A case of circumferential multi-vessel coronary intramural hematoma in a post-menopausal woman

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Abstract

Spontaneous coronary dissections are a well described entity which are often difficult to treat. Intramural hematoma is one type of coronary dissection. Previous case reports have described the treatment for angiographically visible dissection. We describe the first report of diffuse intramural hematoma visualized only on intravascular ultrasound with no angiographically obvious evidence of dissection treated with multivessel drug eluting stenting. This case highlights the importance of a high level of suspicion and atypical presentations of coronary dissection, and the use of multimodality imaging with intravascular ultrasound and computerized tomography for both diagnosis and therapy.

Introduction

Spontaneous intramural coronary hematoma is an uncommon cause of myocardial infarction. Causes of intramural hematoma or spontaneous coronary dissection (SCD) include a peripartum state, coronary vasospasm, connective tissue disease, chronic infection, post-percutaneous coronary intervention or stenting, and any condition that compromises the integrity of the arterial wall.[1-6] The optimal treatment approach for intramural hematoma has not been well defined and discussion is limited to case reports.[7-9] Previously described treatments include medical therapy and percutaneous coronary intervention.[3-9]

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Case Report

A 53-year old post-menopausal woman with a past medical history of hypertension and hyperlipidemia came to our attention four months prior to the current presentation with a non-ST segment elevation myocardial infarction. This was thought to be secondary to diffuse moderate to severe luminal stenosis in the second diagonal vessel (Figure 1A and B, Figure 2). This was presumed to be routine branch vessel coronary disease and this was treated medically at that time. Four months later, the patient presented with new onset refractory chest pain, anterior ST depression and congestive heart failure with elevated biomarkers. Angiography revealed diffuse moderate coronary stenosis in the proximal and mid-left anterior descending artery followed by a 99% occlusion in the mid LAD with TIMI 2 flow and severely compromised first diagonal vessel (Figure 1C). Interestingly, the second diagonal ostium which was previously *diseased* was now widely patent with no luminal narrowing; this perhaps indicates a healed intramural hematoma or dissection in this branch vessel from four months before. There was also diffuse moderate to severe luminal narrowing in the distal RCA and its branches (Figure 1D). In comparison to the angiogram performed four months earlier before, there appeared to have been a significant and diffuse progression of coronary luminal narrowing. Initially, a small 2.25 mm balloon was used to dilate the subtotal mid LAD occlusion at 6-8 atmospheres (atm) with complete balloon expansion at a low pressure of 1-2 atm, with no improvement in stenosis and no plaque shift into the side branch. In spite of complete balloon expansion, the stenosis appeared to remain occlusive. Given the dramatic progression in luminal narrowing and atypical response of the stenosis to balloon inflation, routine atherosclerosis was not suspected. There was no angiographic evidence of dissection, i.e. contrast hang-up or luminal linear opacity. Intravascular ultrasound (IVUS) was, therefore, performed to evaluate coronary luminal anatomy. IVUS showed diffuse intramural hematoma with circumferential detachment of the intima from the media along the proximal two-thirds of the LAD right up to the left main bifurcation (Figure 3A). Surgical consultation was requested and an attempt at complex multivessel PCI was recommended which was subsequently implemented. The RCA was treated medically due to diffuse and distal branch vessel involvement precluding percutaneous intervention or bypass grafting. A total of four drug-eluting stents were used to revascularize the LAD and circumflex vessel with a kissing V-stent technique for the LM, LAD and circumflex (Figure 3B), due to milking of the intramural hematoma from the ostial LAD to the ostial circumflex. A cardiac CT which was performed 24 h

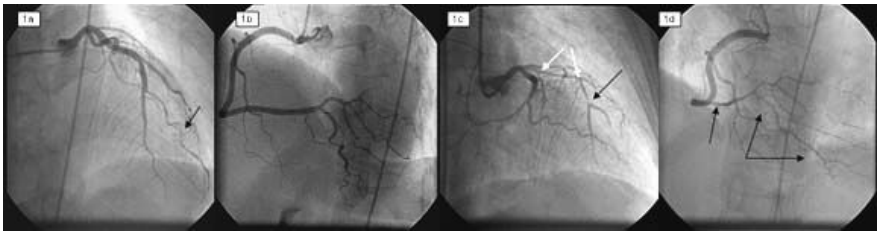

Figure 1. (A) Original picture of LAD, proximal-mid second diagonal demonstrating diffuse luminal narrowing (arrows) with increase in distal caliber. (B) Original picture of RCA (normal). (C) Current picture of LAD, smooth diffusely narrowed proximal-mid segment (white arrows), subtotal occlusion in midportion (TIMI 1-2 flow) (black arrow), no evidence of angiographic dissection. (D) Current picture of RCA, smoothly tapered distal RCA, multiple severe occlusive lesions in distal branches (arrows).

post stenting demonstrated widely patent stents and a reduction in vessel caliber of the distal RCA with abnormal soft tissue adjacent to this segment, consistent with diffuse intramural hematoma with luminal compression (Figure 3C). Although an unenhanced fine cut gated CT is not routinely performed at our institution prior to contrast administration, such a scan may have demonstrated high attenuation along the artery wall in this setting of intramural hematoma.[9] The final angiographic result was excellent in all vessels including the LAD (Figure 3D). The patient was in excellent clinical condition at six months. Laboratory investigations including ANA, lupus inhibitor, rheumatoid factor and complement studies were unremarkable.

Discussion

We present a novel case of multivessel diffuse intramural hematoma treated with selective percutaneous intervention. A high index of suspicion for intra-mural hematoma or dissection is required when atypical presentations of atherosclerosis or infarction are encountered. Intravascular ultrasound and multi-slice CT are complementary and important multimodality imaging techniques when routine coronary angiography may not demonstrate dissection flaps.

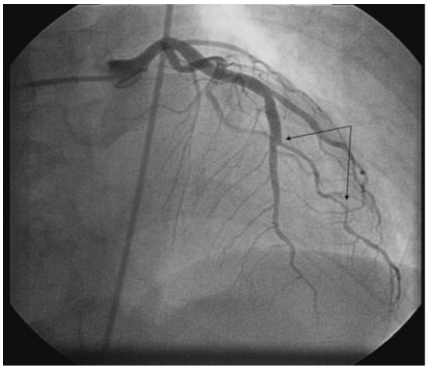

Figure 2. Diseased proximal to mid second diagonal vessel on prior angiogram likely representing diffuse intramural hematoma (mistaken for routine atherosclerosis).

The true incidence of intramural hematoma and SCD is unknown as patients may present with sudden cardiac death.[5] However, post-PCI, intramural hematomas have been diagnosed in up to 6.7% of cases with intravascular ultrasound guidance.[6] Not all risk factors for intramural hematoma or SCD have been identified, but there appear to be four groups of patients who present with intramural hematoma or SCD: women in the peri-partum period, atherosclerosis associated coronary hematoma, idiopathic cases (including patients with connective tissue diseases, vasculitis, lupus erythematosus, extreme exercise, and also drug use), and post percutaneous coronary intervention (PCI).[1,2,6,9] A new stent-edge lesion or *milking* of a *de novo* coronary lesion distally, proximally or into an adjacent branch or vessel has been described previously.[10] Single vessel spontaneous intramural hematomas have been described and previous therapies have included watchful waiting, as the healing process for these lesions may involve simple hemorrhage reabsorption as opposed to routine plaque healing.[7] The typical vessel involved outside the post-PCI setting is often relatively healthy to allow propagation of blood into the media without being impeded by calcific or fibrotic plaque.[6]

The diagnosis of SCD is made through identification of a false coronary lumen on diagnostic imaging. Angiography may fail to reveal a clear dissection flap and may not accurately delineate the extent of the dissecting hematoma. Intramural coronary hematoma may simply appear as diffuse coronary luminal narrowing unless the integrity of the vessel wall can be evaluated directly. The use of IVUS can provide a diagnosis of SCD due to its ability to provide images of the coronary vessel layers.[6] Cardiac computed tomography (CT) has also been reported to be of value in identifying intramural hematoma / SCD and may play a role in monitoring patients after definitive therapy.[4,9] Cardiac CT angiography offers a non-invasive evaluation of luminal patency as well as coronary anatomy, and may also help identify the extent of dissection and progression.

The identification of SCD is important in the setting of myocardial ischemia as classic management may differ from that of thrombotic coronary occlusion.[6,7] Although routine angiography is the main stay of diagnosis of SCD or intramural hematoma, IVUS and cardiac CT angiography are critical multimodality imaging methods which help in the accurate diagnosis and evaluation of the extent of coronary intramural hematoma and other atypical coronary pathologies. Although there is no clear consensus on management of multivessel intramural hematoma or SCD, the key principal of re-establishing coronary flow in the setting of ongoing ischemia holds true as in any acute coronary syndrome.

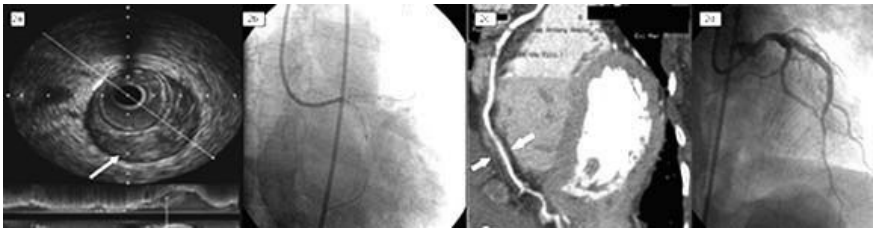

Figure 3. (A) Cross sectional IVUS, intramural hematoma (arrow). (B) Kissing technique for stenting of LAD, circumflex bifurcation. (C) CT angiogram of RCA, demonstrating intramural hematoma surrounding lumen in distal RCA (arrows). (D) Final post stenting angiographic images of LAD, circumflex.

References

1. Maeder M, Ammann P, Angehrn W, et al. Idiopathic spontaneous coronary artery dissection: incidence, diagnosis and treatment. Int J Cardiol 2005;101:363-9.
2. Kamran M, Guptan A, Bogal M. Spontaneous coronary artery dissection: case series and review. J Invasive Cardiol 2008;20:553-9.
3. Kanwar M, Gill N. Spontaneous multivessel coronary artery dissection. J Invasive Cardiol 2010;22:E5-6.
4. Schroder C, Stoler RC, Branning GB, et al. Postpartum multivessel spontaneous coronary artery dissection confirmed by coronary CT angiography. Proc (Bayl Univ Med Cent) 2006;19:338-41.
5. Kamineni R, Sadhu A, Alpert JS. Spontaneous coronary artery dissection: report of two cases and a 50-year review of the literature. Cardiol Rev 2002;10:279-84.
6. Maehara A, Mintz G, Bui AB, et al. Incidence, morphology, angiographic findings, and outcomes of intramural hematomas after percutaneous coronary interventions: An Intravascular ultrasound study. Circulation 2002;105:2037-42.
7. Fujikura H, Hata Y, Morino Y, et al. Images in Cardiovascular Medicine: Acute coronary syndrome due to intramural hematoma. Circulation 2006;114:e644-5.
8. Tremmel JA, Koizumi T, O'Loughlin A, et al. Images in Intervention: Intramural hematoma appearing as a new lesion after coronary stenting. JACC Cardiovasc Interv 2011;4:129-30.
9. Haden G, Polenta S, Jelnin V, et al. Images in cardiology: postpartum intramural hematoma - evaluation by computed tomographic angiography. J Clin Hypertens 2009;11:656.
10. Hirose M, Kobayashi Y, Kreps EM, et al. Luminal narrowing due to intramural hematoma shift from left anterior descending coronary artery to the left circumflex artery. Cath Cardiovasc Interv 2004;62:461-5.