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Non-Cardiac Symptoms of Moderate to Severe Hypokalemia in a Patient with a Syncardia™ Total Artificial Heart

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ABSTRACT

The Syncardia™ total artificial heart (TAH) is an option for patients as a bridge to transplant in those who are not candidates for left ventricular assist devices (LVAD) due to right ventricular failure. Postoperative course is highly dependent on volume status and aggressive diuresis is often necessary. One complication from aggressive diuresis is hypokalemia; however, in these patients we tolerate a lower potassium level because cardiac arrhythmias are not a concern. However, in two separate instances non-cardiac symptoms related to severe hypokalemia occurred. These symptoms included nystagmus in one patient and agitation, tremors, and having an "out-of-body" experience in the other patient. Both these patients had resolution of symptoms with potassium replacement.

CASE REPORT

A forty-four-year-old male with non-obstructive hypertrophic cardiomyopathy underwent placement of a 70 mL Syncardia™ total artificial heart as a bridge to transplant. On echocardiogram, the patient's ejection fraction was less than 10% and he had severe right ventricular dilation and reduced systolic function. The decision was made to implant a total artificial heart into the patient. The postoperative course was stable but the patient needed to be aggressively diuresed with intravenous bumetanide and chlorothiazide due to high fill volumes on both the right and left side of the artificial heart. The patient's potassium level dropped between 2.5-3.3 mmol/L. With these variations in potassium, the patient complained of agitation and tremors of his hands and on one occasion he described having an "out-of-body" experience. These symptoms were resolved with additional doses of potassium chloride. Due to the diuresis, the patient was started on 40 mEq three times a day of potassium replacement.

Another case involved a 47-year-old female with non-ischemic cardiomyopathy with both right and left chronic heart failure who underwent a Syncardia™ total artificial heart placement due to the severely reduced right ventricular systolic function, and the absence of any other severe comorbid disease that would make her ineligible for a cardiac transplant. The postoperative course was also stable and she was extubated the following day. Due to high fill volumes, the patient required diuresis with bumetanide, which caused her potassium to drop from 2.6-3.2 mmol/L for about five days. During this time in severe hypokalemia she developed a nystagmus that was resolved with extra doses of potassium chloride. At this time her potassium level was between 2.6-2.8 mmol/L and these symptoms resolved when the level was brought to above 3 mmol/L.

DISCUSSION

Total physiologic body potassium is about 50 mEq/kg with a 98% intracellular component, of which 75% is concentrated in the muscle. Only 2%, or between 65-70 mEq, is extracellular constituting a serum concentration of 3.5-5.0 mmol/L, with the transcellular gradient being maintained by the Na-K-ATPase pump [Marino 1998; Mount 2004]. Hypokalemia is defined as a serum potassium level less than 3.5 [Rastergar 2001; International Consensus on Science 2000]. Moderate hypokalemia, defined as potassium levels of 2.5-3.0 mmol/L, and severe hypokalemia, defined as a potassium level less than 2.5 mmol/L, have been associated with a number of symptoms, the most severe being cardiac dysrhythmias and heart failure [Smith 2003; Cohen 2001]. Serum potassium levels of greater than 4.5 mmol/L for heart failure and acute myocardial infarction patients have been recommended. A five-fold risk of ventricular fibrillation has been reported in patients with acute MI and potassium levels below 3.9 [Macdonald 2004; Hulting 1981].

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Non-cardiac symptoms of hypokalemia have been reported and affect almost every organ system from weakness and paralysis of skeletal muscles to ileus of the gastrointestinal tract to nephrogenic diabetes insipidus [Lin 2003]. With severe hypokalemia, usually lower limbs are affected to a greater degree than upper limbs, and intact sensory ascending symmetric paralysis has been reported [Riggs 2002; Gennari 1998]. Usually neurologic symptoms are related to acid-base balance and not directly related to potassium levels [Gennari 1998].

Our patients underwent a cardiectomy and Syncardia™ total artificial heart placement, so the most feared complication of hypokalemia, cardiac dysrhythmias, was not an issue with their electrolyte disturbances. With other mechanical circulatory support devices, such as left ventricular assist devices, there is aggressive electrolyte control due to the native right ventricle and the possibility of arrhythmias. Therefore, in those types of patients hypokalemia is treated aggressively and usually severe hypokalemia does not occur. In our total artificial heart patients, one patient did have a correlation to symptoms of shakiness of his upper extremities, agitation, and hallucinations of an "out-of-body" experience with moderate to severe hypokalemia. Our other patient had nystagmus, which is not usually associated with hypokalemia but to acid-base abnormalities. However, because his cardiac function was never affected by the hypokalemia, and since his pH was always normal, acid-base dysfunction could not explain his neurologic findings. Once their potassium was replete to a level above 3.0 mmol/L, their unique symptoms resolved.

REFERENCES

Cohen HW, Shantha M, Alderman MH. 2001. High and low serum potassium associated with cardiovascular events in diuretic-treated patients. J Hypertens 19:1315-23.

Gennari FJ. 1998. Current concepts: hypokalemia. N Engl J Med 339:451-8.

Hulting J. 1981. In hospital ventricular fibrillation and its relation to serum potassium. Acta Med Scand Suppl 647:109-16.

International Consensus on Science. 2000. Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care. Part 8: Advanced Challenges in resuscitation: Life-threatening electrolyte abnormalities. Circulation 102:217-22.

Lin SH, Davids MR, Halperin ML. 2003. Hypokalemia and paralysis. Q J Med 96:161-9.

Macdonald JE, Struthers AD. 2004. What is the optimal serum potassium level in cardiovascular patients? J Am Coll Cardiol 43:155-61.

Marino PL. 1998. Potassium. In: the ICU book. 2nd edition. Baltimore: Williams & Wilkins.

Mount DB, Zandi-Nejad K. 2004. Disorders of potassium balance. In: Brenner BM, ed. Brenner & Rector's the Kidney. 7th edition. Philadelphia: Elsevier, 2004. P.997-1004

Rastergar A, Soleimani M. Hypokalaemia and hyperkalaemia. 2001. Postgrad Med J 77:769-4.

Riggs JE. 2002. Neurologic manifestations of electrolyte disturbances. Neurol Clin 20:227-39.

Smith NL, Lemaitre RN, Heckert SR, et al. 2003. Serum potassium and stroke risk among treated hypertensive adults. Am J Hypertens 16:806-13.