up metabolic profile during his hospitalization did not reveal any creatinine elevation.
- Patient was discharged in a stable state of health 3 days later.
- On discharge, patient's creatinine was at his baseline of 2.6 mg/dL.

Learning Points

- Stratification for CIN risk is of outmost importance in patients undergoing angiography.
- The RenalGuard device is an investigational device which balances fluid administration rate with second-to-second furosemide induced diuresis.
- This device has been shown to decrease the risk for CIN compared to standard hydration protocols in patients at high risk for CIN.
- Additionally, RenalGuard protects patient from overhydration and pulmonary edema.

CASE 2: IN-STENT RESTENOSIS OF LEFT CIRCUMFLEX

Georgios Christodoulidis, Usman Baber, Roxana Mehran

History

- An 82-year-old female with known history of stable coronary artery disease presented to our center with worsening angina symptoms over the last month.
- She denied any symptoms at rest.
- *Past medical history*: Hypertension, dyslipidemia, diabetes, chronic kidney disease, known coronary artery disease, systolic heart failure with an ejection fraction of 38%.
- *Surgical history*: Coronary artery bypass grafting.
- *Medications*: Metformin, furosemide, atorvastatin, aspirin, clopidogrel, enalapril, carvedilol, isosorbide mononitrate, ranolazine.
- *Family history*: No premature coronary disease.
- *Social history*: Former smoker, no alcohol use.

Basic Investigations

- Physical examination was only remarkable for dry mucous membranes.
- Admission EKG showed nonspecific ST and T waves changes.
- CBC was remarkable for a hemoglobin of 16 mg/dL (normal range 11.7–15.0 g/dL), possibly as a result of hemoconcentration.
- Metabolic panel was remarkable for elevated creatinine of 2.4 mg/dL (normal range 0.6–1.4 mg/dL).
- Cardiac enzymes were negative × 3.
- A coronary angiogram was planned for further evaluation.

Risk Stratification

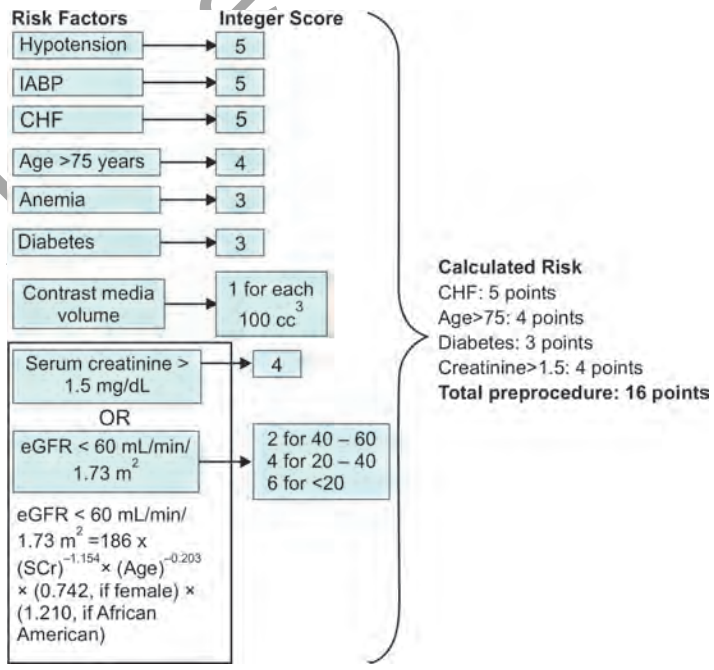

Fig. 88.2: Risk stratification.
Source: Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. J Am Coll Cardiol. 2004;44:1393-9.

Intervention (Management)

- Based on risk stratification, patient was considered as very high risk for CIN (projected risk of 57.3%) with the risk expected to increase even further if contrast media (CM) was given in excess.
- Furosemide was held and patient was given normal saline at 1.5 mL/kg for 12 hours.
- Repeat serum creatinine was 1.4 mg/dL (reflected her baseline from 4 months ago).
- At that time, an angiogram was performed which revealed an in-stent restenosis of left circumflex that was treated with a drug-eluting stent.
- A 90% lesion was also noted in the right coronary artery (RCA), however the lesion was not treated so as to minimize the amount of CM.
- Postprocedure fluids were continued at the same rate for an additional 6 hours.

Outcomes

- Patient was monitored in the hospital for an additional 3 days.
- Her volume status was optimized, and prior to discharge, she was restarted on furosemide albeit at a lower dose.
- During her hospital stay, metabolic panel revealed no creatinine elevation, and on discharge, her creatinine was 1.2 mg/dL.

Learning Points

- Patients should always get risk stratified for CIN prior to angiography.
- If possible, the volume status of the patient should be optimized prior to angiography.
- Hydration with normal saline should be given to all patients before and after the procedure.
- The amount of CM should be minimized, and if further nonurgent intervention is needed, a subsequent staged procedure should be scheduled.

CASE 3: POST-PROCEDURE BLEEDING AND VASCULAR COMPLICATION

Ziad Sergie, Jennifer Yu, Roxana Mehran, Gregg W Stone

History

- A 63-year-old female presented to the hospital with recent onset of chest discomfort with minimal exertion, relieved with rest.
- *Past medical history*: Obesity, hypertension, hypercholesterolemia, diverticulosis (with recent gastrointestinal bleeding), gastroesophageal reflux.
- *Medications*: Amlodipine 5 mg daily, metoprolol tartrate 25 mg twice daily, atorvastatin 10 mg daily, lansoprazole 15 mg daily.
- *Allergies*: None.
- *Family history*: Negative for coronary artery disease.
- Physical examination was unremarkable.

Basic Investigations

- *CBC*: White count 8.6 × 10^3/uL, hemoglobin 12.3 g/dL, hematocrit 39.2%, platelet count 225 × 10^3/uL.
- *Chemistries*: Sodium 138 mEq/L, potassium 4.4 mEq/L, blood urea nitrogen 13 mg/dL, creatinine 0.8 mg/dL.
- *Coagulation parameters*: APTT 28 seconds, INR 0.9.
- *Nuclear stress test*: Modified Bruce protocol to 78% maximal heart rate (5 minutes), terminated because of chest pain (no ECG changes). Myocardial perfusion imaging revealed extensive apicolateral, inferolateral, and posterolateral ischemia.

Cardiac Catheterization

- *Access*: Right femoral artery
- *Anatomy*: Right dominant, two-vessel coronary artery disease (distal RCA subtotal occlusion with a large vessel distally, RPL 60–70%; OM1 subtotal occlusion with a moderate size vessel distally)
- *Ventriculogram*: Ejection fraction 50%, moderate hypokinesis of diaphragmatic and posterobasal segments
- *Pharmacology*: Aspirin 325 mg, clopidogrel 600 mg, bivalirudin IV (0.75 mg/kg × 1, then 1.75 mg/kg/h).
- *Percutaneous coronary intervention (PCI)*: Bare metal stent 3.0 mm diameter, 23 mm long implanted in the distal RCA.
- Sheath was removed 2 hours post-PCI, and access site was managed with manual and mechanical compression.

Outcomes

- The patient was admitted overnight, and was noted to have a large right groin hematoma the following morning.
- Lower extremity ultrasound: right inguinal hematoma 7 × 5 cm, and 3.2 × 2 cm pseudoaneurysm of the distal common femoral artery (at the origin of the superficial femoral artery).
- She received ultrasound guided thrombin injection, with resolution of the pseudoaneurysm.
- Her nadir hemoglobin was 8.4 g/dL (nadir hematocrit 26.6%).
- She received two units of packed red blood cells, and her hemoglobin stabilized at 10.2 g/dL prior to discharge (hematocrit 31.1%).
- There was no acute cardiac event or recurrent bleeding at 1-year follow-up.

Learning Points

- Her bleeding complication can be classified as BARC type 3a (overt bleeding plus a hemoglobin drop 3 to ≤ 5 g/dL).
- Her estimated bleeding risk based on her clinical presentation was in the intermediate range (NCDR score of 55, >2% but ≤6.5% rate of postPCI major bleeding).
- Access site bleeding might have been averted with the use of radial artery access or a vascular closure device.
- A bare metal stent was implanted because of her high risk of recurrent gastrointestinal bleeding (to minimize the duration of dual antiplatelet therapy).
- The access site complication was managed without discontinuation of dual antiplatelet therapy.
- Whether blood transfusion was warranted in this case is uncertain.

CASE 4: BARC TYPE 2 BLEEDING IN A HIGH RISK PATIENT

Ziad Sergie, Jennifer Yu, Roxana Mehran, Gregg W Stone

History

- A 52-year-old female presented with sudden onset chest pressure and dyspnea at rest.
- *Past medical history*: Coronary artery disease (PCI to distal LCx 8 months ago), hypertension, hypercholesterolemia, smoking, end-stage renal disease on hemodialysis for 5 years (via a right AV fistula), medication nonadherence
- *Cardiac medications*: Aspirin 81 mg daily, clopidogrel 75 mg daily, labetalol 400 mg twice daily, clonidine 0.1 mg twice daily, nifedipine ER 90 mg daily, simvastatin 40 mg daily.
- *Allergies*: None.
- *Family history*: Her father had a MI at age 54.
- In the emergency room, her blood pressure was 190/115 mm Hg, heart rate 112 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation 92% on 3L nasal oxygen.
- *Physical examination*: Elevated jugular venous pressure, tachycardia with a S3 gallop, bibasilar rales, lower extremity edema.

Basic Investigations

- *CBC*: White count 9.4 × 10^3/uL, hemoglobin 9.8 g/dL, hematocrit 29.6%, platelet count 197 × 10^3/uL.
- *Chemistries*: Sodium 136 mEq/L, potassium 4.8 mEq/L, blood urea nitrogen 52 mg/dL, creatinine 8.8 mg/dL.
- *Coagulation parameters*: APTT 33 seconds, INR 1.1
- *Troponins*: 0.1 ng/mL (negative × 3).
- *ECG*: Sinus tachycardia, left ventricular hypertrophy, ST depressions in the inferior leads, prolonged QTc (487 ms).
- *Chest X-ray*: Cardiomegaly, small area of atelectasis in the right base, no pleural effusions.
- *Echocardiogram 3 months prior*: LVEF 42% (diffuse), severe LVH, mild aortic stenosis

Cardiac Catheterization

- After treatment of hypertension with IV nitroglycerin infusion, coronary angiography was performed because of persistent symptoms.
- *Access*: Right femoral artery.
- *Anatomy*: Left dominant, two-vessel coronary artery disease; 80–90% in-stent restenosis at former DES site of distal LCx, large vessel distally; 80–90% stenosis in the proximal RCA (small nondominant vessel) not treated.
- *Pharmacology*: Aspirin 325 mg, clopidogrel 300 mg, bivalirudin [0.75 mg/kg × 1, then 0.25 mg/kg/h (adjusted for dialysis)].
- *PCI*: Cutting balloon 3.5 mm diameter, 6 mm long inflated at 12 ATM for 10 seconds at distal LCx ISR with good result.
- *Hemostasis*: Vascular closure device used.

Outcomes

- The following day, she received a hemodialysis session that was terminated early because of hypotension.
- Access site bleeding occurred after hemodialysis requiring mechanical compression.
- The bleeding ultimately responded to the topical application of thrombin powder.
- Following hemostasis, there was no evidence of hematoma or pseudoaneurysm at the access site.
- Her nadir hemoglobin was 8.3 g/dL (nadir hematocrit 24.8%).
- She subsequently tolerated hemodialysis and was discharged home.

Learning Points

- This bleeding event can be classified as BARC type 2 (overt, actionable sign of hemorrhage).
- The estimated bleeding risk based on her clinical presentation was high (NCDR score of 80, or >6.5% rate of post-PCI major bleeding).
- Bleeding avoidance strategies were employed in this case including the appropriate renal dosing of bivalirudin and successful deployment of a vascular closure device.
- Her right radial artery could not be catheterized because of the pre-existing right AV fistula; however left radial access might have been an option.
- Note that blood transfusion was not required.