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Research Article

Computational Fluid Dynamics Assessment of Omega Phenomenon; a Systolic Kinking of the Coronary Arteries in a Patient Presenting with Ischemia in the Absence of Coronary Artery Obstruction

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ABSTRACT

Omega phenomenon is an angiographic observation, conceptualized and defined as systolic, exaggerate excursion of epicardial artery segment resulting in aberrant looping/kinking (producing inlet and outlet eccentric angulation) with transient partial luminal obliteration. This entity may be part of the causes of myocardial ischemia in non-obstructive coronary arteries, and it is so called due to its resemblance of the last Greek alphabet.

Introduction

Patients with ischemic heart disease as result of imbalance between myocardial oxygen demand and supply can present with stable angina, acute coronary syndromes, or even myocardial infarction with non-obstructive coronary arteries on angiography or invasive computer tomographic imaging and even without electrocardiographic or elevated biomarker evidence [1-4]. The prevalence shows gender disparity (more in women than men) [5] with, in some cases, a worse prognosis than with obstructive coronary atherosclerotic disease [6]. The most common causes are occult coronary plaques, spontaneous coronary dissection, coronary artery spasm, microvascular dysfunction, Takotsubo cardiomyopathy, myocarditis, and coronary thromboembolism; but some are of uncertain etiology [7]. However, kinetic systolic kinking of the epicardial arteries is not well studied. Here we present Omega Phenomenon, an entity we believe can cause transient ischemic attacks of the myocardium (TIAMYCA) and could partly explain symptoms seen in some patients with Myocardial infarction with non-Obstructive coronary arteries.

Case in Brief

A 61-year-old gentleman presented with chest pain. He was hypertensive, a non-smoker and non-diabetic and had normal left ventricular wall motion, thickness, and chamber sizes. His cardiac biomarkers were not raised. There was only slight ST depression in the anterior-septal leads (V1 and V2). Angiography (Figure 1A) revealed a long diffuse borderline lesion from proximal to middle left anterior descending (LAD) coronary artery that did not interfere with flow (TIMI 3). The FFR, CFR, and IMR of the LAD at maximum hyperaemia before intervention were 0.73, 1.4, and 46, respectively (Figures 2A, 2B, 2C and 2D and Table 1). The LAD

Table 1: Intracoronary physiological studies of the LAD before and after intervention.

Parameters	Before stenting	After stenting
FFR	0.73	0.94
CFR	1.40	2.50
IMR	46.20	45.14

Note: FFR: Fractional Flow Reserve; CFR: Coronary Flow Reserve (CFR); IMR: Index of Microcirculation Resistance

was treated with a 3.0 × 33 mm drug-eluting stent. The FFR, CFR, and IMR after intervention were 0.94, 2.5, and 45, respectively; however, his symptoms persisted. This patient was a referred case from a rural facility, the systolic kinking was noticed before the procedure but was not deemed significant enough to have accounted for his symptoms. But upon further scrutiny and discussion the unusual kinetic motion (looping/kinking) of the LAD, ramus, and OM1 and OM2 epicardial vessels (Figures 1A and 1B) during systole were to be further investigated through simulation studies. Due to its resemblance with the last Greek letter, we called this "Omega phenomenon" and conceptualized it as systolic, exaggerate excursion of epicardial artery segment resulting in aberrant looping/kinking (producing inlet and outlet eccentric angulation) with transient partial luminal obliteration. We hypothesized that this phenomenon could affect coronary blood flow to induce transient myocardial ischemia angina-like chest pain.

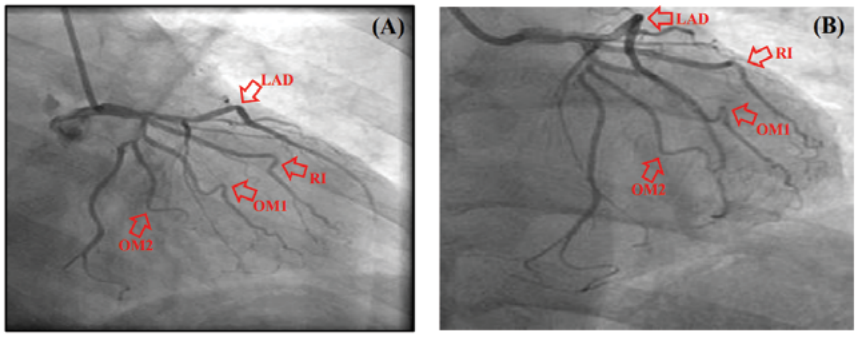

Figure 1: The angiogram of the left coronary artery before (Panel A) and after (Panel B) stent implantation showing systolic kinking of multiple coronary arteries (red arrows).

Computational Fluid Dynamics

Computational fluid dynamics (CFD) and simulation are useful for accurate measurements of changes in flow patterns, velocity, and pressure that occur in blood vessels during contraction and relaxation of the heart [8]. After the intervention, we built idealized 3D geometric models of the coronary vessels (LAD, ramus intermedius, and obtuse marginal) of this omega phenomenon assuming the absence of any artery stenosis. A peak parabolic flow of 0.5 m/s was applied at the inlet. Blood was simulated as a Newtonian fluid with a density of 1060 kg/m^3 and a viscosity of 0.0035 Pa.; and simulation was done during both systole and diastole. Pressure drop (△P), wall shear stress (WSS), and flow velocity were analysed to identify flow disturbance.

The model with a proximal systolic angle of 15° and a distal angle of 165° (Figure 3A) was associated with a dramatic drop in pressure (>90% of total △P) from 1500 Pa to 300 Pa through the proximal angle (15°), compared with an inappreciable pressure gradient at the distal obtuse angle side. Flow velocity in zones associated with drastic pressure fluctuation was increased by 2-fold compared to the relatively insignificant change of velocity (~0.5 m/s) in areas with normal (straight) vessel segments. Flow velocity and pressure fluctuation could only be explained by the sharp and sudden systolic change in the course of the epicardial vessel and eccentric luminal squeeze, leading to much smaller size and irregular vascular morphology. Moreover, we studied the influence of the different angles on WSS, an instigator of endothelial injury that has a strong influence on the initiation and development of vascular disease. In Figure 3B, the WSS (which corresponded to significant pressure drop in Figure 2A) recorded at angle of 15° was much higher. The WSS decreased as the proximal angles changed from acute to obtuse to straight (thus WSS at 90°>165°>180°). Table 2 summarizes the maximum WSS and the averaged WSS with different angles.

Discussion

Omega phenomenon, like other functional disorders of the coronary vasculature, may affect

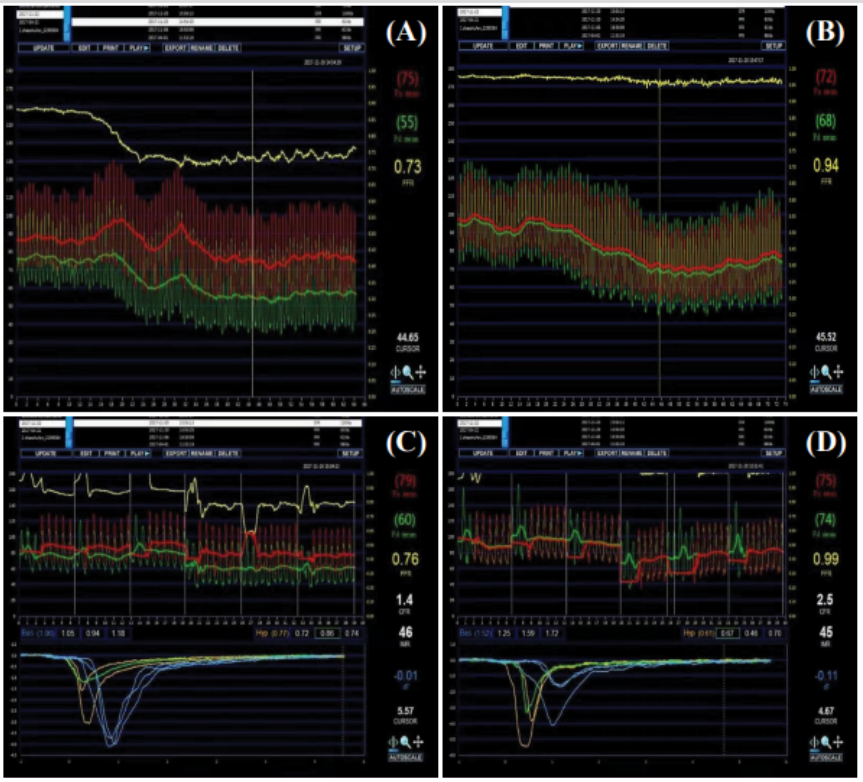

Figure 2: Intracoronary physiological studies of the LAD. A: Fractional flow reserve before intervention was 0.73; B: FFR after intervention was 0.94; C: Coronary flow reserve (CFR) before intervention was 1.4, and index of microcirculation resistance (IMR) was 46; D: After intervention CFR improved to 2.6; however, IMR remained unchanged.

Table 2: Maximum Wall Shear Stress (WSS) and average WSS at various proximal angles, assuming normal distal segments.

Angle (°)	15	45	60	75	90	105	120	135	165
Maximum WSS (Pa)	387	76	65	33	34	36	24	15	27
Average WSS (Pa)	18.7	10.1	10.1	8.6	8.2	7.3	6.6	6.3	6.1
Note: Pa: Pascal unit									

coronary blood flow and perfusion pressure. Contraction and relaxation of the left ventricle induces compression, constriction, dilatation, elongation, traction, and looping/kinking of the epicardial arteries to impact on coronary vasculature and in some cases to interfere with luminal patency and jeopardize coronary blood flow. In the omega phenomenon, it appears that kinetic motion of the coronary arteries and eccentric tandem luminal obliteration or "knotting" induced by the proximal and distal angulations may play roles as resistors with high impedance to blood flow. Based on the CFD results, the persistence of this patient's symptoms could be partly due to 1. ultra-rapid looping/kinking and systolic-diastolic angular variability of the epicardial vessel inducing Dean's vortex flow and whirling associated drop in pressure; 2. progressive loss of elasticity and decreasing compliance in aging atherosclerotic vessels resulting in comparatively longer diastolic straightening time; 3. flow velocity and turbulence, increasing wall shear stress; and 4. redundant volume combined with turbulent flow that may be micro-thrombogenic and a catalyst for impaired microvascular circulation. The omega phenomenon does not fit the conventional definition of coronary tortuosity (fixed and continuous with two or more consecutive 180° turns) [9], because it only produces transient angulation during systole and nearly straightens during diastole. The omega phenomenon and myocardial bridges [10] could co-exist, but they differ because the sudden luminal diameter reduction in the omega phenomenon is a passive auto-obliteration process while coronary artery segment tunnelling/creeping under a strand of heart muscle is actively compressed during contraction of the heart. However, similar to a myocardial bridge, the omega phenomenon does not oppose coronary perfusion occurring during diastole. Omega phenomenon (OP) may offer an alternative explanation for patients with myocardial infarction with non-obstructive coronary arteries (MINOCA). Some of the cluster of entities under

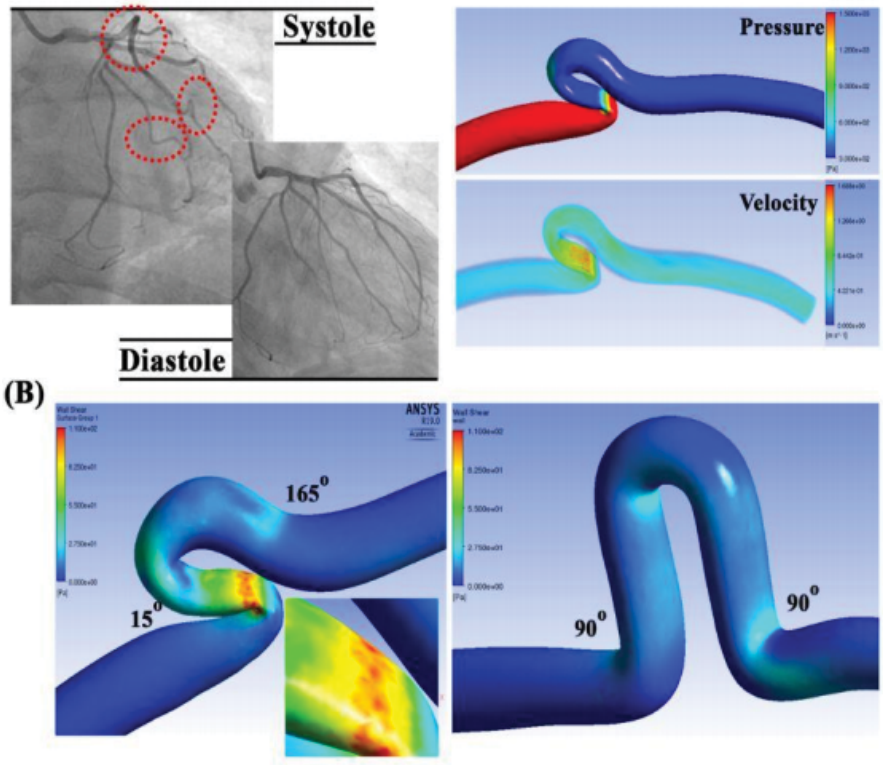

Figure 3: 3D CFD reconstruction showing a sharp pressure drop and increase in velocity at the angle of 15° (proximal) while minor changes at angle of 165° (distal) that was not present when the angles were 90° (proximal) and 90° (distal).

MINOCA include inflammation, erosion or injury of the intima producing coronary vasospasm and conditions that produce dysfunction and abnormal circulation of the microvascular system of coronary arteries. There is no standard treatment or guideline recommendation for patients with MINOCA because it is a new complex clinical entity. The core principles in the management of patient with MINOCA and for that matter this type of case focuses mainly on symptomatic relief, and seek to reduce microthrombus formation and burden of thrombi, restore vascular structure to a state that allows unimpeded coronary blood flow downstream, reduce myocardial oxygen consumption (slowing of heart rate or increasing relaxation (diastolic phase) time especially during period of strenuous activities like exercise, reducing force of cardiac contraction) which could be achieved with the use of patient tailored beta adrenergic blockers. In suitable and selected patients nicorandil or other nitrate analogues may be applied, these help in relaxing coronary vascular smooth muscles, lowers index of coronary microvascular resistance and reduce myogenic tone thereby alleviating angina chest pain. Controlling risk factors associated with adverse outcomes in ischemic coronary diseases are important in the management of patients with OP as well. Invasive treatment like stenting in an attempt to straighten out the kinking segment or coronary artery bypass graft (CABG) may be used. Future work should include functional studies and clinical outcomes to ascertain whether or not stenting and CABG of conductance coronary vessels reduces symptoms.

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