Case Report

Coronary artery vasospasm during awake deep brain stimulation surgery

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Although vasospasm usually occurs in the presence of normal coronary arteries, its clinical course is indistinguishable from coronary ischaemia, and actual myocardial damage frequently occurs, as demonstrated by ECG changes and troponin rises seen in such cases. Spasm can be promptly and effectively treated if recognized early, and treatment with nitrate therapy is often sufficient to abolish spasm. As patients are awake during deep brain stimulation (DBS) surgery and may be under considerable distress should vasospasm occur, based on the present case report, it is our opinion that in all cases of DBS surgery, full patient monitoring should be mandatory and an anaesthetist should be present throughout the case. Furthermore, if there is a previous history of cardiac disease or vasospasm, the use of 5-lead ECG monitoring and premedication with beta-blockers and nitrates are indicated.

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Deep brain stimulus (DBS) is increasingly being used for movement disorders, such as Parkinson’s syndrome, dystonias, and essential tremor. The stimulating electrodes are inserted into the deep brain nuclei either under general anaesthesia or more commonly awake, which enables immediate assessment of the clinical effect of stimulation of the nuclei. Usually, the procedure is uneventful, but major complications such as intracerebral haemorrhage may occur. This article describes two patients who demonstrated significant coronary artery vasospasm that occurred during placement of the stimulating electrodes for awake DBS.

Case 1

A 70-yr-old female with Parkinson’s syndrome underwent bilateral pallidal stimulation. She had a past medical history of hypertension and gastro-oesophageal reflux disease. A myoview cardiac scan 1 yr previous to surgery had excluded angina. Medications included clopidogrel (prescribed after an episode of amaurosis fugax and stopped a week before surgery), madopar, bromocriptine, sinemet, esomeprazole, and simvastatin.

A Cosman–Robert–Wells (CRW) stereotactic frame was fitted under local anaesthetic (LA) and sedation (midazolam 1 mg i.v.) with full monitoring without incident. Then, the patient was transferred from the theatres to the X-ray department for a computerized tomography (CT) scan of her head in the frame and subsequently back to the theatre.

The patient tolerated the initial stages of the first (R) electrode placement including scalp incision and burr hole under LA, with normal non-invasive arterial pressure, oxygen saturation, and ECG monitoring. During advancement of the electrode towards the target, the patient suffered crushing central chest pain, became clammy, nauseated, hypertensive to 180/80 mm Hg and tachycardic (95 beats min⁻¹), and desaturated (oxygen saturation 70%), despite O₂ via a facemask. The 3-lead ECG showed ST segment depression of 0.5 mm on lead II and a later 12-lead ECG showed ST segment depression in the lateral leads.

The patient was treated with sublingual glyceryl trinitrate (GTN), oxygen, and diamorphine and the procedure abandoned after securing the electrode. The chest pain lasted for 60 min during which the patient was given three further doses of sublingual GTN. After the operation, she
Coronary vasospasm during deep brain stimulation

was transferred to the coronary care unit where a significant increase in troponin (1.7 ng ml$^{-1}$; values >0.3 ng ml$^{-1}$ indicate myocardial damage) was seen in a blood sample obtained 12 h after the initial chest pain was reported. She made an uneventful recovery and coronary angiography 6 days later showed normal coronary artery vasculature; a diagnosis of coronary artery vasospasm was made by the consultant cardiologist.

Case 2
A 72-yr-old female with essential tremor underwent awake stimulation of the right thalamus. The patient had undergone a successful left thalotomy 2 yr previously. Preoperative history revealed no other significant medical problems, and she had no risk factors for coronary artery disease. At the time of presenting for surgery, the patient was not taking any medications.

Framing with a CRW frame was undertaken using LA and sedation (remifentanil 0.05 μg kg$^{-1}$ h$^{-1}$ and midazolam 1 mg i.v.) under monitoring with non-invasive arterial pressure, oxygen saturation, and ECG. The patient was subsequently transferred to CT scan and back uneventfully. Initial surgery was well tolerated. However, during insertion of the trial electrode towards the thalamus, the patient complained of central chest pain and became clammy. Heart rate and systolic arterial pressure became elevated by 40 beats min$^{-1}$ and 30 mm Hg, respectively. There was ST segment elevation in lead II of the 3-lead ECG, and the patient desaturated (oxygen saturation 72%). She was treated with increased inspired oxygen and GTN. The procedure was abandoned. The chest pain persisted for approximately 45 min.

A subsequent 12-lead ECG showed ischaemic changes in the lateral chest leads, and a serum troponin I sample obtained 12 h after the initial chest pain demonstrated a level of 1.67 ng ml$^{-1}$. She was started on beta-blockers and clopidogrel. An exercise ECG using the modified Bruce protocol did not demonstrate ischaemia. Given the lack of any significant risk factors for coronary artery disease and the negative exercise ECG, the decision was made to not proceed to angiography and a diagnosis of coronary vasospasm was made.

Discussion
Coronary vasospasm has been previously well described in a number of clinical settings, although not during DBS surgery. The precise mechanism is unclear, and it remains a significant clinical and diagnostic problem. The previous work has attempted to elucidate possible aetiologies. Increased parasympathetic tone and vagal activity are frequently cited as causative factors; as spasm can often be reproduced angiographically via direct injection of acetylcholine (ACh) into the coronary artery lumen, the role of local vasoactive mediators has also been studied. It has also been suggested that respiratory alkalosis due to hyperventilation may trigger coronary artery vasospasm. Vasospasm would appear to occur more commonly in post-menopausal females, and sufferers often have risk factors for coronary artery disease, notably hypertension and diabetes.

Coronary vasospasm describes a syndrome of chest pain which is typically cardiac in nature, associated with classical ST changes on the ECG and in some instances elevated cardiac enzymes. The diagnosis is confirmed angiographically by the presence of normal coronary arteries, or presence of stenoses of <20% of the coronary artery lumen (which would be deemed clinically insignificant). Symptoms are thought to arise due to intense smooth muscle contraction within the coronary arterial wall and hence narrowing of the coronary artery lumen.

Several possible theories have been suggested to explain the syndrome of coronary vasospasm. It has been widely suggested that microvascular dysfunction plays a significant role in disease aetiology, and that the patients display either exaggerated responses to endogenous vasoconstrictors or blunted vasodilatory responses within coronary artery smooth muscle.

Abnormal vasoconstrictor responses have been demonstrated experimentally using coronary angiography in patients with known vasospasm. After direct intraluminal injection of ergonovine and, in different studies low-dose ACh, at coronary angiography, reduced coronary blood flow secondary to vasospasm has been demonstrated in this patient group. The effects were not reproduced in patients with significant coronary artery disease, suggesting that an abnormal vasoconstrictor response is present. A similar exaggerated response has been demonstrated in known vasospasm patients with normal coronary arteries after exposure to other stimuli such as oesophageal acid reflux stimulation, hyperventilation, and mental anguish or stress.

It has also been suggested that vasospasm may occur in patients with impaired vasodilatory responses in coronary vasculature to both endothelial-derived and endothelial-independent mechanisms. Substances known to be direct coronary vasodilators such as dipyridamole, adenosine, and papaverine have all been demonstrated to mediate less vasodilatation in subjects with known vasospasm than in other patients. This would suggest that endothelium-independent coronary vasodilatation is impaired in patients with coronary vasospasm.

Endothelium-dependent coronary vasodilatation has also been studied in patients with coronary vasospasm. In normal coronary arteries, acetyl choline has been demonstrated to stimulate release of nitric oxide from endothelial cells, which in turn promotes coronary vasodilatation. However, in patients with coronary vasospasm, this mechanism is impaired and thus reduced vasodilatation is seen. This may be compounded by the exaggerated vasoconstrictive effects that occur in response to low levels of ACh.

Despite having symptoms and signs practically indistinguishable from those generated by coronary artery disease,
acute coronary vasospasm rarely progresses to major cardiac damage, such as myocardial infarction or sudden cardiac death. However, minor cardiac damage, as illustrated by a raise in troponin I levels, and symptoms of anginal chest pain are frequently encountered and management of coronary vasospasm is directed towards preventing symptomatic episodes. Beta-blockers, if tolerated and not contraindicated, are the first-line treatment of choice; where appropriate calcium channel antagonists or long-acting nitrates may be added. In the acute setting, sublingual GTN has been shown to be very effective at relieving coronary vasospasm.

Coronary vasospasm has been reported during a number of surgical procedures, including thoracic spine surgery and during carotid sinus manipulation. It has also been reported in a variety of neurosurgical procedures, notably trigeminal nerve stimulation, and also during anaesthesia for decompressive craniotomy and craniotomy for left cerebellar meningioma. However to our knowledge, coronary vasospasm has not previously been reported during DBS surgery.

References