CASE REPORTS

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Hemodynamic Responses to Electroconvulsive Therapy in a Patient 5 Years after Cardiac Transplantation

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CARDIAC transplantation leads to persistent denervation, with major consequences. Myocardial norepinephrine stores are depleted, neuronal catecholamine uptake is lost, and adaptation to stress and exercise is altered. The heart responds primarily by increasing stroke volume via the Frank-Starling mechanism, whereas changes in heart rate (HR) are minimal. In the normally innervated heart, these responses occur simultaneously. These alterations are of special interest in a cardiac recipient who presents for electroconvulsive therapy (ECT), which is associated with acute hemodynamic perturbations mediated by both the parasympathetic and sympathetic nervous systems. In a series of seven patients, we documented the hemodynamic responses in a cardiac transplant recipient during and after ECT.

Case Report

A 62-yr-old man with a 6-month history of major depression was admitted for ECT. He was being treated with immunosuppressants including azathioprine, cyclosporine, and prednisone for cardiac transplantation 5 yr earlier. Other medical diagnoses included insulin-dependent diabetes mellitus; essential hypertension, which was treated with daily doses of hydralazine, clonidine, and atenolol; Parkinson's disease, treated with levodopa; and a cerebrovascular accident, which had resolved without residual motor disturbances. Four weeks previously, peritoneal dialysis was initiated for chronic renal failure, which was related to bilateral renovascular occlusive disease. However, because of small vessel disease, a revascularization procedure was ruled out. A magnetic resonance image of the brain showed lacunar infarcts and microangiopathy.

Because of severe motor retardation, exercise tolerance could not be assessed, but the patient denied prepericardial discomfort or angina. The electrocardiogram showed sinus tachycardia (124 beats/min) and nonspecific ST-T wave changes in AVI and V2. A transthoracic echocardiogram showed concentric LV hypertrophy but normal LV and RV size and function; ejection fraction was 59%. Right heart catheterization disclosed PA and RV pressures of 58/22 and 58/10 mmHg, respectively, and a cardiac index of 2.4 L·min⁻¹·m⁻².

On each day of ECT, arterial blood pressure (BP) was monitored noninvasively (Dinamap, Critikon, Tampa, FL) every 1 min and HR and leads II and V5 of the electrocardiogram were monitored immediately before and up to 15 min after application of ECT by a monitor (PC Express, Spacelabs, Redmond, WA) equipped with ST segment interpretation software. The electrocardiogram signal was calibrated at 10 mm for each 1 mV, with a frequency response of 0.01-100 Hz. All BP, HR, and ST segment data were collected by a microcomputer, using a fast analog-to-digital converter and customized software. This arrangement permitted simultaneous display (in real time) of all data, both in tabular form and as an XY graph (vs. time); 15 min divisions on the time axis spanned half the computer screen for detailed viewing. Simultaneously, all data were time-stamped and stored with clinical annotations in the order of their occurrence.

After at least one control measurement of BP and several measurements of HR, anesthesia was induced with sodium methohexitol and succinylcholine (both 0.75 mg/kg), and the lungs were ventilated with oxygen via face mask. Taking the absence of a foot sole reflex as evidence of adequate muscular relaxation, a unilateral brief pulse square wave stimulus was delivered by a monitored ECT apparatus. Data collection continued until spontaneous ventilation resumed.

In all treatments, awake systolic and diastolic BP values ranged from 150 to 165 mmHg and from 88 to 92 mmHg, respectively, and HR from 124 to 133 beats/min. To protect against the possibility of supersensitivity of the heart to catecholamines, in the first treatment, we administered 100 mg esmolol immediately before induction of anesthesia. One minute later, the ECT stimulus was applied (fig. 1, top). Given the moderate responses, β-blocker pretreatment was withheld in the subsequent ECT treatments. The hemodynamic responses observed during subsequent ECTs were remarkably uniform (fig. 1, bottom). Relatively small changes in BP and HR were observed. Changes in ST segments were minimal in all treatments.

Discussion

In this patient with essential hypertension, severe vascular occlusive disease in three major vascular beds,
and cardiac denervation, a clinically significant hypertensive response was anticipated after ECT. Short-acting β-adrenergic blocking drugs may be used for attenuation. However, in this long-term cardiac recipient, several considerations relating to the combined effects of β blockers and cardiac denervation were important.
ECT sessions, and changes in BP were less than 50% of predicted by data obtained in patients without cardiac denervation.15 Hence, it is unlikely that cardiac supersensitivity was present. It is recognized that this patient presented with symptoms suggestive of multiorgan consequences of severe cardiovascular disease and that he was treated with antihypertensive drugs that act centrally and peripherally. It is possible, therefore, that responses recorded in this patient might not be representative for all cardiac transplant recipients presenting for general anesthesia during ECT. However, treatment with antihypertensives in cardiac transplant recipients is not the exception, but the rule, as shown by a previous study.6 Of 34 cardiac transplant recipients, 31 were treated with a-adrenergic blocking drugs, calcium channel blocking drugs, converting enzyme inhibiting drugs, or a combination of these.6

The modest BP responses recorded in this patient are in accord with the view that supersensitivity to catecholamines after orthotopic cardiac transplantation is not of practical concern. This view is based on results obtained in the setting of a cardiac catheterization laboratory8,9 and in the setting of clinical anesthesia in large heart transplant centers.11

In this patient, the decrease in BP immediately after ECT was unexpected. One possible explanation is that it occurred secondary to methohexitol, with a delay in appearance of pressor effects, typically seen after ECT. The persistence of derangement nearly 5 yr after cardiac transplant was confirmed by the absence of bradycardia after ECT. This is in agreement with human studies showing that bradycardia did not occur in heart transplant recipients after administration of methoxamine, whereas it could be demonstrated in age-matched control subjects.14 Finally, because accelerated post-transplant coronary atherosclerosis frequently is observed by the third year after transplant,15 cardiac transplant recipients are at increased risk for myocardial ischemic events. However, anginal pain is often absent after cardiac denervation. Therefore, close monitoring of ST segment trends is especially important.

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References