Refractory Vascular Spasm Associated with Coronary Bypass Grafting

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Diffuse refractory vascular spasms associated with coronary bypass artery grafting (CABG) are rare but devastating. A 42-year-old male patient with a past history of stent insertion was referred for the surgical treatment of a recurrent left main coronary artery disease. A hemodynamic derangement developed during graft harvesting, necessitating a hurried initiation of cardiopulmonary bypass (CPB). Although CABG was carried out as planned, the patient could not be weaned from the bypass. An emergency coronary angiography demonstrated a diffuse spasm of both native coronary arteries and grafts. CPB was switched to the femorofemoral extracorporeal membrane oxygenator (ECMO). Although he managed to recover from heart failure, his discharge was delayed due to the ischemic injury of the lower limb secondary to cannulation for ECMO. We reviewed the case and literature, placing emphasis on the predisposing factors and appropriate management.

Key words: 1. Coronary vasospasm  
2. Coronary artery bypass

CASE REPORT

A 42-year-old male patient, who was working for a high school as an administrative staff, was referred for coronary bypass grafting due to recurrent left main coronary disease. Although he was physically active, he had been suffering from anginal pain for several years. Since coronary angiography taken at another center revealed no significant coronary lesion, no specific treatment was given. He also had a history of left carotid stent insertion due to repeated transient ischemic attacks (TIA) three and a half years prior.

A year previous to admission, the patient had suddenly collapsed while playing football. He was resuscitated from cardiac arrest at a nearby community hospital. On echocardiography, ST-T changes were shown on precordial leads, and bedside echocardiography revealed akinesia of the anterolateral wall suggesting the acute coronary syndrome. The patient was transferred to a tertiary medical center for further evaluation and appropriate management. Emergency coronary angiography was performed, and a tight lesion was managed with balloon angioplasty and stent insertion. After a month’s rehabilitation, he was discharged without sequelae.

One year later, a follow-up coronary angiography revealed recurrent stenosis in the distal left main coronary artery that was distal to the previous stenting site, extending to its bifurcation of the left anterior descending artery (LAD) and circumflex artery (Fig. 1). The patient was not obese; in fact, he is rather slim, exhibiting neither hypertension nor diabetes.
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Fig. 1. (A) Coronary angiogram taken before coronary bypass artery grafting (CABG) showing left main stenosis (arrowhead). (B–F) after CABG while cardiopulmonary bypass is still running and (G, H) two months after operation. (B) The guide wire is passed into the left coronary artery, but the whole left coronary system fails to be visualized from the os lesion (arrowhead). (C) Diffuse spasm of the right vertebral and RITA thoracic artery (arrowheads) is well documented with the interruption of the right internal thoracic artery (RITA) flow. (D) The vein graft to the LAD is patent, but a diffuse spasm of the distal left anterior descending artery (LAD) is seen (arrowheads). The retrograde filling of the distal RITA is seen (white arrowheads). (E) The angiogram from left internal thoracic artery (LITA); LITA is patent but the diffuse spasm of circumflex branches can be clearly observed (arrowheads). (F) Right coronary artery is also diffusely spasmodic. (G, H) Relieved spasm of right coronary artery and left anterior descending artery with a widely patent vein graft.
Further, bilateral internal thoracic artery (ITA) grafting was planned considering the patient’s age. While harvesting the right ITA after left ITA harvesting, a hemodynamic derangement developed suddenly. As the patient was unresponsive to a catecholamine infusion, cardiopulmonary bypass (CPB) was instituted hurriedly and a double bypass (left ITA to the left circumflex and right ITA to the left anterior descending) was carried out under standard hypothermic CPB and cold blood cardioplegia. The aortic cross clamp was released, but the heartbeat did not return. Intra-aortic balloon pump (IABP) support was begun, and an additional bypass with a vein graft was made to the distal LAD, but the situation progressively worsened. The patient was rushed to the angiographic suite room, and the angiography revealed a diffuse coronary spasm, which was unresponsive to a nitrate infusion, although the bypass grafts were patent (Fig. 2). The CPB was switched to the femorofemoral venoarterial extracorporeal membrane oxygenator (ECMO). Since the heartbeat was so sluggish and weak, no one expected his recovery and he was put desperately on an immediate transplantation list. Surprisingly, on the next day, signs of recovery were seen and cardiac contractility began to improve. A small perfusion cannula was connected to the arterial line of the circuit and introduced to the distal femoral artery for distal perfusion because of the corresponding ongoing lower leg ischemia. Eventually, the patient could be weaned off ECMO in the afternoon on the second postoperative day. Although the femoral cannula was removed, lower leg ischemia continued to progress and an emergency left femoral Fogarty thromboembolectomy was carried out on the very night of the ECMO weaning, yielding only a scant amount of thrombus. Meanwhile, accidental perforation of the popliteal artery by the thrombectomy catheter necessitated an emergency angiography, which also documented a diffuse spasm of the left lower leg arteries (Fig. 3). His cardiac recovery was rather straight after ECMO weaning; IABP was weaned off the next day and the ventilator on the 7th postoperative day. He was transferred to the general ward on the 9th postoperative day. To our surprise, the condition of his leg, which at first led to the belief that an amputation would be unavoidable, gradually improved. However, his postoperative hospital days were significantly prolonged due to repeated debridement and skin grafting of the leg. On the follow-up coronary angiography taken two months later, the spastic changes were subsided. The vein graft was widely patent, but two ITAs were deemed occluded.

Currently, the patient is at the one-and-a-half-year follow-up point. Although his left foot drop has remained as a sequela, he has returned to work and is in a relatively fair condition. The latest transthoracic echocardiographic result shows an improved left ventricular ejection fraction (49%) and regional wall motion abnormality of the anterior wall and apex.

**DISCUSSION**

Although it occurs infrequently, a vascular spasm in association with CABG can bring about catastrophic results. A refractory vascular spasm has been reported to develop in 0.8% to 1.3% of all CABG procedures [1,2], although transient native coronary or implanted graft spasms have been shown to affect up to 11% of the patients undergoing an operation [3]. Usually, a spasm is confined to a single coronary artery or graft, whereas it may occur in a more diffuse form characterized by a dismal outcome in spite of intensive mana-
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Fig. 3. Carotid angiogram showing the vascular spasm. (A) Before a stent insertion. (B) Spasm (arrowheads) along the distal segment developed shortly after the stent insertion necessitated an additional stent placement.

agement. Recently, Lorusso et al. [4] reported seven vascular spasms out of 5,762 isolated CABG procedures. During the same study period, they reported 18 patients with a perioperative single coronary or a grafted conduit. Out of these seven patients, the first five patients died due to rapidly evolving refractory cardiogenic shock and unresponsive cardiac arrest. The remaining two patients of their series survived after ECMO, emphasizing the importance of the early institution of ECMO for optimal management.

The predisposing factors leading to a vascular spasm include vascular damage, high levels of endogenous or administered vasoconstrictors, and oxidative stress, but no conclusive agreement exists in this respect. Whether the isolated and diffuse patterns of a refractory spasm have different mechanisms in terms of predisposing or causative factors is also poorly understood [3]. Most spasms have been reported to occur within 8 h after the operation in the intensive care unit setting (range, 3 to 8 hours; mean, 5.6 hour) [4]. This case is peculiar in that it occurred during the operation at the time of graft harvesting and before any manipulation of the heart. On tracing the patient’s history, we came to know that there was also a spasmodic event at the time of carotid stenting. Furthermore, we could not exclude the possibility of a vascular spasm in relation to the repeated TIA and the cardiac event leading to stent replacement at the left main branch. A severe spasm of the lower extremity arteries initiated by the femoral ECMO cannula also reflects the patient’s increased vascular tone and spasm-prone characteristics, which could not be defined clearly, although several insights on the mechanism and predisposing factors, including smoking, diabetes, lipid metabolic disorders, and gene expression, have been proposed [5]. There are also reports from off-pump CABG suggesting that the constant administration of vasoressors needed to support low blood pressure can represent an additional vascular stimulus [6,7].

As for the management of the refractory vascular spasm, an intracoronary direct injection of vasodilators has been shown to be effective but may not be sufficient to overcome profound cardiogenic shock or refractory cardiac arrest and to face subsequent significant myocardial stunning. Coronary angiography should be performed at all events to identify and confirm the etiology. The use of IABP, although usually effective in most cases of reduced coronary or CABG conduit flows, is obviously insufficient or useless in this clinical setting, calling for more effective cardiopulmonary support. The efficacy of the prompt institution of ECMO in cases of un-
responsiveness to the above conventional therapies to decompress the suffering and distended heart has been shown, as ECMO provides immediate support to the ischemic myocardium and increased oxygenation, usually impaired by acute lung edema. The catecholamine release may be reduced, myocardial oxygen consumption usually declines rapidly, and the marked ongoing metabolic acidosis may be counteracted, eventually blocking the vicious cycle caused by a refractory myocardial ischemia and contractile depression. ECMO may also play a role in removing cytokines and other inflammatory molecules that are likely implicated in a vasospasm through hemofiltration [4,8].

Hence, we report a case of refractory vascular spasm that occurred during CABG and subsequently managed with ECMO, resulting in a life-salvaging outcome. Further studies are necessary to elucidate the perioperative or patient-specific condition that causes or predisposes the patient to such a catastrophic complication.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.