

Potassium: A New Treatment for Premenstrual Syndrome

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Abstract

A pilot study was conducted using potassium to successfully treat premenstrual syndrome in seven women with severe symptoms. After following a specific protocol all subjects found their symptoms gradually decreasing in intensity and duration until all were free of PMS. In addition, most found their overall health and energy levels improve. PMS frequently occurs or worsens after situations which are known to deplete potassium. Many PMS symptoms, aside from their unique timing, are the same as many known potassium deficiency symptoms. Serum potassium levels have been proven not to be an accurate indicator of total body content, allowing a mild to moderate deficiency to go undetected.

Introduction

Premenstrual syndrome is a biochemical disorder that afflicts a large segment of the female population. As many as 80 percent of all women who are of childbearing age have noticed at least some degree of premenstrual change in health at one time or another. Approximately 30 to 40 percent of these women regularly experience mild to moderate changes in their physical and emotional health each month prior to their period. In addition, there is an estimated 10 percent who suffer symptoms so severe during this time that it drastically interferes with their lives. For these women, debilitating exhaustion, severe depression, uncontrollable anger and the inability to cope with even the most minor problems are just some of the symptoms that may strike with clock-like regularity each month. Absenteeism from work, admissions to psychiatric facilities, suicide attempts and alcohol

abuse are more common during the premenstrual phase than at any other time of the menstrual cycle. Some common symptoms of PMS include abdominal bloating, water retention, acne, sensitivity to light and noise, headache, fatigue, cravings for sweet and salty foods, depression, hostility, irritability, inability to control anger and suicidal thoughts. An estimated 150 different symptoms have been recorded. Although no one woman will have every symptom, she will usually notice the same set of symptoms each month.

A severe sufferer typically experiences between seven and 12 each of both physical and emotional symptoms every cycle. What characterizes PMS is not so much the symptoms themselves, but the timing of the symptoms. PMS is a unique disorder in that the symptoms, many of which are present in other illnesses, occur only in a cyclic nature in conjunction with the menstrual cycle and are not present all of the time. Having periods is not a prerequisite for having PMS. Women of reproductive age who have had a hysterectomy with their ovaries left intact may still have PMS. For a diagnosis of PMS to be made, a woman must have at least seven consecutive symptom-free days in each cycle that begin by the time her period ends.

The cause of PMS has not yet been established. The consensus of the medical authorities is that PMS involves the sex hormones and brain neurotransmitters, such as serotonin and GABA. Drs. Rubinow and Schmidt from the NIMH have demonstrated that estrogen and progesterone are definitely involved even though these two hormones are present in the same concentrations as women with no PMS.¹ One theory involves endogenous opiate peptides. These protein fragments have the

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same characteristics of opiates and are found within the brain, spinal cord, adrenals, pancreas and the gut. Evidence suggests that EOP's act as neurotransmitters. They have been shown to regulate hormone release from the pituitary, inhibit prostaglandins, influence thirst and appetite, alter glucose metabolism and adversely affect mood and behavior. Measurements in the female rhesus monkey show that the activity of the EOP beta-endorphin is cyclic within the brain. It has been postulated that PMS is the result of either an oversensitivity to, or an abnormal release of, an EOP such as beta-endorphin within the hypothalamic-pituitary axis. Another theory suggests that PMS is a result of progesterone withdrawal in the brain. Progesterone is metabolized to a compound that enhances the functioning of the neurotransmitter GABA. GABA is the brain's major inhibitory neurotransmitter, which has a sedative and anti-anxiety effect. The theory is that PMS may be due to alterations in GABA's pharmacology as a result of progesterone withdrawal.²

The onset of PMS seems to occur or worsen after specific events. The menarche, after stopping birth control pills and after an episode of amenorrhea are frequently associated with the beginning of PMS. Other events linked to the onset or worsening of PMS include physical and emotional trauma or stress, surgeries involving the reproductive organs, illnesses and certain drugs, such as caffeine. Pregnancy is undoubtedly the most common event that triggers or worsens PMS. Some women who develop PMS afterwards report a severe and lengthy post-partum depression that gradually becomes the cyclic pattern of PMS once their periods resume.

Treatments for PMS

Treatment for PMS can include diet, stress reduction, counseling, exercise, specific vitamins and minerals and drugs such as diuretics and tranquilizers. Severe PMS

is often treated with high doses of natural progesterone and this helps an estimated 70% of women providing they strictly observe their diet and monitor their stress levels. Antidepressants such as Prozac®, which increase brain serotonin levels, have been found helpful for about 60% of women, particularly with their emotional symptoms. (Of course, many of the original participants in this study had to drop out due to the drug's side-effects).³ Suppression of estrogen and progesterone using the gonadotropin-releasing hormone agonist leuprolide acetate has been found in clinical trials to help some women with PMS, however women who experience severe depression during their PMS time find it ineffective.⁴ Without some sort of treatment, PMS tends to get worse over time. Fortunately, PMS ends after menopause.

When I was 23 I developed a severe case of PMS immediately after taking a course of steroids. After four years of debilitating symptoms and seeing numerous doctors I decided to research the drug that started my problems. I read that, among other things, steroids deplete potassium. Having just seen a doctor and knowing I had no medical condition that should prevent me from taking potassium, I began taking 600-900mg K/day, everyday. Over the next three months my symptoms gradually decreased in intensity and duration until I was completely free of PMS. In addition, my overall health and energy level significantly improved. Shortly thereafter I developed a bladder infection and was given a 10 day course of sulfonamide antibiotics (i.e. Septra™, Bactrim™). My PMS returned and again I was off to the library to discover why. Sulfonamide antibiotics are chemically similar to carbonic anhydrase inhibitors which are potent potassium wasters. In addition, bicarbonate is lost and chloride is retained so the form of potassium to be taken is an alkaline form, such as citrate, bicarbonate or gluconate. Potassium chloride cannot be used in this

instance so I took potassium gluconate. This time I had to use 1.2g K/day to notice improvement in my PMS and within three cycles of taking this amount, I was again free of symptoms. In the next year two of my friends decided to try potassium for their PMS. One took 600mg K/day as the gluconate and the other took 500 mg K/day as the gluconate. Each noticed a gradual decrease in intensity and duration of symptoms until they, too, were free of PMS. Both stopped taking potassium after a few months and they remained symptom free.

Potassium's Role in the Body

Potassium is one of the most important minerals in the body. Over 98 percent of the total body content of this electrolyte is located inside the cell. In general, it is about 30 times more concentrated inside the cell than outside. This high concentration gradient is essential for life and is maintained by a magnesium-containing enzyme within the cell membrane that pumps sodium out of the cell in exchange for moving potassium to the inside. It is the most abundant intracellular cation in the body and is involved at some level in virtually every life process. Its many roles range from assisting various enzymes, to ensuring proper nerve and muscle activity. High concentrations of intracellular potassium are required for maximal activity of protein synthesis. In addition, its concentration within the cell effects water retention, influences acid-base balance, enhances the production of adrenal steroids and is essential to the kidney's ability to concentrate urine. Potassium is important for proper carbohydrate metabolism, allowing unused glucose to be stored as glycogen in liver and muscle.

Depletion of potassium decreases insulin release in response to glucose. Progesterone production may also be related to the concentration of potassium. Drs. Bedwani and Wong of Cambridge Univer-

sity investigated the influence of the ionic environment on the progesterone production of rabbit ovary. They concluded that intracellular potassium was important for optimal production of progesterone by the ovary in response to luteinizing hormone.⁵ Although less than two percent of the body's potassium is located in the serum and other extracellular fluids, this small amount is nonetheless important. Adequate serum levels must be maintained for proper functioning of heart and muscle. The kidney is the primary organ that regulates the concentration of potassium in the serum. Excretion of this electrolyte is effected by certain diseases, acid-base balance, the activity of the adrenal cortex and some drugs. Severe losses of extracellular potassium can result in muscle paralysis and potentially fatal cardiac arrhythmias.

A potassium deficiency can result from a number of situations. These include the overuse of laxatives and bicarbonate, illnesses (especially those involving vomiting or diarrhea), ulcerative colitis and a diet low in potassium, which, more often than not, is the average diet. Deficiencies can also result from the use of many diuretic agents. The frequently prescribed sulfonamide antibiotics (i.e. Septra,[™] Bactrim[™]) are chemically similar to carbonic anhydrase inhibitors, which are potassium and bicarbonate wasters.

Excessive or prolonged fasting or starvation depletes potassium stores. In addition, high protein diets, liquid protein diets and protein-modified fasts, which are frequently used for quick weight loss, lead to a substantial loss of this mineral. Overactivity of the adrenal glands leads to an increase in steroid production with a resultant loss of potassium. The glucocorticoids act on the intestines to increase sodium and water uptake and to increase potassium secretion into the GI tract, thereby increasing potassium lost through the stool. Also, the general action of the glucocorticoids at the cellular level and

their effect on protein metabolism promote potassium depletion. The adrenal mineralocorticoids effect the transport of electrolytes and water distribution in the tissues. The main hormone is aldosterone, whose primary action is on the excretion of electrolytes by the kidney. It promotes sodium and water reabsorption back into the bloodstream and increases the urinary output of potassium. The glucocorticoids produce this effect to a lesser degree. For women, aldosterone secretion is lowest during the follicular phase. This level doubles during the luteal phase and increases up to tenfold during pregnancy. The estrogen in birth control pills also raise aldosterone levels. It is of interest to note that Drill's Pharmacology in Medicine states that the use of high doses of steroids can produce cyclic emotional symptoms.⁶

Pregnancy significantly increases the activity of the adrenal cortex. The high levels of progesterone present during this time block much of the potassium-wasting effects of the elevated aldosterone. Glucocorticoids increase two to threefold and are blocked only partially by the high progesterone concentration. However, even with the progesterone, body tissues are continually exposed to higher than normal levels of steroids for many months. In addition to the increase in these potassium-wasting hormones, the baby and placenta take potassium preferentially over the mother because they take from the serum which the body strives to keep constant, depleting intracellular reserves if necessary.

Adrenocorticotrophic hormone regulates the synthesis and release of all adrenal hormones, but only slightly that of aldosterone. An increased production of ACTH results in increased adrenal cortex activity. The release of ACTH is increased by physical and emotional stress, estrogens, surgery, exposure to cold, illness and depression. Besides ACTH, the administration of certain drugs also increases adrenal activity. Common ones are stimulants, such as nicotine, caffeine and theophylline.

Other minerals play a role in potassium metabolism. A high sodium or salt diet increases the body's requirement for potassium. A loss of chloride can lead to a metabolic alkalosis and this in turn can result in a potassium deficiency. Chloride deficiency can occur from loss of gastric juice (vomiting, gastric suction), profuse sweating and overactivity of the adrenal glands. Metabolic alkalosis usually accompanies the majority of potassium deficiencies. Magnesium deficiency eventually leads to a loss of potassium because of its role in maintaining the enzyme responsible for pumping potassium into the cell. A magnesium deficiency occurring in a pre-existing potassium deficiency increases the potassium loss even more.

Clinical conditions associated with the depletion of magnesium include congestive heart failure, diabetic acidosis, acute and chronic alcoholism, cirrhosis, and pancreatitis. In addition, depletion may occur after prolonged fasting, liquid protein and high protein diets, persistent vomiting, ulcerative colitis, extensive burns, in some kidney diseases and a poor diet. A U.S. government survey found that the average American diet supplies only 40% of the RDA for this mineral.

The most common lab test performed to evaluate potassium is a blood test measuring the serum potassium level. The normal range is typically 3.5-5.0 meq/l. Since potassium is found primarily inside the cells and the serum potassium represents only that small fraction present in extracellular fluids, this test is not an accurate indicator of the true status of total body content. Serum is the last body storage area to be depleted of potassium. Low intracellular levels can occur in the presence of normal serum values, as proven by tissue biopsies and isotope dilution methods. Most doctors do not treat for potassium deficiencies, even if symptoms and history suggest it, unless serum levels go below 3.5 meq/l or if an EKG shows the abnormal

electrical activity of the heart typical of advanced potassium depletion. Unfortunately, potassium levels are usually assumed adequate if the serum level falls anywhere within the normal range, even though it is well established that this is not always the case. If a potassium deficiency does exist, eating potassium-rich foods will usually not significantly restore it. A potassium-rich diet is very important in maintaining a healthy potassium level, but if a deficiency exists, supplements must be used. This is because the acid-base imbalance that usually accompanies the potassium loss must also be corrected, so the counterion becomes important.

Often sodium, and therefore salt, is restricted from the diet because sodium increases the body's need for potassium. In the case of an alkalosis where salt may be restricted, potassium chloride must be used to correct the imbalance due to the accompanying chloride deficit. If salt is not restricted, potassium gluconate can also be used. If an acidosis is present, an alkaline potassium such as the citrate, bicarbonate or gluconate (this is considered alkaline because it is eventually metabolized to provide bicarbonate) form must be used. The dosage generally prescribed to treat deficiencies is 1.6-4.0g K/day. In response to a rise in the concentration of body potassium, the rate of its excretion increases. Therefore, with normal kidney and adrenal function, it is difficult to produce potassium intoxication when potassium is given orally. Toxic elevation, or hyperkalemia, is generally limited to those persons with kidney failure and Addison's disease.

Other conditions in which potassium should not be given include advanced dehydration, heat cramps, immediately following extensive tissue breakdown as in severe burns or crushing injuries, inadequate urine output, shock and while taking potassium-sparing medication. Hyperkalemia can also develop if potassium is accidentally administered too quickly

by intravenous infusion. Common symptoms of potassium depletion are muscle weakness, fatigue, constipation, irregular heartbeat and water retention. Some of the neuropsychiatric symptoms include confusion, poor memory, irritability, nervousness, headaches, lethargy, apathy, muscle weakness, visual disturbances and depression. Common PMS symptoms include all of the above.

Treating PMS with Potassium

PMS has been noticed to most frequently occur or worsen after potassium-wasting drugs and situations such as pregnancies, surgeries, illnesses, stressful episodes and drugs, such as caffeine. I brought these observations to the attention of a doctor at a university research hospital who was interested in PMS. He said that he would be interested in observing women with PMS take potassium. I then conducted a year-long clinical trial for him to observe using seven women whom he validated as having severe PMS. (Since this was not a hospital-sanctioned research project, we could not legally use placebos. Therefore, in order to minimize placebo effects as much as possible, only women with severe PMS were used). Each kept daily symptom calendars, rating each symptom on a scale from 1(mild) to 7(severe). Each charted two cycles before beginning potassium in order to establish a baseline pattern and continued charting daily for the next four to five cycles after beginning the potassium.

Most subjects took 400 mg K/day as the gluconate and 200mg K/day as the chloride for the first two cycles. From then on only the gluconate form was used. In one case where one of the subjects had taken a recent course of sulfa antibiotics, it was decided she would take only the gluconate form. Two of the subjects took 800mg/day.

Without exception, all the women found their symptoms decreasing in intensity and duration over three cycles until

they were free of all PMS their fourth cycle. There is a definite progression of improvement throughout the first three cycles of potassium supplementation that seemed common to everyone in the study. Physical symptoms, such as fatigue and sensitivity to light and noise, seem to get better faster than the emotional symptoms. Mild irritability seemed to be the last to disappear. Abdominal bloating usually began to decrease immediately except with two of the subjects who consumed a lot of salt and did not want to lower their sodium intake. For them, water retention actually became worse during the first couple cycles on potassium. However, this symptom also resolved completely by the fourth cycle even when salt intake remained high. Acne was a symptom that could become slightly worse before it resolved. The first cycle on potassium sometimes increased breast tenderness and menstrual cramps. Breast tenderness then resolved gradually over the next couple cycles and cramps returned to normal baseline.

One of the most interesting observations was a shifting of symptoms into the period, which was usually a symptom-free time for the subjects. This occurred only after the first cycle on potassium. Unfortunately, at the end of this preliminary study, there was no money to pursue a double-blind medical study. Obviously, no drug company would be interested in funding a study on potassium since it cannot be patented, so that ended the research.

Over the years I have worked with a number of women who have taken potassium for their PMS. (These women were asked to first check with their doctors). So far potassium has never failed to work. There is, however, a very definite protocol to successfully use potassium for PMS.

First, most people do fine on 600 mg K/day as the gluconate. Potassium gluconate is relatively mild on the GI tract as compared with the chloride form and as long as the person uses a little salt they are

getting adequate amounts of chloride. Also, there is a balance between sodium and potassium and I didn't want anyone severely restricting their sodium intake. For a small percentage of women the quantity had to be doubled to 1.2g, however, I cannot say that a smaller amount may not have worked, such as 1.0g.

Second, potassium must be used everyday. This is critical to success. A common occurrence with some women not on the study was forgetting to take the potassium after a couple months, when they were feeling much better. Prematurely stopping the potassium for just a few days can result in the complete return of all symptoms. The body adapts to a particular intake and can quickly excrete potassium. Going back on the potassium will work again but it will be as if the person is starting from day one.

Third, potassium supplementation must begin early in the cycle. If supplementation begins after the first five or six days very little if any improvement may be noted that month. I noticed that many women who began mid-cycle actually had increased breast tenderness and cramps for that cycle only (in addition to their usual PMS symptoms) and I began to interpret this as a good sign in that they were on the correct dosage, as they never failed to improve substantially the following month. All women in the study began on day one (this corresponds to the first day of the period).

Fourth, do not use phosphorus-free calcium supplements (carbonate or oyster shell, lactate, citrate, gluconate, etc.). Doing so for the first few months of taking potassium seems to completely stop PMS from improving. After the PMS is gone for a month or so, these calcium supplements could be used with no return of PMS. I have no idea why this is but this is a reproducible result. Dairy products do not have this same effect so the women on the study were told to get their calcium solely from

their diet and to avoid calcium-fortified products.

After the PMS is gone for a couple months, it is possible to discontinue the potassium gradually and experience no return of symptoms. However, chances are the person is going to have to change their diet to include potassium-rich foods and limit potassium-wasting items such as caffeinated drinks. One can always safely, with the exception of certain diseases, continue to take a half gram of potassium each day to make sure they are getting enough.

Conclusions

Based on my own experience with PMS, as well as working with other women over the years, I want to propose the following: PMS is the result of a mild to moderate potassium deficiency which is not severe enough, necessarily, to cause symptoms all of the time or be detected by routine lab tests. It is worth noting, however, that the majority of women who have taken potassium for their PMS noticed a substantial improvement in their overall energy level during their non-symptom times as well.

If optimal levels of potassium are required for the proper functioning of some cyclic hormone, like progesterone, or some other cyclic substance, such as an endogenous opiate peptide or serotonin receptors (which have been shown to fluctuate with changes in estrogen and progesterone concentrations), a mild to moderate deficiency would manifest itself by causing a disturbance in the metabolism of this substance.⁷ As this potassium-dependent substance fluctuated with the menstrual cycle, than so might symptoms. One of the women on the study was the secretary of a local PMS Society chapter who was already taking natural progesterone suppositories. (She was the only subject who was on any treatment for PMS before the study). She was too afraid not to use her progesterone and decided to wean herself off gradually once

she began taking the potassium. Progesterone is used from about mid-cycle on so when she began taking potassium she was not on the hormone yet. When she started out using her normal progesterone dosage, (during her first cycle on potassium) she found herself experiencing the same symptoms she had when she had once taken too much of the hormone. Cutting back on the progesterone made her overdose symptoms disappear. It was as though the potassium was making her body more sensitive to the hormone. Within a couple cycles on potassium she was able to give up her progesterone and by the fourth cycle was free of PMS. I kept in touch with her for more than a year after the study concluded and although she elected to continue taking potassium, she never had PMS again.

One important study on PMS was conducted by Dr. Guy E. Abraham and co-workers at UCLA.⁸ They evaluated the serum and erythrocyte magnesium levels of 26 women with PMS and nine symptom-free women. They found that although their serum levels were comparable, the women with PMS had statistically significant lower intracellular magnesium levels than the controls. Potassium deficiencies occur more readily than magnesium deficiencies.

Many of the things that cause low magnesium levels are also direct causes of potassium deficiencies. A magnesium deficiency increases the potassium loss even more. In view of these facts and Abraham's results, it is certainly possible that women with PMS have lower intracellular potassium levels than women without PMS. A large percentage of women have PMS. Is it unreasonable to believe that so many people have potassium deficiencies? Not when you consider what people actually do to themselves. The food selection of most people is high in refined and processed foods, dairy products, fats, sugar salt, and caffeine. Although potassium is found in many foods, it is not found to any great extent in these. This type of diet, coupled

with the physical and emotional stress of modern life, pregnancy and illness increases the probability even more for a low potassium level. Diuretics are used to excess and potassium is often not given in conjunction with many potassium-wasting drugs prescribed. Persons with mild to moderate potassium deficiencies are truly in a bind. Not only can they not fully eat the potassium back, but the problem probably won't even be diagnosed. Unfortunately, most doctors only treat for potassium deficiency if it is severe enough to show up in a test. Is a potassium deficiency too simple an explanation for an illness that has so many different symptoms? Potassium deficiency symptoms are known to be numerous and diverse. The ramifications of lowering one of the most abundant intracellular minerals should be expected to alter all major body functions to some degree, depending on the individual's biological make-up. It is probably no coincidence that many potassium deficiency symptoms are the same as many PMS symptoms and that PMS develops or worsens after potassium-wasting drugs and situations.

Author Note: A detailed protocol is available on request.

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