Multivessel variant angina after a radical nephrectomy operation

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A case of multivessel variant angina after an open radical nephrectomy operation (RNO) is presented. A 52-year-old man was admitted to the coronary care unit with recurrent chest pain and dynamic ST-T wave changes on electrocardiogram early after an RNO. The first diagnosis of the clinical condition was non-ST segment elevation acute coronary syndrome. However, recurrent angina with ST segment elevation occurred after the standard medical therapy, which included beta-blockers. Emergency coronary angiography showed diffuse and multiple narrowing of all the three major coronary arteries during the chest pain, which was relieved by intracoronary nitroglycerine injection. Variant angina was suspected, and beta-blocker therapy was replaced with calcium channel blocker treatment. No angina attacks were observed during the clinical follow-up. Although a direct relationship between the type of surgery and variant angina was not established, coronary vasospasm after an RNO should be kept in mind, especially in the differential diagnosis of a patient with recurrent angina and dynamic ST-T changes on electrocardiogram. Although beta-blocker therapy is a first-line treatment for all acute coronary syndromes, it can be harmful in patients with variant angina and should be stopped immediately after verification of diagnosis.

Key Words: Adrenalectomy; Multivessel variant angina; Nephrectomy; Postoperative; Vasospasm

A first presentation of variant angina pectoris rarely occurs after surgical procedures. It can be a life-threatening disorder, especially in the early postoperative period (1,2). In the present report, a case of multivessel variant angina following a radical nephrectomy operation is presented.

CASE PRESENTATION

A 52-year-old man with no history of coronary artery disease was admitted to the coronary care unit with rest angina that developed postoperatively. His preoperative electrocardiogram (ECG) was normal. He was hospitalized because of recurrent gross hematuria and a hypoechoic mass in the left kidney. Spiral abdominal computed tomography showed a 16 cm × 13 cm × 10 cm tumour in the upper pole of the left kidney in the area of the abdominal aorta and inferior vena cava. Open radical nephrectomy was performed, including removal of the kidney with the ipsilateral adrenal gland and a regional para-aortic lymph node dissection. In the early postoperative period, cardiology consultation was requested because of the patient's recurrent chest pain attacks. There were dynamic ECG changes as biphasic T waves in lead V4, and T wave inversion in leads V5 and V6. His troponin I level was 0.1 µg/L at the time of index presentation and increased to 0.4 µg/L 6 h later. The patient was admitted to the coronary care unit with the diagnosis of non-ST segment elevation acute coronary syndrome. Acetylsalicylic acid, clopidogrel, enoxaparin, beta-blocker therapy and nitrate infusion were started. During the first two days, 12 episodes of angina at rest with 0.5 mm ST segment depression in leads V4 to V6 were observed. Subsequently, one typical attack with ST segment elevation in leads D2 to D3, aVF, and

Un angor pluritronculaire de Printzmetal après une néphrectomie radicale

Un cas d'angor pluritronculaire de Printzmetal après une néphrectomie radicale ouverte (NRO) est présenté. Un homme de 52 ans a été hospitalisé à l'unité de soins coronariens en raison de douleurs thoraciques récurrentes et d'ondes ST-T à l'électrocardiogramme peu après une NRO. Le premier diagnostic clinique en était un de syndrome coronarien aigu avec élévation du segment ST. Cependant, une angine récurrente accompagnée d'une élévation du segment ST s'est produite après la pharmacothérapie standard. Une coronarographie d'urgence a révélé un rétrécissement diffus et multiple des trois principales artères coronaires pendant les douleurs thoraciques, soulagées par une injection intracoronaire de nitroglycérine. Les auteurs ont soupçonné la présence d'un angor de Printzmetal et ont remplacé la bêtabloquants par des inhibiteurs calciques. Ils n'ont observé aucune crise d'angine pendant le suivi. Bien qu'on n'ait pu établir de lien direct entre le type d'opération et l'angor de Printzmetal, il faudrait se rappeler de la possibilité de vasospasme coronaire après une NRO, surtout dans le diagnostic différentiel d'un patient souffrant d'angines récurrentes et de modifications dynamiques des ondes ST-T à l'électrocardiogramme. Un traitement aux bêtabloquants aurait pu être nuisible dans ce cas. C'est le traitement de premier recours de tous les syndromes coronariens aigus.

Figure 1) ST segment elevation in leads D2, D3, aVF with reciprocal ST segment depression in leads D1 and aVL were seen

reciprocal ST segment depression in leads D1 and aVL developed (Figure 1). The patient underwent emergency invasive evaluation for primary coronary angioplasty. A coronary angiogram showed

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Figure 2) Diffuse and multivessel vasospasm in the right coronary (A) and left coronary (B) systems. C and D All stenoses improved with intracoronary nitroglycerine injection

multivessel focal and diffuse coronary stenoses (Figures 2A and 2B). All of these improved with intracoronary nitroglycerine injection (Figures 2C and 2D). Variant angina was suspected, and beta-blocker therapy was replaced with calcium channel blocker and nitrate treatment. No chest pain attack was observed during the clinical follow-up period. The patient was discharged and experienced no further events by the end of the first month of therapy.

DISCUSSION

Although coronary spasm rarely occurs during the postoperative period, sudden death associated with malignant arrhythmias is probable in this situation (1). Multivessel spasm may cause more extensive myocardial ischemia and can be more life-threatening than a one-vessel spasm. There are a limited number of reports of spontaneous multivessel variant angina in the early postoperative period (3). To the best of our knowledge, the present case is the first of a spontaneous

multivessel coronary spasm that developed immediately after a radical nephrectomy operation.

Koshihara and Hoka (2) reviewed 115 case reports of perioperative coronary spasm and suggested that the most probable underlying pathologies of vascular hyper-reactivity include inadequate depth of general anesthesia, use of vasopressors, vagus nerve stimulation, drugs other than vasopressors and epidural block. In the present case, vagus nerve stimulation due to celiac nerve plexus irritation during the operation is the only possibly related factor among these.

The causal roles of the type of operation on sympathovagal imbalance are limited. Only two case reports of neck surgery showed that a sympathovagal imbalance due to carotid sinus stimulation caused severe coronary spasm (4,5). In the present case, both nephrectomy and unilateral adrenalectomy could have triggered autonomic alterations. Although the direct relationship between specific surgery and variant angina was not established in the present unusual case, we hope it will serve to be an example for promoting new investigations in these situations.

Beta-blocker therapy is another triggering factor for coronary spasm in patients with variant angina. This possibility should be kept in mind, especially when evaluating patients with angina in the early postoperative period. In the present case, beta-blocker therapy probably caused prolongation of the variant angina attacks. We did not observe any angina attacks after stopping beta-blocker therapy in our patient.

In our opinion, multivessel variant angina may develop after a radical nephrectomy operation, and a first attack of this kind can mimic an acute coronary syndrome. It should be considered a differential diagnosis, especially in the presence of ECG changes and/or life-threatening arrhythmias following operation. Beta-blocker therapy should be stopped immediately after the verification of diagnosis.

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