

THE AFIB REPORT

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The Importance of Potassium

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Important: Please read this entire report before acting on its findings

Potassium is fairly abundant in the body with a total content of about 135 grams (3500 mmol). Most, 98% to be exact, is found inside the cells, while the remaining 2% or about 2700 mg is found outside the cells, more specifically in blood serum. Blood serum level is normally maintained between 3.5 and 5.3 mmol/L. Humans evolved on a diet rich in potassium and low in sodium, so the body is designed to retain sodium and excrete potassium. Homeostasis (level between 3.5 - 5.3 mmol/L or 3.5 - 5.3 mEq/L) is maintained by excretion through the kidneys matching oral intake and by shifting potassium between intracellular and extracellular compartments.

Unfortunately, our modern diet tends to produce sodium overload and potassium depletion (hypokalemia). Hypokalemia (potassium level below 3.6 mmol/L[1]) is a serious condition that has been implicated in many aspects of cardiovascular disease including atrial fibrillation, stroke, heart attack, hypertension, and sudden cardiac death (SCD). Hypokalemia is also a strong predictor of early death in heart failure. One study found that as many as 20% of all hospitalized patients have potassium levels below 3.6 mmol/L[1].

Drs. John Macdonald and Allan Struthers of Ninewells Hospital in Dundee, UK have produced an excellent summary of the many consequences of hypokalemia in relation to cardiovascular disease[2]. Among the highlights of their findings:

- Hypokalemia is intimately associated with ventricular ectopy (PVCs) and an increased risk of ventricular fibrillation. Both can be prevented by increasing potassium levels.
- High blood levels of potassium inhibit platelet aggregation and thus help prevent ischemic stroke.
- Adequate potassium levels retard the progression of atherosclerosis.
- Heart attack patients with low serum potassium levels are significantly more likely to go into ventricular fibrillation (often fatal) than are patients with levels between 4.5 and 5.5 mmol/L.

- Potassium supplementation can significantly reduce blood pressure in patients with hypertension.
- A low magnesium level (hypomagnesemia) increases potassium excretion and it is difficult to remedy hypokalemia without first attaining normal magnesium levels. One study found that 42% of people with low magnesium levels also had low potassium levels[1].

The Scottish researchers also outline, in considerable detail, what can be done to remedy hypokalemia. They suggest that supplementation with potassium, on its own, is unlikely to increase levels significantly. The problem is that increased potassium intake activates the renin-angiotensin-aldosterone system (RAAS) which promptly proceeds to generate large amounts of aldosterone which, in turn, causes potassium to be excreted and more sodium to be retained. They estimate that a serum potassium increase of just 0.25 mmol/L results in an aldosterone increase of 50-100%.

Heart failure and heart attack patients should aim for a serum potassium concentration between 4.5 and 5.5 mmol/L. People without cardiovascular disease will probably be OK at levels between 3.5 and 5.0 mmol/L, but there is some evidence that levels of 4.4 mmol/L or higher are required to prevent atrial fibrillation. Dr. Mina Chung of the Cleveland Clinic recommends a minimum level of 4.0 mmol/L for afibbers[3].

It is of interest that Austrian researchers recently discovered that cardiac surgery-induced atrial fibrillation is significantly more common among patients with serum potassium levels below 3.9 mmol/L than it is among patients with levels of 4.4 mmol/L or greater[4].

The National Council on Potassium in Clinical Practice supports the recommendation of a minimum blood serum level of 4.0 mmol/L (4.0 mEq/L), but further suggests that an optimal level for patients without renal dysfunction would be 4.5 - 5.0 mmol/L[1].

For those with low potassium levels the Scottish researchers recommend supplementation with potassium and magnesium combined with an aldosterone blockade to prevent increased potassium excretion. There are four main approaches to blocking aldosterone production or counteracting the effects of an excessive production.

ACE Inhibitors

Angiotensin-converting-enzyme inhibitors prevent the conversion of angiotensin I to angiotensin II and thereby interrupt the RAAS system's efforts to produce aldosterone. Some of the more common ACE inhibitors are enalapril (Vasotec), lisinopril (Zestril), ramipril (Altace), and captopril (Capoten). Unfortunately, there is evidence that the aldosterone-blocking effects of ACE inhibitors may only be transient and that the body eventually finds a way around the ACE inhibition and produces aldosterone just the same[2].

Angiotensin II Type 1 Receptor Blockers

These medications act by preventing angiotensin II from docking at its receptors and thereby inhibit the formation of aldosterone. There is evidence that lone afibbers have more angiotensin II receptors in the left atrium than do non-afibbers, so blocking these receptors could be important for reasons other than the prevention of excessive potassium excretion[5].

There is actually evidence that valsartan (Diovan) reduces aldosterone levels and also some limited evidence that candesartan (Atacand) and irbesartan (Avapro) can help prevent or shorten afib episodes[6,7,8]. Thus, all in all, angiotensin II receptor blockers combined with potassium and magnesium supplementation may be worth evaluating for afibbers with low potassium levels and no indication of hyperaldosteronism.

Potassium-Sparing Diuretics

These help prevent the excretion of potassium through their direct action on the kidneys. Combining them with potassium and magnesium supplements should result in increased potassium levels. The two main medications in this field are triamterene (Dyrenium) and amiloride (Midamor).

Aldosterone Receptor Blockers

These work by blocking aldosterone (mineralocorticoid) receptors and thereby prevent aldosterone from doing its dirty work of excreting potassium at an excessive rate. The main pharmaceutical aldosterone receptor blockers are spironolactone (Aldactone) and eplerenone (Inspra). There is evidence that potassium and magnesium supplementation combined with these blockers increases potassium levels and effectively replenishes tissue levels of both potassium and magnesium[2]. Spironolactone and eplerenone work equally well, but spironolactone is far less expensive. Eplerenone can be used if side effects (gynecomastia, impotence, irregular menses or bleeding, and gastrointestinal irritation) are experienced from using spironolactone.

The findings of the Scottish researchers, to a large extent, fly against conventional medical wisdom. The medical profession has always considered potassium levels above 5.1 mmol/L a far greater risk of ventricular arrhythmia than levels below 3.5 mmol/L. Yet, the Scottish report points out that the risk of ventricular fibrillation is 8 times higher in heart attack patients with potassium levels below 3.5 mmol/L than it is in patients with levels above 4.3 mmol/L. It is also generally considered a definite “no-no” to combine aldosterone receptor blockers and potassium-sparing diuretics with potassium supplementation, yet the report emphasizes that potassium and magnesium supplementation without concomitant aldosterone blockage is ineffective.

The suggestion that both approaches may be needed was also pointed out in a recent paper which concluded that a daily potassium intake (via supplements) of 9 grams combined with a spironolactone intake of 250 mg/day was required to raise serum potassium level from about 4.0 to 5.2 mmol/L in a group of patients with inherited long QT syndrome type 2. The authors of this report concluded that a sustainable, mild increase in serum potassium can be safely maintained by oral potassium supplementation and spironolactone[9].

Other researchers have, however, found that potassium supplementation, on its own, can indeed be effective in reducing blood pressure and the risk of stroke. Daily supplementation with 60 mmol (2.5 grams) of elemental potassium has been found to decrease blood pressure significantly over a 12-week period[10,11]. So if potassium supplementation decreases blood pressure, then it is obviously getting into the system. It should be pointed out though that about a third of the supplemented potassium was excreted in the urine indicating that the RAAS was activated by the supplementation[11]. Sixty mmol of elemental potassium is equivalent to 4.5 grams of potassium chloride, 6 grams of potassium bicarbonate, 7.5 grams of potassium gluconate or 20 grams of potassium citrate.

The National Council on Potassium in Clinical Practice recommends the use of oral supplementation with potassium chloride (800-2300 mg/day of elemental potassium) to replenish potassium, but point out that potassium bicarbonate may be more appropriate if metabolic acidosis is present[1]. If increasing dietary intake or supplementation does not bring potassium to the desired level, then potassium-sparing therapy with ACE inhibitors, angiotensin II receptor blockers, potassium-sparing diuretics or aldosterone receptor blockers may be added to the potassium supplementation regimen[1].

I am currently experimenting with spironolactone and potassium supplementation and have found that a potassium-rich diet (lots of fruits and vegetables) combined with 500 mg/day of elemental potassium (from potassium gluconate), 375 mg/day of elemental magnesium (from

magnesium taurate), and 75 mg/day of spironolactone raised my potassium level from its usual 3.5-3.7 mmol/L to 4.5 mmol/L over a 1-month period. I should mention that I have been diagnosed with hyperaldosteronism, so plain ACE inhibitors or angiotensin II receptor blockers would probably not work for me. I am now attempting, with the cooperation of my physician, to optimize my intake of potassium and spironolactone so as to take the minimum amount of the drug.

I have also observed that I can quickly and completely eliminate ectopic beats and other uneasy feelings in the heart by drinking a special potassium drink. The drink consists of an 8 oz glass of warm water into which I dissolve a pouch of Emergen-C (containing 1000 mg of ascorbic acid + 200 mg of elemental potassium + 60 mg of elemental magnesium) as well as 1/4 teaspoon of potassium chloride providing about 1000 mg of elemental potassium. I drink this concoction over a 10-minute period and also use it to help swallow a 500 mg magnesium taurate capsule providing 125 mg of elemental magnesium.

Other afibbers have observed similar benefits by drinking low-sodium V8 juice. It would seem that the ingestion of a high potassium drink when increased ectopy is felt could help to avert a full-blown episode. The potassium drink could be particularly beneficial for afibbers whose episodes occur after a meal or when lying down to sleep. Research has shown that blood levels of potassium vary significantly during the day. It is as much as 0.6 mmol/L lower during the night than during daytime and also decreases substantially after ingesting a meal containing carbohydrates[1]. So having the potassium drink just before dinner or bedtime may be worth a try for these afibbers.

The high potassium drink may also be useful if consumed throughout the day for afibbers with the diarrhea type of irritable bowel syndrome. Diarrhea can lead to major losses of both potassium and magnesium as stool content of potassium can reach 90 mmol/L. Of course, consuming a potassium-rich drink throughout the day is likely to benefit all afibbers with serum potassium levels below the optimal range of 4.5 to 5.0 mmol/L.

Conclusion

Low serum levels of potassium (most likely accompanied by low intracellular levels) could be an important cause of afib. Potassium supplementation may be required by some afibbers to bring their blood serum level up to the recommended range. It is possible that just supplementing with potassium and magnesium is enough to do the trick; however, if it is not, or if hyperaldosteronism has been diagnosed, then a combination of aldosterone inhibition and oral supplementation with potassium and magnesium would appear to be highly effective.

Moderate supplementation and increased dietary intake of potassium is likely to be safe for most people. HOWEVER, and this cannot be emphasized enough, aggressive supplementation and supplementation combined with aldosterone blockage SHOULD NOT BE UNDERTAKEN without the cooperation of a physician. Potassium levels need to be monitored regularly since potassium supplementation, if kidney dysfunction is present, can be fatal. Please take this as a SERIOUS WARNING!

References

1. Sica, DA, et al. *Importance of potassium in cardiovascular disease. Journal of Clinical Hypertension (Greenwich), Vol. 4, May/June 2002, pp. 198-206*
2. Macdonald, JE and Struthers, AD. *What is the optimal serum potassium level in cardiovascular patients? Journal of the American College of Cardiology, Vol. 43, January 21, 2004, pp. 155-61*
3. Chung, MK. *Vitamins, supplements, herbal medicines, and arrhythmias. Cardiology in Review, Vol. 12, March/April 2004, pp. 73-84*

4. Auer, J, et al. Serum potassium level and risk of postoperative atrial fibrillation in patients undergoing cardiac surgery. *Journal of the American College of Cardiology*, Vol. 44, August 18, 2004, pp. 938-39
5. Boldt, A, et al. Expression of angiotensin II receptors in human left and right atrial tissue in atrial fibrillation with and without underlying mitral valve disease. *Journal of the American College of Cardiology*, Vol. 42, November 19, 2003, pp. 1785-92
6. Cohn, JN, et al. Sustained reduction of aldosterone in response to the angiotensin receptor blocker valsartan in patients with chronic heart failure. *Circulation*, Vol. 108, September 16, 2003, pp. 1306-09
7. Kumagai, K, et al. Effects of angiotensin II type 1 receptor antagonist on electrical and structural remodelling in atrial fibrillation. *Journal of the American College of Cardiology*, Vol. 41, June 18, 2003, pp. 2197-2204
8. Madrid, AH, et al. Use of irbesartan to maintain sinus rhythm in patients with long-lasting persistent atrial fibrillation. *Circulation*, Vol. 106, July 16, 2002, pp. 331-36
9. Etheridge, SP, et al. A new oral therapy for long QT syndrome. *Journal of the American College of Cardiology*, Vol. 42, November 19, 2003, pp. 1777-82
10. Whelton, PK, et al. Effects of oral potassium on blood pressure. *JAMA*, Vol. 277, May 28, 1997, pp. 1624-32
11. Gu, D, et al. Effect of potassium supplementation on blood pressure in Chinese: a randomized, placebo-controlled trial. *Journal of Hypertension*, Vol. 19, July 2001, pp. 1325-31

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