THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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Editorial

We have finally managed to get a large portion of the data obtained in the second lone atrial fibrillation survey (LAFS II) entered on a spreadsheet so that it can be evaluated. In this issue we present the first (general) results of the evaluation. With now over 200 individuals in the database we are seeing some definite and important trends, which will be discussed in future issues of The AFIB Report. I would like to engage a professional medical statistician to take a look at the data, but funding is a problem. Please let me know if you can think of an organization or individual who might want to support this work.

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As promised, we discuss atrial flutter in this issue and also feature an article by Sadja Greenwood, MD in which she describes her experience with lone atrial fibrillation. I know I always enjoy reading about the experiences of other afibbers and the coping mechanisms they have developed. Rounding out this issue there is some exciting AFIB News. Enjoy!

Yours in health and sinus rhythm,
Hans Larsen

Findings from LAFS II – Part 1

We received 83 new completed questionnaires in our second lone atrial fibrillation survey (LAFS II). This brings our total database to 203 afibbers, enough to enable us to draw valid conclusions.

The majority (43%) of the survey participants have the vagal variety of LAF, 29% the mixed form, and 12% the adrenergic. The remaining 16% have chronic LAF. The average (mean) age of respondents with paroxysmal LAF is 53 years (median 54 years) while that of chronic afibbers is 58 years (median 57 years). The average age at diagnosis for paroxysmal (intermittent) afibbers was 46 years (median 48 years) with a range of 14 to 74 years. The majority of respondents (51%) were between 40 and 55 years of age when first diagnosed. A significant 24% of respondents were in their 20s and 30s and only 5% were over 65 when diagnosed. This finding refutes the generally held belief that atrial fibrillation is an "old age" disease – it clearly is not.

The average age at diagnosis for chronic afibbers was 49 years (median 51 years) with a range of 8 to 72 years. The majority of respondents (52%) were between 40 and 55 years of age when first diagnosed. A significant 32% were over 55 when diagnosed and only 16% were below the age of 40 when first diagnosed. Chronic afibbers are thus somewhat older than paroxysmal afibbers, but even chronic LAF is by no means an "old age" disease.

The majority (81%) of respondents are male. Whether this reflects the distribution of LAF in the general population or is an indication of the relative use of the Internet among men and women is not clear.

Severity of Episodes

The number and duration of afib episodes and the total time spent in fibrillation over a 6- or 1-month period is our "gold standard" measure of the severity of paroxysmal LAF. It is an essential component in evaluating the effectiveness of drugs, supplements and other interventions. It is, unfortunately, very difficult to calculate a meaningful average of these values for a group of afibbers. The problem is that most respondents have fairly low values, but a small majority has greatly elevated values, which essentially makes a normal average (mean) quite meaningless in describing the overall severity for a particular group. For example, the calculated average time spent in afib per month for paroxysmal afibbers is 15 hours despite the fact the 81% of them spend less than 15 hours in afib. The average is skewed because a small group spends between 50 and 120 hours in fibrillation per month. We have, therefore, decided to use median rather than mean (average) values in describing group averages. The median is the value in the middle, i.e. the value above which half of all individual values can be found and below which the remaining 50% can be found. Using the median eliminates the bias introduced by a small group of "heavy hitters".

The median number of episodes over a 6-month period was 3 (mean: 13 episodes) for all paroxysmal afibbers. The median duration was 4 hours (mean: 10 hrs) and the total time spent in fibrillation over a 6-month period was 22 hours (mean: 90 hrs). The median time spent in fibrillation per month was 3.7 hours (range: 0-120 hrs). In comparison the time spent in fibrillation by chronic afibbers is 720 hours/month.

Other Findings

77% of paroxysmal afibbers have amalgam dental fillings, 52% have digestive problems, 51% take aspirin on a regular basis, 11% take warfarin and 38% take no anti-platelet or anti-coagulation medications. 78% of chronic afibbers have dental amalgams, 32% have digestive problems, 35% take aspirin regularly, 52% take warfarin and 13% take no anti-platelet or anti-coagulation medications.

Fifteen respondents have undergone ablation therapy; two have had the maze surgery. One hundred and twenty-three afibbers (61%) are taking drugs to prevent or ease episodes whilst 39% are not. A total of 81 afibbers (40%) are taking supplements, 13 have had their amalgam (silver) fillings removed, and 8 have taken other measures to prevent future episodes. In upcoming issues of The AFIB Report we will discuss the effectiveness of these interventions. In the remaining part of this issue we will briefly cover the "statistics" for the four different forms of LAF – vagal, adrenergic, mixed and chronic.

Vagal LAF

Eighty-eight respondents have the vagal form of LAF. Their median age is 53 years, age at diagnosis is 47 years, and 90% of them are men. The median number of episodes over a 6-month period was 3 with a range of 0 to 78. Median duration was 5 hours with a range of 0 to 65 hours. The median number of hours spent in fibrillation per month was 4 with a range of 0 to 120. 56% have digestive problems, 57% take a daily aspirin, 11% are on warfarin (Coumadin) and 32% take no anti-coagulation medication.

Adrenergic LAF

Twenty-four respondents have the adrenergic form of LAF. Their median age is 52 years, age at diagnosis is 47 years, and 79% of them are men. The median number of episodes over a 6-month period was 2 with a range of 0 to 20. Median duration was 7 hours with a range of 0 to 72 hours. The median number of hours spent in fibrillation per month was 4 with a range of 0 to 45. 50% have digestive problems, 35% take a daily aspirin, 10% are on warfarin and a remaining 55% take no anti-coagulation medication on a regular basis.

Mixed LAF

Fifty-nine respondents have the mixed form of LAF. Their median age is 58 years, age at diagnosis is 50 years, and 68% of them are men. The median number of episodes over a 6-month period was 4 with a range of 0-90. Median duration was 2 hours with a range of 0 to 36 hours. The median number of hours spent in fibrillation per month was 4 with a range of 0 to 120. 46% have digestive problems, 48% take a

daily aspirin, 12% are on warfarin and the remaining 40% take no anti-coagulation medication on a regular basis.

Chronic LAF
Thirty-two respondents have chronic LAF. Their median age is 57 years, age at diagnosis is 51 years, and 81% of them are men. 32% have digestive problems, 35% take aspirin daily, 52% are on warfarin and the remaining 13% take no regular anti-coagulation medication.

Conclusion
There is a slight trend for mixed and chronic afibbers to be older. The most common form of LAF for women is the mixed variety (32%) with vagal being the least common (10%). There is not a great deal of difference in episode severity between vagal, adrenergic and mixed LAF. Digestive problems are common affecting from 56% of afibbers with the vagal variety to 32% among chronic afibbers. A daily aspirin is the most popular stroke prevention measure taken by paroxysmal afibbers while warfarin is the choice among chronic afibbers. Over 50% of adrenergic afibbers and 40% of mixed do not take anti-coagulation medication on a regular basis.

Although the overall difference in episode severity between the various forms of LAF would appear to be small it is clear that choice of preventive drug (antiarrhythmic) can have a profound effect on episode severity – more on this in the next issue of The AFIB Report.

Atrial Flutter: Mechanism and Treatment

Atrial flutter and atrial fibrillation are similar in that they both involve abnormal, sustained, rapid contractions of the heart's upper chambers (atria). In atrial flutter the atria contract 220 to 350 times a minute in an orderly rhythm. In atrial fibrillation the rate of contraction may be as high as 500 beats/minute and the rhythm is totally chaotic. Atrial flutter and atrial fibrillation can be either intermittent (paroxysmal) or permanent (chronic), but chronic atrial flutter is relatively rare. Atrial fibrillation is about 8 times more common than atrial flutter[1]. Atrial flutter is often connected with diseases affecting the right atrium while atrial fibrillation is mostly seen in diseases affecting the left atrium[2]. The two arrhythmias can both occur as a result of an enlarged atrium or in the aftermath of open-heart surgery, but the mechanism underlying them is quite different. Nevertheless, they can coexist in the same patient and one may convert to the other[1,3].

Types and Mechanism
There are two major types of atrial flutter – common or type 1 and atypical or type 2 flutter. Type 1 flutter is by far the most common (65 to 75% of all cases) and is characterized by a specific conduction abnormality in the lower right atrium. Type 2 or atypical flutter, on the other hand, has no easily discernible origin and is therefore harder to deal with[1].

The phenomenon underlying atrial flutter is called circus movement. Normally the heart's electrical impulses proceed in an orderly manner from the SA (sino-atrial) node to the AV (atrio-ventricular) node thus giving rise to a steady heart beat of between 60 and 100 beats per minute. In common atrial flutter a "rogue" electrical circuit is established in the lower right atrium in an area bordered by the inferior vena cava, the tricuspid valve, the coronary sinus, and the eustachian ridge[1]. Within this area the heart's electrical impulses chase and catch themselves, hence the name circus movement. Circus movement can occur because the atrium is dilated or scared or because the rest (refractory) period of the individual heart cells has been shortened, for example, by the ingestion of certain stimulants. In common (type 1) atrial flutter the circus movement is always counter clockwise while it is typically clockwise in type 2 flutter[1].

The rapid beating of the atria is transferred to the ventricles through the AV node although rarely, except sometimes in children, in a 1:1 ratio. Most commonly the AV node blocks half or even 75% of the

aberrant impulses so the transfer ratio is 2:1or 4:1 meaning than an atrium fluttering at 300 beats per minute will cause ventricular contractions at a rate of 150 beats/minute or 75 beats/minute. The ventricular contraction rate is what is felt as the pulse rate. The pulse rate during atrial flutter is not necessarily steady, but can vary with position; for example, it may be 150 bpm when sitting and 75 bpm when lying down. Having a meal or experiencing excitement can also change the transfer ratio abruptly[2].

Treatment

Intravenous verapamil or diltiazem (Cardizem) can be used to control the heart rate during an atrial flutter episode, but do little to speed up conversion to normal sinus rhythm[4]. Electrical cardioversion, on the other hand, is usually quite successful (95% success rate) at achieving conversion at relatively low energy outputs (10-50 joules)[2,4]. Class IC antiarrhythmic drugs such as flecainide and propafenone have been used in attempts to restore sinus rhythm, but they are usually not successful. The problem being that as they lower the heart rate the AV node often switches to a 1:1 conduction ratio. This means that a patient who was doing tolerably well with an atrial contraction rate of 300 and a 2:1 or 4:1 conduction ratio (giving a pulse rate of 150 or 75 bpm) may all of a sudden find himself with a pulse rate of 220-240 bpm[4].

Two new class IIIC drugs, dofetilide (Tikosyn) and ibutilide (Corvert) have been found useful in converting atrial flutter to sinus rhythm when given as an intravenous infusion (success rate of 60 to 80%)[5]. Both drugs, unfortunately, can have very serious adverse effects including potentially fatal heart arrhythmias.

Antiarrhythmic drugs, with the possible exception of dofetilide, are not very effective in preventing future episodes and the class IC drugs (flecainide and propafenone) have the potential for making atrial flutter episodes considerably worse when they do occur[4,5].

However, all is not lost. Because the location of the origin of atrial flutter, at least in the common type, is so well known and consistent from patient to patient radio frequency catheter ablation can be used with considerable success to permanently eradicate atrial flutter[1,6,7]. Unfortunately, this procedure does nothing to cure atrial fibrillation, which may often coexist with atrial flutter. There is also some evidence that atrial flutter patients who have a successful ablation increase their risk of later developing atrial fibrillation by 10 to 22%[8]. So undergoing radio catheter ablation for atrial flutter may not remove the necessity of dealing with atrial fibrillation.

Stroke Risk

Whether or not intermittent or chronic atrial flutter increases the risk of a stroke is controversial[9]. One study found no increase in stroke risk among over 17,000 atrial flutter patients unless they also suffered from heart disease, hypertension or diabetes or had concomitant atrial fibrillation[8]. Another study found that blood clots in the left atrium (the dangerous ones) were uncommon in patients with atrial flutter and no other risk factors[10]. Based on this study the researchers conclude that long-term anticoagulation is not required for patients with “pure” atrial flutter whether common or atypical[10]. The risk of clot formation after cardioversion is very low[11]. Whether to use anticoagulation just prior to cardioversion is nevertheless controversial. Some researchers recommend it, others say it is not necessary[9,10,12].

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AFIB News

AF increasingly common in the United States. Hospital admissions for cardiac arrhythmias grew by 9% between 1985 and 1999. The growth was almost entirely due to a 70% increase in admissions for atrial fibrillation, which now accounts for 45% of all hospital admissions for arrhythmias. The average stay for atrial fibrillation was 3.5 days leading hospital authorities to predict that atrial fibrillation will have a significant impact on future cardiac health care costs.
Paper presented at the 51st Annual Scientific Session of the American College of Cardiology, March 18, 2002 (abstract)

Vigorous exercise and AF. Medical researchers at the University of Barcelona have confirmed that men who engage in vigorous physical exercise for many years have an increased risk of developing lone (vagal) atrial fibrillation. A review of the records of 1160 patients seen at an outpatient arrhythmia clinic revealed that the incidence of lone AF among long-term exercisers was 60% as compared to only 15% in the general population of Catalonia.
European Heart Journal, Vol. 23, March 15, 2002, pp: 477-82

Different mechanisms for adrenergic and vagal LAF. Several studies have reached the conclusion that adrenergic-type LAF mostly occurs in the presence of some sort of heart abnormality while vagal LAF is usually found in patients with structurally normal hearts. Heart rate variability studies have found that the beginning of an adrenergic episode is preceded by an increase in adrenergic tone. Swiss researchers now report that patients with vagal AF originating from foci in the pulmonary veins also experience an increase in adrenergic tone about 15 minutes prior to an episode. However, with time, the adrenergic response diminishes, and just before fibrillation begins, the vagal system is predominant. It is not exactly clear what this implies, but the finding may lead to more appropriate treatment of LAF.
Journal of Cardiovascular Electrophysiology, Vol. 12, March 2001, pp. 285-91

Vitamin C prevents arrhythmias. Researchers at the Cleveland Clinic report that vitamin C (ascorbic acid) is highly effective in preventing atrial fibrillation occurring after bypass surgery. Their clinical trial involved 50 bypass patients who were given 2 grams of ascorbic acid (extended release) the night before surgery, followed by 500 mg doses twice daily for five days after surgery. The incidence of postoperative atrial fibrillation in the vitamin C group was 16.3% as compared to 34.9% in a comparable group not given vitamin C. The researchers believe that atrial fibrillation episodes are sustained because of oxidative stress and increased peroxynitrite formation caused by the rapid heartbeat. Vitamin C is highly effective in neutralizing peroxynitrite radicals. They also suggest that oxidative stress is a primary mechanism by which calcium overload causes the aberrant electrical activity which leads to AF.
It is interesting to speculate that supplementation with relatively large amounts of peroxynitrite neutralizing antioxidants (vitamin C, gamma-tocopherol and lycopene) may act to prevent LAF episodes and, even more intriguing to speculate, that ingesting vitamin C powder or even orange juice at the time of an episode may shorten it. I hasten to add that I have no scientific evidence to support this idea.
Circulation Research, Vol. 89, September 14, 2001, pp. e32-e38

My Experience with Atrial Fibrillation

by Sadja Greenwood (sadjagreenwood@yahoo.com)

I was a vigorous person with good exercise tolerance until my 68th birthday, in 1998, when I went on a hike with friends on a very hot day. I didn't have enough water, and became a little dehydrated. On the way home I had difficulty walking, and felt very short of breath. For some reason, which I cannot fathom, I didn't take my pulse. Friends helped me get home, and I recovered quickly. Perhaps that was my first episode of afib, or perhaps that damaged my heart in some way. I had a treadmill stress test the next week, which showed no sign of heart attack. So, I didn't think much of this episode

I began to have short attacks of afib, diagnosed by EKG one time when I had to call the paramedics; these came on after strenuous exercise, eating too much at one time, drinking ice water or sharply carbonated beverages, or being strongly emotionally upset. This leads me to believe that my LAF is mixed, but predominantly adrenergic. The attacks initially lasted 1-2 hours, and now last 3-4 hours. When they occur I have learned to do the following things, which seem to help me. I carry a vial in my pocket at all times with aspirin, 10 mg propranolol (Inderal), 2 magnesium pills (protein chelate or citrate) each 200 mg, and half of a .125 mg triazolam (Halcion) tablet. I chew all these pills for quicker absorption, and lie down. The aspirin is to prevent clotting, the propranolol slows the heart beat, irregular though it is, and the triazolam is a short acting sleeping pill similar to valium, which helps me to relax. I do deep, slow breathing and wait for the episode to go away. Since the episodes usually occur at home, at night or in the morning, I also take a few fish oil capsules for good measure. I continue aspirin for 5 days or so after an attack. I don't take aspirin daily because I do take fish oil capsules and vitamin E, which are both anticoagulants. When I take aspirin I get spontaneous nose bleeds and bruises under my skin when I play the drums. I don't want to have a GI bleed or a brain hemorrhage. It's a balancing act. Of course I am frightened of having a clot and a stroke when I have an episode, and I don't advise my course of action to anyone else!

I went to several cardiologists, and had echocardiograms and a thallium stress test. No underlying heart disease was found. I decided not to take the medications that were suggested to me, such as digitalis(!), sotalol, long acting beta-blockers, and coumadin. Even the lowest dose of any medication seems to affect me adversely, and when I studied the side effects of these meds I decided against them. Fortunately I found Hans Larsen on the Internet, which has been a tremendous blessing.

I no longer do strenuous exercise, such as jogging or going to the gym, and I always warm up very carefully now. I walk, lift light weights at home, and dance. My exercise abilities have definitely decreased since the afib began, and I could say it is age, but my partner Alan is 75 and is amazingly fit, as are many of my friends, so I think the afib has affected my aerobic capacity. I am working on increasing it very slowly, avoiding exhaustion. Walking in nature and dancing are really important to me, and since I can do these even in a limited way, I am happy.

I had very large varicose veins in my left leg due to a foot infection in my youth, and I had these removed in 2000. The surgeon estimated that I had about 400 ml of blood in my leg at all times, which was causing a strain on the heart. I had no afib for 6 months after the operation, but then it started again.

In 2001 I found a new cardiologist, who told me that several of his patients had improved after stopping the supplement glucosamine sulfate (GLS). This was an important revelation to me. I realized that I had started taking GLS for joint problems 4-5 months before my first episode of afib. I was also having two gastrointestinal problems, which are considered adverse effects of glucosamine. One was "heartburn", otherwise known as acid reflux, and the other was abnormal hunger, due to reactive hypoglycemia. I felt as if I had to eat many times a day, every few hours, to prevent a rapid heartbeat and a feeling that afib was impending. Glucosamine is an amino sugar; a sugar to which nitrogen is attached and it causes insulin resistance in some type 2 diabetics. It was giving me rapid swings in blood sugar, as measured by blood tests, and corresponding swings in heart rate. I have been off of it for 8 months now, and am

gradually improving. No more acid reflux, and more normal hunger patterns. I still need to eat quite frequently, and prefer to have dinner at 4-5 pm, which is hard on my partner who likes it at 6-7. I am very careful to eat healthy food, avoiding simple carbohydrates, and emphasizing vegetables, fruits, whole grains, beans and small amounts of fish or poultry. I eat lots of walnuts or almonds between meals. No caffeine or alcohol – I never liked alcohol, but I really miss drinking tea.

Currently my afib episodes occur every 2-4 weeks, and last 3-4 hours. I have found that taking L-carnitine is very helpful, and I take 250 mg throughout the day, amounting to 2 grams daily. L-carnitine seems to stop the premature atrial contractions that can lead to afib. It makes me feel stronger. I have read a few booklets and articles on this amino acid, and feel quite certain that it has benefits for the heart and little downside.

I am a retired general practice doctor, and my new careers are working as a mediator in a small rural county, being a volunteer music therapist and playing various instruments in a local band. I figure that if I am not in pain, and my heart beat is regular, that I am very lucky. I conclude this lengthy treatise by saying how much I appreciate the work of Hans Larsen. I would not have had the courage to pursue my own course without all the information he brings to the problem.

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