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VIRTUAL LAF CONFERENCE

Proceedings of 62nd Session
March 3rd, 2008 –

SUBJECT: LAF and Migraine

ANP = atrial natriuretic peptide
PFO = patent foramen ovale
ASD = atrial septal defect
MVPS = mitral prolapse syndrome
OSA = obstructive sleep apnea

Two months ago an interesting article, entitled “Relationship Between Migraine And Patent Foramen Ovale: A Study Of 121 Patients With Migraine,” appeared in the journal Headache.

http://www.medscape.com/viewarticle/566276_print

In addition several recent posts on the BB have again raised a possible connection between PFO and LAF. This latter has always fascinated me, since I have both a PFO and migraine with visual aura. Could this ASD have contributed to the development of my arrhythmia and ironically provided access to my left atrium for Prof Haissaguerre’s ablation? I even asked him at that time in 2005 if he knew of any association between PFO and AF.

PFO is a one-way valve in the septum between both atria, enabling a right to left shunting of blood under certain conditions, e.g., Valsalva maneuver. It normally closes at birth, but can persist in 15% (echocardiography) to 27% (autopsy) of the population. LAF, on the other hand, develops later in life and is much less frequently encountered. Clearly any association between the two can lend insight into only part of the LAF enigma. Could PFO contribute to the development of the trigger substrate (v. maintenance substrate) of LAF, i.e., PACs?

ANP and BNP in those with ASD (PFO is the most common ASD) are significantly higher than in controls and decrease to normal values within 3 months post closure.

“Quantitative Evaluation Of The Changes In Plasma Concentrations Of Cardiac Natriuretic Peptide Before And After Transcatheter Closure Of Atrial Septal Defect”

<http://www.blackwell-synergy.com/doi/abs/10.1111/j.1651-2227.2002.tb03295.x>

It is stated in the above introductory article on PFO and migraine

“The most likely explanation of the coexistence of PFO and migraine may be the study of Yankovsky et al.[63] Yankovsky et al[63] recently reported altered atrial natriuretic peptide (ANP) levels in migraine patients. ANP released from atrial myocytes is known to cause vasoconstriction and platelet aggregation. Thus ANP might also play a role in pathophysiology of migraine.”

Yankovsky has even reported an acute increase in migraines immediately following ASD closure.

<http://www.ncbi.nlm.nih.gov/pubmed/12752756?dopt=Abstract>

Magnesium is known to prevent hypertension and to have anticoagulant properties. Could a deficiency of magnesium (rather than ANP directly) explain the above vasoconstriction and platelet aggregation?

Natriuretic peptides certainly cause urinary sodium wasting, as the name implies. However, not many seem to know that it also causes urinary magnesium wasting. Indeed it is rather shocking that the ANP, BNP/urinary magnesium wasting “dots” have not been connected for either MVPS or PFO, both of which increase these peptides.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=2976727&dopt=Abstract
<http://jcem.endojournals.org/cgi/content/abstract/66/3/465>

In Session #51 of the CR Proceedings (mid 2006) the connection between ANP and LAF was explored

<http://www.afibbers.org/conference/session51.pdf>

and the potential for urinary magnesium wasting posed by the natriuretic peptides has been previously underscored

[http://www.medical-hypotheses.com/article/S0306-9877\(06\)00563-9/abstract](http://www.medical-hypotheses.com/article/S0306-9877(06)00563-9/abstract)

In addition to exacerbating magnesium deficiency ANP also has vagotonic properties – bradycardia, increased HRV. Furthermore, the natriuresis leads to a chronic hypovolemic state, perhaps enhancing sensitivity to catecholamines. Therefore, it may not only impact the trigger substrate via electrolyte imbalance but also maintenance substrate via autonomic factors.

Recall the finding of low or low normal intracellular magnesium (range 30-35 mEq/liter v. normal range 34-42) in all seven LAFers responding to that question in LAFS-11.

If there is an association between PFO and migraine/LAF, why do the latter conditions afflict primarily adults?

Perhaps this is because the foramen ovale approximately doubles in size by adulthood.

“In the newborn the foramen ovale is about 3.85 mm and in the adults about 7.2 mm in length. The width extend from 1.81 mm in the newborn to 3.7 mm in adults.”

<http://lib.bioinfo.pl/pmid:6486466>

Perhaps the process of deteriorating magnesium absorption as we age and the gradual depletion of total body magnesium via natriuretic peptides are long and drawn out.

There have also been numerous posts on the BB about the association between OSA and AF.

“Obstructive Sleep Apnea, Obesity, And The Risk Of Incident Atrial Fibrillation”

<http://www.ncbi.nlm.nih.gov/pubmed/17276180?dopt=Abstract>

Conclusion: Obstructive sleep apnea is a risk factor for AF independent of obesity.

“Prevalence of Patent Foramen Ovale and Its Contribution to Hypoxemia in Patients with Obstructive Sleep Apnea”

<http://www.chestjournal.org/cgi/content/abstract/113/1/91>

Combining these two findings leads inevitably to the role elevated natriuretic peptides in OSA may play in increasing the odds of developing AF?

You might now be asking whether migraines are increased in those with LAF, i.e., is there more than anecdotal evidence for this.

Well, it just so happens that Hans in LAFS-6 looked at this very question. The January 2004 AFIB Report detailed the results of a survey of 65 LAFers with 23% female and 77% male respondents. Prevalence of migraine was 12.3% v. 10.3% in the general U.S. population. The logical conclusion was that “it is unlikely that there is an association between migraine headaches and LAF.” However, migraines are more frequently encountered in females by a factor of three. Given the gender differential in both the survey and migraines, it appears that migraines are encountered about twice as frequently in LAFers v. the general population (12.3% v. 6.0%). Furthermore, magnesium deficiency seems to be more strongly associated with migraine with aura v. migraine without aura, the most common variant.

<http://www.ncbi.nlm.nih.gov/pubmed/12110110>

It would be interesting to hear how many of you have migraine with aura or optical/ocular migraines. The latter are another variant in which there is visual aura without headache. A comprehensive discussion is available at

<http://en.wikipedia.org/wiki/Migraine>

Migraines have been strongly associated with PFO and MVPS, which frequently coexist. Magnesium deficiency is well recognized as a contributing factor to many of the symptoms in MVPS, and magnesium supplementation is a recommended therapy for migraines. We all know the important role magnesium plays in avoiding LAF episodes. Could ANP be the common denominator linking all of these entities?

The above observations are speculative in nature. Association does not prove causation. However, the association between ANP and LAF/MVPS/PFO via a magnesium deficiency mechanism is provocative. The association between migraine and these three conditions introduces additional intrigue.

Your feedback on the below speculative comments would be most welcome. If you suffer from migraines in addition to LAF, please describe them for us. If there appears to be an association between LAF and auras, perhaps such data might be formalized via a future survey by Hans.

PC

PC,
Your information is very interesting. A bit above my head.....but interesting.

I do have a history of migraine with aura. In fact, my gp put me on Inderal approximately 8-9 years ago to help reduce the frequency and severity. After being on 60 mg a day and having a slight reduction in the migraines, but not enough, the dr. changed it to 120 mg time release each day. My HR was very, very slow and I was concerned, but he said it was fine. This last level stopped the aura and reduced the frequency and severity of the migraines even more. So there I stayed.

Approximately 2 years later, out of the blue, had my first episode of a-fib. I have always wondered if my slow HR from the Inderal was a trigger for the start of my a-fib.. I will never know.

Another interesting aside is that I almost always check out low on sodium. In fact, a surgery was canceled by the anesthesiologist due to low sodium on pre-op. My magnesium has not been checked for over a year, but last year I had an ION done and my magnesium was low and my calcium was so high it was almost off the charts (and I don't even take any calcium).

Anyway, I feel like I have written a book. Don't usually write much. I do still have migraines, but not as often. When I do, they can be pretty severe but better than in the past.

Hope this helps. Thanks for all the information. I plan to read the links.

Jodi

PC - this whole subject interests me as well, as I've often wondered about the similar triggers between migraine sufferers and LAF's. I do not have migraines, but my daughter does. (not with the aura though).

I WAS told that I had MVP for 20 years, and then told that I did not, so, not sure what that was all about. I had something else happen after I delivered my first child that makes me wonder about the "urinary wasting". After my caesarean (sp?), I had trouble urinating so was given a catheter and emptied out about a quart. About an hour later, I repeated this, much to the amazement of the nurses who thought I couldn't possibly have to "go" again. I also get up often in the middle of the night when I am having an afib episode....and then find out I have sometimes lost almost 3-4 pounds overnight and reduced my blood pressure quite a bit.

I know this is not about migraines, but you did refer to the "urinary wasting" and ANP, and I'm wondering if any of the above means anything to you, as you are clearly much more versed on this than most of us are.

Thanks for bringing this topic up. I'm sure there will be a lot more people with interesting things to add here.

Barb H.

PC

i'm not sure if i'm getting it please correct me if i'm wrong and add anything to it, i've tried to break it down into my thinking

ANP, hormone produced by the atrial myocytes in response to stretch/high blood pressure, target organ the kidneys to adjust water and Na⁺/K⁺ etc from the blood the decrease burden on the heart

does PFO, ASD, MVPS contribute to the stressors to that would make the myocytes produce ANP

will ANP increase Mg wasting ?

what part of the process is causing the migraine, the high blood pressure before or something about the ANP when its released decreasing Mg content in the body

please input or correct any part of my breakdown as simple as possible :)

Tonigirl x

Hi all,

Visual migraines and full visual/aura/scintillating Scotoma history.

Had migraine from puberty, visual with lightening strike pattern and tunnel vision. Always on right side of head, left hand would go numb. I was terrified, age 11, and undiagnosed by family doctor at that time. At school the teacher would have me putting my left hand under the cold tap and hot tap alternately. I would vomit and the migraine would end, leaving me washed out.

In adulthood I continued to have visionary migraine, slightly different, where I would see golden coloured comma's, have vicious one-sided headache always on right-hand side. They would last all day, only going after sleep, leaving me ill sometimes for a couple or 3 days. A curious thing with them was that if I had to write anything during one, I frequently missed the last letter off many words I was writing!

More frequent in 1982, when I discovered they were caused by MALT (from Barley). As a child age around 9 - 11 I hid in the under stair's cupboard and ate Horlicks (malted milk powder) by the jarfull! I was addicted to Horlicks tablets (like a sweet made from Horlicks) when pregnant. Now paying for my sins!

I then found out in 1984 that Chocolate eaten at night produced bad migraine.

As a child I had migraine if I drank a small glass of red wine that my mum would sometimes give me.

I added caffeine to the list of banned foods around 1987. Withdrawal for a few hours one morning produced the most severe migraine bout ever, with vomiting, and odd bubble shaped liquid filled small circular things that lined my lower eyelids, scratching my cornea as I closed my eyes. I could not believe that this could be caffeine withdrawal so went back to tea and coffee.

I repeated the exercise two weeks later, same result. I was having a driving lesson at the time, cancelled the rest of them, went home by bus, needing to vomit and eying up my carrier bags of shopping (bought on the way home) in case I did vomit. Three days in bed. No more caffeine - just longing looks at a cup of tea for WEEKS afterwards.

Then four years ago at the age of 56, had true visionary migraine. I thought I was going to die as I thought it was a stroke coming on.

It started off as a small circle of bright light, like what happens when you look directly at a light-bulb then a wall. It grew bigger and bigger, appearing to move towards me at a steady rate. The circle had the most beautiful colours flashing all around the edges in a zig-zag pattern, pinks, purples, shiny, but the middle of the circle obscured my vision - I could

only see with peripheral vision, scared silly! Called the doctor, and optician, got two appointments for that afternoon. The circle got bigger and bigger and began to break away making a "C" shape, and was now it appeared surrounding my head like a halo tipped on edge - it appeared to pass towards my ears and beyond - then was gone! All that remained was a very frightened woman with a very mild headache! It lasted about 15 - 20 minutes as I recall.

I later went on the Internet as my optician, quite rightly, said it was a true "visionary migraine" called a Scintillating Scotoma. I found a site and added my post of the event and felt tremendous relief to know others had them and that it was harmless.

I told my sister (who is 2 years younger than me) about it. She also has a history of migraine, and she had a SS just 3 months later. She had to sit in her Bank until it passed, but was not frightened as I had told her what happened to me. My sister also has AF, in her case due to alcohol addiction.

Hope those reading this are not scared if they have a Scintillating Scotoma as they will now know what it is.

Heather h

hi, I get the visual aura without headache. I have to watch what i eat also. No chocolate, red wine, peanuts. I also cannot skip meals and i believe weather changes affect me also. I have had these since my teenage years and i handle the anxiety better now because i know i have had them before and other than the inconvenience of the eye aura were harmless. I have LAF and have had it for more than 5 years. I do however notice that I am getting more of the auras in recent years. I also have dry eye which my eye doctor said could stress my eye and possibly cause the auras.

Steve

PC - good post and interesting topic. I only can offer a minor contribution.

My personal experience is the aura/migraine from hormonal imbalance when I was perimenopausal. Once the hormones were balanced, the migraines disappeared and it was almost 10 years later that the onset of AF arrived for me.

I did have a neighbor who had PFO and the AF; she had an ablation that was mostly successful (still would get AF if she drank alcohol). At the time, I didn't have AF so I didn't question her much about the incidence of the PFO and AF.

Jackie

Re: If there is an association between PFO and migraine/LAF, why do the latter conditions afflict primarily adults? Perhaps this is because the foramen ovale approximately doubles in size by adulthood.

"In the newborn the foramen ovale is about 3.85 mm and in the adults about 7.2 mm in length. The width extend from 1.81 mm in the newborn to 3.7 mm in adults."

Why would that explain it? A baby's PFO is proportionately larger than an adult's.

Sziv

Aloha PC,

Glad to have the pleasure of reading your contributions again!

I don't have a lot to add, however my wife (not an afibber) does suffer from migraines. After seeing this post of Jackie's in December http://www.afibbers.net/forum/read.php?f=6&i=19739&t=19628#reply_19739

I ordered the book, "What Your Doctor May Not Tell You About Migraines: The Breakthrough Program that Can Help End Your Pain" by Alexander Mauskop, MD and Barry Fox, PhD for her.

This book suggests the following:

http://www.townsendletter.com/Nov2007/book_migraines1107.htm

1. Get a proper diagnosis from a medical doctor.
2. Use the triple therapy.
3. Identify and avoid your migraine triggers.
4. Eat to avoid migraines.
5. Take the edge off.
6. Walk it off.
7. Use medicines as necessary.

The "triple therapy" advised in the second step is integral to this program and was developed by Dr. Mauskop at his New York Headache Center. When he was not getting very good results from standard medications, he first turned to magnesium and found through established research and his own research, that magnesium indeed could play an important role in preventing migraines and reducing their severity. The herb feverfew, which has been known as a successful headache treatment for hundreds of years, was added to the regimen to strengthen the therapy. Then, recent research showed riboflavin was able to prevent migraines, and it was added as well.

The three components "magnesium, feverfew, and riboflavin" are combined to form the triple therapy, which is to be taken daily in oral supplement form and likely for years. The triple therapy is discussed in great detail with emphasis on the important role of magnesium. Dr. Mauskop found in his studies that those patients lowest in magnesium benefited the most from magnesium infusion. He also found that the standard measure of total magnesium was not adequate. After new laboratory techniques were developed, it was possible to separate the bonded magnesium levels from serum ionized magnesium, or free magnesium, levels. Mauskop's studies were then able to confirm that the free magnesium levels were more relevant to migraine treatment than the total magnesium level. It also became possible to look at the ratio of the serum ionized magnesium and serum ionized calcium, which must maintain a proper balance for appropriate muscle contraction.

My wife takes magnesium as glycinate and additionally started using the product, MigreLief <http://www.migrelief.com/index.htm> as well. It contains magnesium, feverfew, and riboflavin. Although it is still early days on this program, she thinks it helped her avoid her monthly migraine (hormone based).

Cheers,

George

Thank you all for taking the time to post your experiences. Hopefully there will be others.

It would also be interesting to know if those with migraines are more frequently afflicted with muscle cramps and/or fasciculations (muscle twitching). All have diminished greatly for me since paying more attention to Mg and K.

Barb,

That's an interesting story. MVP and PFO are frequently encountered in the general population and often fly under the radar on echocardiograms and cardiac auscultation.

Tonigirl,

Don't sell yourself so short. You've got it all correct. It is all related to intra-atrial pressure and distention => increased ANP. AT1s (angiotensin II type 1 receptors), which are a marker for stretch, are increased in LAFers.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=14642689&dopt=Abstract
<http://circ.ahajournals.org/cgi/content/abstract/101/23/2678>

Heather,

Talk about visual aura. I also remember well my first visual aura. It lasted several hours while travelling in Singapore about 25 years before my first episode of AF. Counting me, that makes it 4 for 4 on visual aura in those with LAF and migraines. Hans, does that sample size enable statistical significance (ha-ha)?

Sziv,

You appear to have lost sight of the forest for the trees. My concluding comment (the forest) was "Perhaps the process of deteriorating magnesium absorption as we age and the gradual depletion of total body magnesium via natriuretic peptides are long and drawn out," was the take home comment here.

Regarding increasing size of PFO as we age =>? more RLS => more ANP => more urinary magnesium, relationships are not always linear. By this I mean that perhaps the cellular threshold for triggering the upregulation of AT1s (see above reference) or ANP production or ? does not change from newborn to adult. Furthermore, a doubling of a diameter means a quadrupling of area. And we're talking about blood flow and pressure. But I wouldn't concentrate too much on this one rather speculative remark.

George,

Thanks again for your invaluable and informative contributions. Not a day goes by that I don't thank my stars for NSR due to the efforts of Prof Haissaguerre.

PC

PC:

So good to see you posting, I always learn something. I too get Aura Migraines, they start as a tiny zigzag light which gets larger and interferes with my vision. I do get a slight headache and it will last for about 30 min., then it will clear, I sometimes feel a little dragged out for a while afterwards. I have a slight MVP, seems to remain the same and told not to worry. My afib has lessened, I had gone for about 9 months without an episode, taking one tab. of propafenone (125mg.) at night before bed, I am a classic vagal. Then last year around May, I started getting a few episodes--once a month, then twice a month, the last episode was about 6 weeks ago.

Last March (07) I started using a bio-identical hormone cream--I use the cream for 25 days, stop for 5 days and resume. My migraines have greatly lessened, I have noticed however that when I stop the cream after the second or third days I will get the aura. I spoke to my Holistic doc and he said I needed more mag., I know I always test towards the lower end of the values. I take mag., called Ionic Fizz from IHerb, my doc. had said it seemed fine, it is supposed to be more absorbed, still isn't enough. My doc said he now has mag. in a spray form that is absorbed epidermally and finds this to be much better. I will pick it up next week and try it out.

My mother had aura migraines as well, also, afib in her later years. She never had all the tests that I have had, so I don't know when and if she always had MVP, but, when she was in her last few years of her life she had MVP. My grandmother died in her late seventies from what they called at that time leakage of the heart, she was very weak, I believe she probably had CHF.

My mother said that my grandmother used to lay down because of bad headaches---my granddaughter gets a migraine once in a great while--seems to get it from being in the hot sun, she doesn't get the aura however.

So, PC perhaps this is more than you wanted to know---hopefully it will be helpful. At least you are in a warm climate, I live in Michigan and it has been a very cold and snowy winter, global warming be damned.

Warm regards

Liz

PC

thanks for the advice I will remember that for the future one of my teachers said a similar thing, I need to build on my confidence in understanding because I can do it

as for migraines I don't get them often, but I call mine hungry and dehydrated headaches, its comes on behind one eye, if I eat and drink something at this point and take a couple of painkillers I can usually get away with a full blown migraine, but if I don't and the pain gets worse, I become nauseas and have to lie down in a dark room with my eyes shut untill it passes, it this point nothing I can take will work to ease it

I would never have linked it to ANP and Mg, I might increase Mg as I am not taking that much at the moment

Tonigirl x

PC, I remember you were doing this conference and thought I would add something to it. Years ago I had my first encounter with the "aura lights" when I had handled a plastic product with petroleum in it for several hours. I went to the ER and while sitting in the waiting room the aura went away. I had more of them over the years and was diagnosed with an allergic reaction to latex products, especially those with petroleum. I was put on a medication but couldn't take it for the side effects. I now just let them ride their course. My youngest son has the same problem although his allergic reaction shows up in eczema on his hands. Interesting note though - he also has afib, diagnosed last year.

I try to stay away from "known" culprits, but it's hard. I have had several "aura" over the years, they last exactly 20 minutes or less and I have never had a migraine, but do have a very slight headache for an hour or so and distortion of vision. The worst it does for me is it makes me very tired. I feel totally wasted.

This past Sunday I had the "aura" of all auras. I had an afib episode early Saturday morning and after 20 hours went to the ER. Of course they immediately did an EKG. I never thought anything about it because I have had "quick" EKG's where they immediately remove the pads and wires after the test. Well, this time the pads stayed on for about 4-5 ER hours and I was not aware they had accidentally left four pads on my leg and my chest when they removed the others. When I went home at 4:30 a.m. I slept for several hours before getting up and taking a shower. That is when I found the pads and took them off, my skin was red where they had been. That afternoon I had not one of the "aura" episodes, but three, one behind the other, about two hours apart. First the flashing lights, lasting exactly 20 minutes and then I had a bad headache for about 3 hours. I also lost some of my vision in my eyes, I could read something, but could only see it in tunnel vision, I couldn't read anything except what was right before me. I was so exhausted already from afib and then this allergic reaction. The doctor said it was probably the jell and the latex on the EKG pads. I had just never had three of them in one day.

Now, I really wonder how many times I may have been exposed to petroleum products which could have eventually led to my having afib even though not all the time causing the "aura and migraine" type effect.

Just thought I would share my experience if it will help. By the way I am okay now (Weds), full vision recovery, no more headache. I am still tired, but hope to regain my energy.

Sharon

If I could add to this. I have suffered from migraine with aura for the last 30 years but only maybe two or three a year, these attacks being the usual bright lights around the outside of my vision, headache etc. I started to suffer from AF about 2 years ago and last week had an appointment with my cardiologist for a follow up to cardioversion last Dec .I have noticed that the migraine attacks recently have increased to about 2 per month so I ask the cardiologist if there is a link between AF and migraine, his answer was no but there is potential link between migraine and hole in the heart (PFO) and started to check the tests he had done over the last 2 years. An ultrasound from 2006 had showed a small PFO which he had not told me about due to it being very common. He suggested I see a Neurologist and possible

inclusion in trials that are underway in the UK into the possible link, I decided to monitor the migraines for the near future, but I can't help but wonder if the link goes further than just migraine.

Steve

Hi Sharon and Steve,

Both experiences are fascinating. Whether there is a connection between LAF and migraine with aura at this point is difficult to say. The above evidence is only anecdotal.

Migraine with aura is less common than migraine without aura.

While it appears that migraine, not otherwise specified, is more common in LAFers v. the general population, I would hazard a guess that migraine with aura is considerably more common in LAFers v. the general population.

Furthermore, amigrainous migraine with aura and its variants in which the migraine is minimal camouflage the association with migraine and LAF.

PC

For those of us with a limited vocabulary:

Acephalgic migraine is a neurological syndrome. It is a variant of migraine in which the patient may experience aura symptoms such as scintillating scotoma, nausea, photophobia, hemiparesis and other migraine symptoms but does not experience headache. Acephalgic migraine is also referred to as amigrainous migraine, ocular migraine, or optical migraine.

From: http://en.wikipedia.org/wiki/Migraine#Acephalgic_migraine

George

Women that have aura migraines have a greater propensity to having a stroke, not too comforting. PC what do you think about a possible stroke connection with aura migraines. I will say that my mother had aura migraines and didn't have a stroke. I have had aura migraines off and on since my late teens, which is many moons ago. In Georges' url--hypothyroidism was a culprit, it does seem that I have had more auras since I had my thyroid nuked. I have had hypo periods, I will keep a log.

Liz

When I mentioned my aura to my GP after I had the afib episode he responded without even hearing me out about my allergy he said, "You have probably been throwing tiny blood clots in your brain." That upset me so bad, I stopped him and said, "Wait a minute, you didn't hear what I said, I have had these for years, I have studied them, I know what the lights look like for my situation and I know what to look for in the event it is a blood clot." I then carefully described the aura to him, he backed down and recognized what it was and that it wasn't me "throwing tiny blood clots." I was upset when I walked out of his office and I wondered how many people don't know enough about the "aura" signs and end up being treated for stuff they don't have. If I had not been knowledgeable about this, that would have scared me to death. With that said, I think I read somewhere that everyone throws small clots often and they don't even know it. People need to be aware or you will end up with medication and/or treatment you don't need. I am sure his recommendation would have been blood thinner.

Sharon

Hi Liz,

The connection between ischemic stroke and migraine with aura in females is well-known.

http://heartdisease.about.com/od/otherriskfactors/a/migraine_aura.htm

Thank you for bringing that up.

Several articles suggest that the mechanism for the aura is most likely vasoconstriction to compensate for poor autoregulation at the microcirculatory level in the posterior lobe of the brain (the "visual cortex").

<http://neurology.jwatch.org/cgi/content/full/2002/308/1>

It also turns out that there is a correlation between mitral valve prolapse syndrome and migraine as well,

Increased prevalence of mitral valve prolapse in patients with migraine.

<http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1440340>

and between mitral valve prolapse and migraine. Just google migraine and MVP.

In Session 59 of the CR Proceedings on Dysautonomia and LAF (third posting) I suggested that perhaps adrenergic LAF was a function of low blood volume. Indeed several experts on MVPS have suggested that it is due primarily to low blood volume, but none have implicated ANP in this process.

Could migraines also be a reflection of low blood volume? If migraines, LAF, MVPS are all associated with low blood volume (and migraines), then perhaps this is associated with increased adrenergic activity to maintain BP. Since catecholamines have not been reported increased in any of these entities, perhaps beta receptors are hypersensitized to maintain BP. This latter would certainly explain the reported excessive sympathetic stimulation and dehydration that accompanies migraines.

In short, I think mainstream medicine is putting the cart before the horse. These conditions are not a function of dysautonomia but a function of low blood volume that creates dysautonomia.

I personally believe that increasing water intake and potassium supplementation is the best course to follow.

Magnesium is also important, but is less important than K. I believe this is because K⁺ supplementation helps spare Mg⁺⁺ from the ravages of aldosterone and because it also shifts the BP regulation equation.

BP is determined by beta receptors and blood volume. That's why beta blockers and "water pills" are the cornerstones of anti-hypertensive therapy. Regulation of blood volume is a function of aldosterone and the latter is regulated by K/Na, RAAS (renin angiotensin aldosterone system) and ACTH. All else being equal, if we increase blood K⁺ => K/Na increases => aldosterone increases. But because blood volume hasn't increased, this means that the other two inputs, RAAS and ACTH, must pull back marginally => decrease in angiotensin (very powerful vasoconstrictor) => BP decreases. This is why K⁺ is such an effective anti-hypertensive.

I have not experienced a migraine with aura for several years and am not sure whether this is due to better hydration or K⁺ intake.

Whatever the risk of stroke in those with migraines, K⁺ and H₂O have to be a good thing. We really can't do much about the low blood volume driven by natriuretic peptides, which are known to be increased in those with migraine, LAF and MVPS.

Many of the above comments are speculative in nature.

PC

PC, what is the difference in "natriuretic peptides" and "B-Type Peptides"? When I had my last afib episode March 8th, I went to the ER and my blood work showed my B-Type Peptide at 248 with normal being 0-100 ph/ml. I just wondered what that was as it appeared as part of my blood work. Sorry, hope this isn't off the conference topic, but my aura event took place a few hours later that same day, so didn't know if there was a connection.

Sharon

PC - I have always been wary of taking supplemental K, as the doctors I've spoken with (General Practitioners) have steered me away from that and said to get my potassium from food, not supplements. Yet so many people on this board seem to be taking K in supplement form. I would welcome your opinion on this.

And if supplemental potassium is OK in "your book", how much is safe to take?

Thanks

Barb

Hi Sharon,

ANP stands for atrial natriuretic peptide and BNP for brain natriuretic peptide. The latter is so named because it was first discovered in brain cells, although later it was determined that the ventricles were the primary producers of BNP (with perhaps a little coming from the atria).

It is well known that ANP is elevated during AF/LAF. However, between episodes this is much less clear. In fact it appears that BNP is elevated in those with LAF and ANP is not.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=15629379&dopt=Abstract

Whether your aura was related to your elevated BNP or not is speculative. However, if the aura is related to diminished Mg⁺⁺, then it very well may have, because natriuretic peptides (ANP and BNP) both cause urinary Na⁺ and Mg⁺⁺ wasting with K⁺ being the beneficiary. Aldosterone, on the other hand, causes urinary K⁺ and Mg⁺⁺ wasting with Na⁺ being the beneficiary. Because these two hormones oppose each other wrt fluid balance (Na⁺ in the urine => net loss of water; Na⁺ reabsorbed from urine => net water gain), increased ANP/BNP => increased aldosterone. Mg⁺⁺ is lost in the urine under the action of both hormones and explains why so many LAFers appear to be urinary Mg⁺⁺ wasters.

I'm one of these. I took about 800-1000 mg aqueous Mg⁺⁺ (the most absorbable form) per day for over a year and my intracellular Mg⁺⁺ went from 34.3 to 34.6 (normal 33.9-41.9).

Of course, the aura could be related to the hypovolemia and beta receptor hypersensitivity (see above) caused by the increased BNP and consequent fluid loss it causes. The increase in ANP caused by the AF episode could have further accentuated the low blood volume, triggering the migraine and aura. Dehydration is a well known cause of migraine.

In any event, I would say that the timing of the AF episode/aura is not coincidence.

PC

Hi Barb,

Your doctors, like most, are afraid of K⁺ supplementation. After all, lethal doses are used for injection of those sentenced to death.

Unfortunately most MDs don't seem to realize that in the absence of renal disease (normal blood BUN/creatinine levels) one is perfectly capable of handling supplemental K⁺. If they don't balk at your eating a large banana, which contains about 40mg per inch, several times a day, why would they shrink from K⁺ supplementation. K⁺ is K⁺. It's combined with gluconate, an organic salt. So what's the problem.

As far as how much is OK, I can only quote Dr. Lam's book, *How to Stay Young and Live Longer* <www.lammd.com>, in which he states up to 15 gm is OK. But again that would be in divided doses and assuming normal renal function. I've never supplemented that much, but I and many others on this BB have done/do 3 - 4 gm in supplements/day.

The discussion has been intriguing. Unfortunately I'll be out of town for ten days starting midweek and won't be able to further respond. Hopefully others will jump right in.

PC

Aloha PC,

When you return, maybe you can comment.

My wife has low blood pressure (commonly 90/60) and after reading this: <http://www.afibbers.net/forum/read.php?f=6&i=17060&t=17060> I assumed low blood volume. I showed her the above and suggested that she increase her (celtic sea) salt intake. She has and it has helped her feelings of low energy.

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"I personally believe that increasing water intake and potassium supplementation is the best course to follow. Magnesium is also important, but is less important than K. I believe this is because K+ supplementation helps spare Mg++ from the ravages of aldosterone and because it also shifts the BP regulation equation."

"BP is determined by beta receptors and blood volume. That's why beta blockers and "water pills" are the cornerstones of anti-hypertensive therapy. Regulation of blood volume is a function of aldosterone and the latter is regulated by K/Na, RAAS (renin angiotensin aldosterone system) and ACTH. All else being equal, if we increase blood K+ => K/Na increases => aldosterone increases. But because blood volume hasn't increased, this means that the other two inputs, RAAS and ACTH, must pull back marginally => decrease in angiotensin (very powerful vasoconstrictor) => BP decreases. This is why K+ is such an effective anti-hypertensive."

=====
If one has low BP and low blood volume, why wouldn't you want to increase Na? It should promote water retention and a higher BP, if I'm not off track here?

Mahalo,
George

Hi George,

You're rarely off track. In fact I agree with you 100%. I think some of us should increase our Na+ intake, but there is already so much of it in our diet. At times I crave salt too. As I see it, the main problem is in those with increased natriuretic peptides. It's kind of a catch 22.

Increased salt and H2O intake => increased blood volume => decreased aldosterone and increased natriuretic peptide secretion (due to PFO, MVPS, LAF or whatever) => increased natriuresis and magniuresis, at least until equilibration between aldo and NP at that particular blood volume.

So, my advice is not to overlook your Mg++ supplementation, when you increase Na+ and H2O intake.

PC

Thanks PC, I have made a copy of the information you sent. I have an appt. tomorrow with my EP's Nurse Physician and I am going to ask her if I could get the test again and see if that is still elevated. That may tell us something. I know you are going out of town, but I will post anything she says and any results and you can address it when you get back. It would be interesting to know if there is a connection between the elevated blood results, the aura and the afib or at least if my EP thinks so.

Sharon

Thanks, PC - you make some good points about supplemental potassium. I will first get my blood tests completed, including the one for renal problems, and if all is OK, start looking getting more K.

Appreciate your advice and wisdom.

Barb

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