Alterations in Hemodynamic Profile in Orthostatic Hypotension

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Citation

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Abstract

Patients with chronic kidney disease may have associated autonomic dysfunction manifesting as orthostatic hypotension and nocturnal hypertension. The blood pressure regulation may worsen after organ transplantation due to the systemic vasoconstriction induced by the immunosuppression medications.1 Patients with nontransplant related autonomic dysfunction may have a hemodynamic profile different from those related to transplant. We have observed that in contrast to the systemic vasoconstriction found in transplant recipients, a fall in systemic vascular resistance on postural change from supine to upright position, and a lack of compensatory increase in systemic vascular resistance was present during impedance cardiography. These hemodynamic findings and the potential for severe adverse effects of uncontrolled orthostatic hypotension in allograft recipients are discussed.

INTRODUCTION

Orthostatic hypotension represents a heterogeneous group of disease states, and it is usually symptomatic.[1] Characteristic 24-hour ambulatory blood pressure monitor usually reveals a reversal of circadian rhythm, postprandial hypotension and noncompensatory heart rate variability. Nocturnal hypertension is a common, but often ignored accompaniment of orthostatic hypotension. It is frequently present in organ transplant patients and has been attributed to the systemic vasocontrictive effect of immunosuppression medications.[2] While the mechanism of the alteration of the hemodynamic profile in organ transplant patients may be known, our limited clinical experience suggests that it may be different in those with nontransplant related causes. We present a case of hemorrhagic cerebrovascular accident associated with orthostatic hypotension in an allograft transplant recipient and discuss the associated alterations in hemodynamic profile.

CASE REPORT

A 34-year-old male was admitted to the hospital with progressive headache, gait unsteadiness and slurred speech over a 6-hour period. He was the recipient of simultaneous pancreas kidney transplantation six months previously and had an uneventful post-transplant course, except for episodes of symptomatic orthostatic hypotension. The patient was diagnosed with orthostatic hypotension 5-6 years previously and suffered from chronic symptoms of weakness, dizziness, postprandial hypotension, and fluctuating (70-80/40 mmHg to 160-170/90 mmHg) blood pressures. The symptoms had improved since the transplantation, but not resolved. He had been ingesting increased amount of dietary salt and fluids to alleviate his symptoms for two weeks prior to his current admission to the hospital.

Past medical history was remarkable for insulin dependent diabetes mellitus (diagnosed at 13 years of age), proliferative retinopathy with retinal detachment, gastroparesis, peripheral neuropathy, sexual dysfunction and end stage renal disease requiring dialysis treatment for 2 years prior to transplantation. Current medications consisted of tacrolimus, mycophenolate mofetil, prednisone, trimethoprim-sulfamethaxazole, lansaprazole and multivitamins.

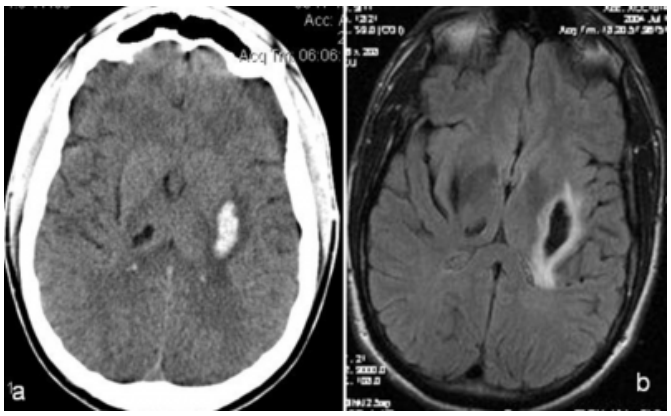
In the emergency department, his supine blood pressure was 160/90, heart rate was 84 beats per minute, and respiratory rate was 16 per minute. Orthostatic blood pressures were not obtained. Significant findings on physical examination were: alert and oriented male with slurred speech, fixed and dilated right pupil (chronic finding, secondary to retinal detachment), round and light-reactive left pupil with intact extraocular movement. The cardiovascular, respiratory, gastrointestinal, genitourinary, musculo-skeletal examination was benign with the exception of the palpation of the transplanted renal allograft in the right lower abdominal

quadrant. Neurological examination was remarkable for decreased lower extremity to touch, pinprick, and vibration in a stocking distribution; mild to moderate motor deficits in the right upper and lower extremity. Laboratory data were: serum sodium 139meq/L, potassium 4meq/L, chloride 99meq/L, total carbon dioxide 25meq/L, blood urea nitrogen 17mg/dL, creatinine 1.2mg/dL; peripheral white blood count $5.5x10^3/mm^3$, hemoglobin 10.1g/dL, hematocrit 30.4% , and platelet 176 $x10^3/mm.^3$

Computer tomography (without contrast) of the head demonstrated a 2.5cm x 1cm hemorrhage with minimal mass effect, located in the left basal ganglia (Figure 1a). Subsequent magnetic resonance imaging showed hemorrhagic infarction in the left putamen and external capsule (Figure 1b). Additionally, evidence for several old hemorrhagic infarctions in the external capsule on the right side and in the globus pallidus region, and small vessel lacunar infarction in the right thalamus were present. The pattern of infarcts was consistent with hypertensive, hemorrhagic infarction.

Figure 1

Figure 1:Noncontrast CT (a) and MRI (b) of the head shows a 2.5cm x 1cm hemorrhage with minimal mass effect, located in the left basal ganglia.



Patient was admitted to the medical intensive care unit and was treated conservatively. His representative blood pressure readings during his hospital course were the following: supine 160/90 mmHg, heart rate 74 beats per minute; sitting 106/66 mmHg, heart rate 84 beats per minute; 70/51 mmHg, heart rate 84 beats per minute. A 24-hour ambulatory blood pressure monitoring was performed (Fig 2), which demonstrated reversal of circadian pattern and nocturnal hypertension (Nighttime mean systolic blood pressure 154.6 + 9.7mmHg with a load of 100%, mean diastolic blood pressure 94.6 + 7.8mmHg with a load of 100%, heart rate 88.6 + 9.3 beats per minute; Daytime mean systolic blood pressure 132.2 + 7.1mmHg with a load of 14.3%, diastolic blood pressure 83.8 + 7.3mmHg with a load of 14.3%, heart rate 96 + 8.6 beats per minute). The hemodynamic profile of the patient, using the BIOZ impedance cardiography device (Cardiodynamics™, San Diego, California, U.S.A.) method, and that of an age-matched healthy male are demonstrated in Figure 3. Systolic and diastolic blood pressures, thoracic fluid content, stroke volume, and systemic vascular resistance index decreased with the change in posture from supine to upright position, but the increases in heart rate and cardiac output were inadequate to prevent the fall in the blood pressures.

Figure 2

Figure 2

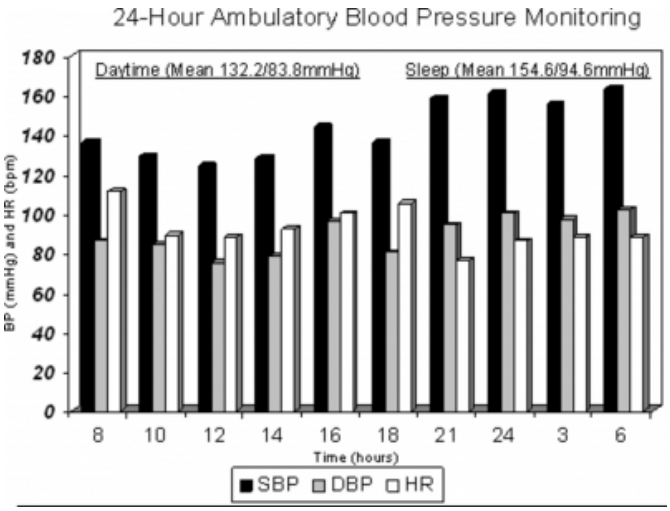
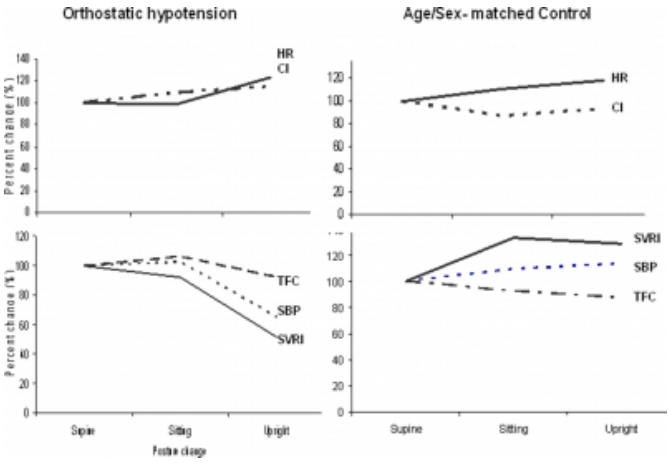


Figure 3

Figure 3: Hemodynamic profile of patient with orthostatic hypotension compared to an age and sex-matched control.



DISCUSSION

Orthostatic hypotension is a difficult clinical problem associated with both neurological and non-neurological

conditions. An increased incidence of autonomic dysfunction has been reported in patients with chronic kidney disease.[3] Autonomic dysfunction is incompletely corrected by allograft transplantation and symptomatic orthostatic hypotension may persist. An important feature of patients with orthostatic hypotension is the concomitant presence of hypertension which may lead to catastrophic end-organ damage,[4,5]as demonstrated in the present case.

Hypertension commonly develops in patients with solid organ transplantation and is characterized by the loss of the normal decrease in nocturnal blood pressure (Fig 2).[2] More than 50% of renal transplant recipients can have nocturnal systolic and diastolic hypertension when assessed by 24-hour ambulatory blood pressure monitor.[6] Hypertension in these conditions is the result of systemic vasoconstriction induced by the immunosuppressive medications. Nocturnal hypertension is also a frequent finding in non-transplant related orthostatic hypotension,[1] however their hemodynamic profile may be different as demonstrated in this report (Fig 3). In contrast to the systemic vasoconstriction found in transplant recipients, a fall in systemic vascular resistance on postural change from supine to upright position was noted. This difference is even more striking when compared with an age and sex-matched healthy individual. It is unlikely that the patient's transplant status or immuno-suppression medications played any major role in his clinical outcome. The lack of compensatory increase in systemic vascular resistance has been frequently observed during impedance cardiography studies on patients with orthostatic hypotension in our limited clinical experience.[1] Despite the notion that orthostatic hypotension represents a common symptom of a heterogenous group of disease states, the mechanism of the loss of the circadian pattern in different disease states may be very different as demonstrated here.

Orthostatic hypotension has been shown to be predictive of ischemic stroke.[7] The significant fluctuations in blood pressure measurements in our patient may have been predictive of the stroke. It has been demonstrated that the greater the change in postural blood pressure, the higher the risk is of advanced silent brain lesions and greater cardiac burden.[8] Intracerebral bleeding associated with hypertension commonly occurs in the tissues of the basal ganglia, pons, cerebellum, and deep white matter of the brain. Evidence for old hemorrhagic strokes was present on CT/MRI imaging of the brain in this patient.

Close observation and management of orthostatic hypotension may influence the long-term survival of patients as a large number of them may have persistent, varying degrees of autonomic dysfunction, even with successful organ transplantation.[9] Although inconclusive, our observations are thought-provoking and warrant further studies to evaluate the mechanisms of a very complex clinical problem.

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