

Amyloidosis News

CARING FOR PATIENTS AND THEIR FAMILIES LIVING WITH AMYLOIDOSIS | ISSUE 1 2008

A young mother's story

by Belinda Gibson

My mother died from a ruptured liver while on holidays in 1985. She was 47 years old. The diagnosis was the rare disease amyloidosis, something her doctors had never seen before. I remember my brother asking whether we could ever get it. We were told that there was a one in a million chance.

Fast forward fifteen years. I am 34 years old, married with three beautiful daughters aged nine, seven and three. I had been relatively healthy all my life. Looking back I did have symptoms of amyloidosis, suffering from dry eyes and stomach ulcers from the age of 16.

In June 2001 over the long weekend I woke feeling like I had been hit by a truck. A day or so later I had pain in my liver area going through to my back and down my right arm. The doctor I visited suggested I go home and have a massage. The next day the pain was no better so my husband took me to the emergency department in Coffs Harbour. In a matter of hours my liver ruptured. I was stabilized and flown to Sydney. I was rushed into theatre where doctors tried to stop the bleeding. My liver had just fallen apart. My surgeon described it like cottage cheese. If I were to survive, I would need a transplant.

Doctors had no idea what was wrong with me until my brother told them of my mother's fate. Following tests I was diagnosed with unclassified familial amyloidosis. I was kept alive by machines for almost a week, and was close to death when incredibly a liver became available on June 21 2001. My spleen and gall bladder were also removed. My doctors called it a gift from God.

It was a very long road to recovery. I was in hospital for three and a half months. During the entire time I was fed through a tube and I could not speak. My voice is now a lot deeper and huskier. The doctors don't know whether this is caused from amyloid involvement or prolonged intubation. Every time I started to regain my strength, something else happened to land me back in intensive care. I only saw my children three times in those few months. I became very depressed and I was extremely lucky to have the constant support of my husband and my brother. At the end of September I was released back to Coffs Harbour hospital and after two days came home to my family and friends. This is when my recovery really began.

Twelve months after my transplant my specialist contacted the Medical Director of the National Amyloidosis Centre in London, Professor Philip Hawkins, who diagnosed lysozyme amyloidosis. Apparently there is no treatment for this type of amyloidosis. However it is said to progress very slowly. So I now just play the waiting game.

My kidneys are slowly getting worse. I now have less than 50 percent kidney function, which may be related to the immunosuppressants I take to stop any rejection from my liver.

It has been seven years now since my transplant and I am pleased to say that I am doing well. I appreciate every moment of my life. One day, in light of my diagnosis, our daughters will have decisions to make but at the moment I am thoroughly enjoying watching them grow up, and I am taking time to smell the roses on the way.





From the editor

Welcome to the winter edition of *Amyloidosis News*.

Amyloidosis is a difficult disease to diagnose and sadly many sufferers consult a number of specialists before receiving a diagnosis, by which time they are often very ill.

Raising awareness of this group of rare diseases is therefore vital and is one of the reasons for producing *Amyloidosis News*. I am indebted to all of the patients and families who help in this awareness raising exercise by distributing this publication to their local community, colleagues, family, health professionals and members of the medical profession.

This edition contains articles on the importance of diagnosing the correct type of amyloidosis and on hereditary amyloidosis. We also touch on how the carer fits in with the treatment team, illustrated by some personal stories.

We hope you find the contents of this edition of *Amyloidosis News* informative and interesting. Please let us have your comments and any suggestions for future editions.

Many thanks go to the Leukaemia Foundation staff for their continuing support in producing this publication, and to all those who have contributed articles and personal stories.

My thanks go to Dr Goodman for his article on Diagnosing and Subtyping Systemic Amyloidosis, to Dr Peter Mollee for checking the article on hereditary amyloidosis and to Belinda, Erina and Patrick for sharing their stories.

Pat Neely
Coordinator Amyloidosis Services
Leukaemia Foundation

Useful websites for further information

National Amyloidosis Centre London.
www.ucl.ac.uk/medicine/nac

Amyloidosis Australia
www.amyloidosisaustralia.org

Amyloidosis Foundation
www.amyloidosis.org

Hereditary Amyloidosis

Amyloidosis is the general name given to a wide group of disorders in which an abnormal substance called amyloid builds up in organs and tissues around the body. Amyloid is an unusually stable material, which has a unique chemical structure, formed when certain proteins fold in an abnormal manner. These deposits progressively accumulate and disrupt the normal function of the tissues, eventually leading to organ failure. The organs most commonly affected include the kidneys, heart, liver, nervous system and the gut. While amyloidosis is not a type of cancer, it is a very serious and life threatening disorder.

There are three main types of amyloidosis. In AL Amyloidosis (also known as primary systemic amyloidosis) amyloid deposits are derived from abnormal plasma cells in the bone marrow. AA Amyloidosis (also known as secondary amyloidosis) results from a chronic inflammatory disease (for example chronic arthritis) or infection, for example tuberculosis or osteomyelitis, in the body. ATTR Amyloidosis is an inherited disease (passed down from one generation to the next). In this case, the amyloid is derived from an abnormal transthyretin protein, which is made in the liver. A number of other types of amyloidosis also exist. Correctly diagnosing the type of amyloidosis is essential as treatment is different for the various types of amyloid.

Familial or hereditary types of amyloidosis

Hereditary amyloidosis is less common than the AL and AA types. Hereditary amyloidosis is caused by inheriting a mutant gene. This mutation or abnormal gene leads to the production of the amyloid forming protein.

This mutation can be passed from one generation to another. It is known as an autosomal dominant disease meaning that someone with the mutant gene may have inherited it from their father or mother and they in turn are capable of passing the gene to their children who each have a 50 percent chance of inheriting it.

If you have not inherited the gene yourself you cannot pass it to your children

Even if you have inherited one of these mutations you may not develop any clinical problems. If you do develop symptoms this will usually not be until middle age.

Types of hereditary amyloidosis

Each family will have its own pattern of organ involvement. However the various types of hereditary amyloid affect individuals differently so it is important that the type of amyloid you have is established to form an appropriate treatment plan.

Types of Familial systemic amyloidosis

Type	Distribution	Usual clinical features
Transthyretin	Most common, worldwide	neuropathy, heart failure, diarrhoea, kidney failure
Fibrinogen	United States, Europe	hypertension, kidney failure
Apolipoprotein A1	United States, Europe	kidney failure
Lysozyme	Europe, Canada	kidney failure, liver failure
Gelsolin	Finland	corneal changes (eye), occasionally heart and kidney disease
Cystatin C	Iceland	intra-cranial haemorrhage
Apolipoprotein AII	United States, Russia	kidney failure

Symptoms

Many of the symptoms of hereditary amyloidosis are similar to those experienced by patients with AL amyloidosis, so it is vital a correct diagnosis is made.

Diagnosis

A tissue biopsy is used to diagnose amyloidosis. From there the type of amyloidosis has to be established by further laboratory work.

Genetic testing DNA

The genes associated with all known forms of hereditary amyloidosis can be analysed by genetic testing. This involves isolating DNA from a simple blood sample. Healthy individuals who are at-risk of having inherited a potentially amyloid causing mutation, may choose to undergo such DNA tests. However this is not advised without talking it over with your physician. Genetic counselling may be recommended.

Treatment

As with AL and AA amyloidosis the main aims of treatments are twofold: to reduce the production of the abnormal amyloid-forming protein by treating any underlying disease, and to support and preserve normal organ function.

Transthyretin amyloidosis ATTR (also known as FAP)

ATTR is the most common type of hereditary amyloidosis. This type of amyloid may deposit in any organ but most commonly deposits in the nerves causing loss of sensation, numbness in the limbs, bowel disturbances, bladder and blood pressure function and sexual dysfunction. The heart, kidneys and other organs are sometimes involved. This disease is progressive.

What is Transthyretin?

Normally the Transthyretin protein in the blood helps to move thyroid hormone and vitamin A. Genetic information for TTR is encoded by a single gene on chromosome 18. More than 80 amyloid forming variants of TTR are known of which the most frequent is called TTR Met30.

Treatment for hereditary ATTR

Liver transplantation can be used to remove the source of

the amyloid forming TTR variant. This can be limited by the presence of amyloid in the heart especially in older patients. Liver transplant is only suitable for a minority of patients with ATTR.

The drugs Diflunisal and one produced by FoldRx in the USA are being trialled to see if they prevent the formation of amyloid.

Other forms of hereditary amyloidosis

Other types of hereditary systemic amyloidosis are even more rare. Nerve damage is not usually experienced. The liver and heart are sometimes affected but in general patients present with kidney disease and high blood pressure in middle age. Some of these forms are:

Fibrinogen A alpha chain amyloidosis

A number of mutations of fibrinogen A alpha chain gene are known to cause amyloid. Patients usually present with kidney disease at the age of 50-60.

As the abnormal fibrinogen is produced solely in the liver, a liver transplant can prevent further amyloid deposition. Kidney transplant can be used to replace the failed organ. Apolipoprotein A1

Several mutations in the gene for apolipoprotein A1 cause amyloidosis. Half of the abnormal protein is produced in the liver. The kidneys are the main organs affected but the heart, liver and other organs can also be affected. Transplant to replace any of these organs may improve the situation.

Lysozyme amyloidosis

This is one of the rarest types of hereditary systemic amyloidosis. There is no specific treatment for this type of hereditary amyloidosis except for liver and kidney transplant to replace the failing organs. Progression is extremely slow.

In conclusion

It is vital to ensure this group of diseases is diagnosed correctly. The progression of this group of diseases, which usually does not produce symptoms until middle age, is often very slow. Much research is being done around the world to develop drugs, which will hopefully inhibit the development of amyloid in the systemic amyloidoses.

Focus on the Carer



By Pat Neely

When a loved one is diagnosed with the rare disease amyloidosis, life will never be quite the same again.

Many of you caring for such a person will remember the shock on hearing the news, followed by the feelings of disbelief, sadness, fear and uncertainty.

Without any warning you adopted a new role of carer, a person who provides physical and emotional care for your loved one.

I remember myself feeling utterly lost and overwhelmingly sad. My identity as a wife, mother and social worker seemed to completely disappear as I struggled with the challenge of keeping hope alive for myself, my family and most of all for my husband.

Practical considerations have to be faced at once such as care for children, financial problems and undertaking jobs you have never done before. Many patients and carers will move temporarily away from friends and the familiarity of home for treatment in a major city.

A new vocabulary of medical terms has to be learnt and you have to share your loved one with people you probably have never met in your life, the medical team. At this time, when barely coping, many carers feel they need to be constantly there for their loved one.

Carers may struggle with where they fit in within the treatment team, with information overload or no information at all. Many doctors are excellent at including carers in all discussions while others prefer to talk to the patient alone presuming they will relay the news. Being at the hospital at dawn or late evening when many doctors in the private system seem to do their rounds, can be challenging.

Carers talk about their feelings of inadequacy, their exhaustion, frustration and anger, emotions they often hide for fear of upsetting the patient.

"It all seemed so impossible," Patrick said. "I had a new job. We had just moved up from New South Wales and had young children. Unimaginable pressure was upon us. All I could do was adopt a state of denial and place our lives in God's care. I know now that this denial prevented me from offering my wife the support she needed at that time."

Research shows that many carers experience enormous stress from this added physical and emotional responsibility leading to depression, anxiety or even physical ailments.

So what can carers do to make their role easier to bear?

Seek and accept help

Try not to be overwhelmed by taking on more responsibility than you can handle. Turn to family, friends or health care professionals for support, love and advice. Friends and family often benefit by feeling they are doing something useful. Understand that every one goes through a range of emotions:

Grief

Many carers mourn the loss of things they held most dear, such as life with their loved one before the diagnosis of amyloidosis.

Anger

Carers say it is common to be angry with themselves, family, the hospital staff or even the patient. Anger can come from feelings that may be hard to show, such as fear and panic. If handled in the right way it can be a motivating force to find out more or make changes, but if you are constantly angry with those around you, talk with a health professional or counsellor.

Depression and anxiety

Anxiety is common, leaving you exhausted and unable to relax. Depression is a persistent sadness usually lasting a few weeks. If either of these emotions are affecting your ability to function normally, don't try to tough it out on your own. Talk with your GP or a support worker at the Leukaemia Foundation, the hospital or some other professional organisation. Relaxation techniques may help.

Loneliness

Carers may feel very alone even when there are lots of people around. Talking with other carers over coffee or in an organised group may help.

Understand your loved one's medical needs

Remember that although most of the attention from the medical team will be focused on the patient, you are a

very important part of that care team, and need to ask questions.

You may have different questions from your loved one. It can be difficult to ask such questions in front of them. Patients and carers often try to protect each other by not discussing their feelings or asking questions in front of one another.

Try to discuss things together before the doctor arrives. If your loved one does not want more information, seek their permission to speak with the doctor on your own. Don't feel you have to know everything immediately. Listen to what is being said and try to ask your questions as you come to understand the illness better.

Be sure you understand what type of care the patient will need, what they should eat, who you can contact if you are concerned and when their next appointment is.

Look after yourself

Remember that you need to eat nutritious meals and not live on snacks. You cannot be all things all the time to your family and your loved one. Accept help and take a break when you can, especially when the patient is in hospital. Thinking positively can help with the challenges, but accept that you may have feelings of anger, fear and

sadness. If these become overwhelming, talk with your doctor or a health professional. Allow the patient to talk about their sadness if they wish to and share your feelings together.

How does the Leukaemia Foundation care for the carer?

Leukaemia Foundation support staff throughout Australia offer support, counselling and advice for carers. For information about these services, contact the office in your state.

A number of states also organise informal gatherings in cafes close to major hospitals where carers of patients with blood diseases can meet and talk. Contact your state office for more information

Caring for the carer is a four-week program offered by the Leukaemia Foundation of Queensland. It is specifically designed to provide meaningful information to carers as they travel on their roller coaster ride of survival with their patient. Areas covered in the program include the role of the carer, the emotional roller coaster ride, carer burnout and strategies for coping. **For more information phone 07 38403844**

Light the Night

The twilight sky will light up during the Leukaemia Foundation's new event, *Light the Night*, to be launched in Queensland, on 18 September and South Australia and Western Australia on 17 September.

Light the Night will give Australians the opportunity to pay tribute and bring hope to patients and families living with leukaemias, lymphomas, myeloma and other related blood cancers.

Participants will take an easy walk for several kilometres each walker carrying an illuminated balloon to create a sea of twinkling lights.

There will be a choice of three different coloured balloons with supporters carrying the Leukaemia Foundation's blue balloon, patients and survivors carrying a white balloon, and those remembering a loved one carrying a gold balloon.

Before the walk, people will share their inspirational stories, celebrating and commemorating loved ones.

According to Amyloidosis coordinator, Pat Neely, *Light the Night* will be an opportunity to gather Foundation supporters, patients, loved ones and anyone whose lives have been touched by leukaemias, lymphomas, myeloma and related blood disorders, in one place.

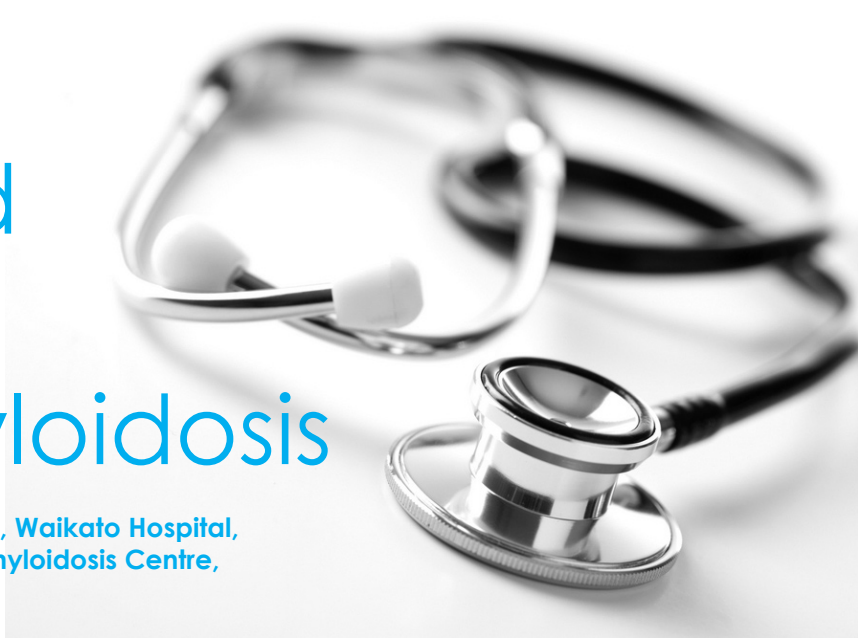
"The focus of the event will be on remembering those we have lost, paying tribute to those who have survived and bringing hope to those who are living with these diseases. It will also be an opportunity for the Foundation to highlight the wonderful care and support it gives to patients and families," Mrs Neely said.

For more information please call the Leukaemia Foundation office in your state.



Diagnosis and Subtyping of Systemic Amyloidosis

by Hugh Goodman, Department of Haematology, Waikato Hospital, Hamilton, New Zealand and formerly National Amyloidosis Centre, London, United Kingdom



Most readers will know that amyloidosis refers to a group of disorders caused by the abnormal folding, aggregation (clumping) and accumulation (buildup) of various proteins in tissues around the body. This can occur in a single spot (localised amyloidosis) but is far more commonly found as a widespread problem (systemic amyloidosis). What is less well understood by both medical staff and patients is that there are many different types of amyloidosis and that it is absolutely vital to know which type is present, as treatments differ hugely from one to another.

The first step in diagnosing amyloidosis is to think of it. As most patients can attest, the symptoms are often very vague and can affect one or many different organs around the body. As the condition is also relatively uncommon, this means that individual GPs or specialists will see amyloidosis very rarely and the vague symptoms naturally will be attributed initially to something far more common. For example, when the heart is affected by amyloid, it becomes stiff and pumps blood less efficiently; this causes breathlessness and fluid retention. These are the usual features of heart failure due to ischaemic heart disease (heart attacks, angina etc, that affect a large slice of the population), uncontrolled high blood pressure, excess alcohol or even infections – or perhaps the person is just getting old and unfit! It is for these reasons that studies have shown an average of 1.5 years from first symptoms to diagnosis of amyloidosis. One of my goals as a physician with an interest in amyloidosis is therefore to make sure my colleagues remember and know about the disease and its symptoms, and it is a goal I know is shared by patient support and advocacy groups.

In fact, the kidney is the most commonly affected, causing abnormal blood tests or frothy urine (due to loss of protein into the kidney); followed by the heart (symptoms above); then nerves (tingling / numbness in the feet), liver (enlargement) and bowel (disturbed function).

Amyloidosis is diagnosed by showing it in a biopsy under the microscope. Often the biopsy will be of an affected organ (e.g. a kidney biopsy done to investigate the cause of kidney impairment on a blood test) but can be from a safer site (e.g. the lip, gum, rectum or abdominal fat) if the diagnosis is already strongly suspected elsewhere. Once

the diagnosis is made, it is vital to check which other organs are affected – for example, if it has been diagnosed in the kidney, the heart is checked (blood tests and scan), as are the nerves, the liver (blood tests and ultrasound scan) etc. Knowing which organs are affected by amyloid helps sort out what type it is and, more importantly, which treatments will be right for you.

The next step is to find out what type of amyloid it is. Each type of amyloid is made of a different protein, although in each case it is folded into the same ‘amyloid’ shape. Each subtype of amyloid is given a code based on these proteins – the code is the letter ‘A’ followed by one or two letters to show the protein type (examples below). These proteins are all quite different to each other, but the common situations in which they form amyloid can be summarised as follows:

1. Completely normal proteins that we all have, but present at abnormally high levels. For example, the normal protein SAA (serum amyloid A) is raised whenever there is inflammation in the body (e.g. infection, burns, immune disorders). If inflammation and therefore high levels of SAA are present for a long time (years), as happens in severe rheumatoid arthritis, AA amyloidosis (A for amyloid, A for SAA protein) can develop.
2. Abnormal amyloid-forming proteins that you are not born with – an ‘acquired’ problem. The commonest form of systemic amyloidosis is AL (old name primary amyloidosis). The amyloid is formed from pieces of abnormal antibody (immunoglobulin) proteins. We all make antibodies but normal antibodies do not form amyloid. As we get older, it is not unusual to make small quantities of abnormal antibody proteins and very occasionally these have the capacity to form amyloid. The amyloid-forming piece of the antibody is known as a light chain – therefore the name AL (A for amyloid, L for light chain) amyloidosis.
3. Abnormal amyloid-forming proteins that you are born with – an ‘inherited’ problem. Rarely, a protein is made incorrectly because the genetic code has a mutation (glitch!) in it, a mutation that usually has been handed down from a parent. Although the vast majority of that protein still does its normal job, this glitch makes the protein more likely to form an amyloid shape. Mutations of a gene (piece of the genetic code) called

TTR (it makes transthyretin, a transport protein) cause ATTR amyloid, commonly affecting the nerves and heart. Mutations of fibrinogen (a clotting protein) cause AFib amyloid, affecting the kidneys. There are many others although they are rare.

Getting these subtypes right is vital for choosing the correct treatment. For example, chemotherapy is usually the right thing to do for AL but is clearly absolutely wrong for any other type. Liver transplantation is sometimes appropriate for inherited ATTR but absolutely wrong for AL or AA.

Unfortunately there is no single test to identify what type of amyloid is present. Many organs are affected by all or some types of amyloid and it is therefore very difficult to be sure of the type merely by knowing which organs are affected. The next step, therefore, is to gather information

about the possible 'causes' of amyloidosis (e.g. looking for inflammation, for abnormal antibodies, for abnormal genes) which can be done by talking to and examining the patient and by various blood and urine tests.

The last piece of information is to look at the amyloid-containing biopsy again, as some types of amyloid can be diagnosed or ruled out with special 'stains' under the microscope. In the end, however, there is often not one single test that absolutely confirms one type or another and the physician must put all the information together to come up with an answer. That physician must know about the different subtypes in order to consider them and rule one in and others out, and education about amyloidosis therefore remains of vital importance for physicians and patients alike.

Being a carer...

by Erina Beruldsen

A carer is an interesting term. I feel I cared no more or less throughout my husband Gordon's long illness than at other times during our life together. We still experienced irritations, disagreements and much happiness. We continued to pursue separate interests. But I certainly developed a heightened awareness of Gordon's needs and feelings.

A diagnosis of AL amyloidosis with a poor prognosis was a shock. Gordon went into a frenzy of activity, updating wills, obtaining power of attorney, making the funeral arrangements, behind my back. But where was I? In a land of unreality!

Depression followed treatment decisions. It was a difficult time for both of us. Gordon needed a goal. A suggested update of his book on birds completely changed his mood and "even if it is the last thing I do" became a favourite saying.

When told he shouldn't fly to Tasmania we did. I administered his daily intramuscular injection at exactly the same time every day, which made me feel useful. It was a journey to be remembered. I left his medication in the fridge and had to get a cab back from the airport to collect it, leaving Gordon to change the flight.

Gordon was far from well on this holiday and gave me lots of scares but we lived to tell the tale and incredibly there would be other trips to Cairns and New Zealand. Although I was often terrified travelling with a man who could only walk a few steps at a time without losing his breath, it had its funny side. As we weaved our way across busy streets hanging on to each other, people probably thought that we, tea-totallers, were drunk.

Our many hospital trips for chemo and doctors visits were filled with laughter and chatter with the staff. We always



looked forward to a cup of coffee afterwards where we shared so many thoughts.

I tried to keep life as normal as possible going out and ironing for three friends. Gordon and I shopped together with the same arguments over where to buy the meat or why I had to visit every supermarket aisle.

Four years after diagnosis Gordon's life ended swiftly. He admitted himself to hospital on finding his blood pressure no longer registered on his machine. He still didn't give up, telling a nurse with a cough to keep away in case her germs killed him. Ripping off his oxygen mask he demanded breakfast and then lay back, his energy spent. I felt so helpless. What could I do except hold his hand. After he squeezed mine a joyful expression came over his countenance and his fight was over. What a privilege it was to be with him after our journey together.

I was no longer a carer. I now had the house and bathroom to myself but there was a steep learning curve ahead of me. With continued support from our children, many friends, the Leukaemia Foundation and my church, life is now looking good and I can sit back and reflect. Life as a carer was never dull. Trying to be one step ahead of Gordon, anticipating his problems, strangely brought us closer together. Yes at times I did boil over with frustration, but wow, what a feeling when I realised I had kept my outward calm and not added to Gordon's woes.

Amyloidosis support luncheon dates

As part of its mission to care and support patients and their families, the Leukaemia Foundation offers a range of education sessions on topics relevant to patients, family members and carers. Contact your state office for more information.

The Leukaemia Foundation of Queensland organises Amyloidosis support luncheons with and without guest speakers.

The dates for the 2008 Amyloidosis luncheons are:

- June 10 Tuesday
- September 9 Tuesday
- December 2 Tuesday (Christmas luncheon)
(Speakers being finalised)

All luncheons held at 11.30am for 12 noon
ESA Village, 64 Raymond Tce, South Brisbane
RSVP Noeleen on (07) 3840 3844

A special amyloidosis meeting is being arranged for 7 & 8 November entitled, *Where have we come in 10 years?* Details will be circulated.



News from South Australia

The Amyloidosis group in South Australia continues to meet regularly and attracts patients and families from all over South Australia. On 22 February 2008, Professor Randall Faull, Senior Consultant in Nephrology at the Royal Adelaide Hospital, spoke on *Amyloid and the Kidneys*. Professor Faull gave a summary of the current knowledge and treatments for amyloidosis, before explaining in detail the impact that it can have on the kidneys.

Amyloidosis patients and their families are welcome to attend any of the education sessions organised by the Foundation. There will be a Patient Education Day on Saturday 31 May 2008 focusing primarily on the current research in haematology. The next session specifically for patients and families affected by amyloidosis will be a lunch on Friday 27 June. **Please feel free to ring Steve Marshall on 08 8273 3555 for more details.**

SUPPORT SERVICES TEAM

Queensland – Barbara Hartigan
Coordinator Amyloidosis Services – Pat Neely
National Support Services Manager – Anthony Steele
Victoria/Tasmania – Sam Schembri
New South Wales/ACT – Ann Schiller
South Australia/Northern Territory – Steve Marshall
Western Australia – Sandy McKiernan

FOR HELP CALL....
Brisbane: 07 3840 3844
All other states: 1800 620 420
www.leukaemia.org.au

Our vision to cure and mission to care.

The Leukaemia Foundation of Queensland is a not for profit organisation focused on the care and support of patients and their families living with leukaemias, lymphomas, myeloma and related blood disorders.

The Foundation does this by providing emotional support, accommodation, transportation and practical assistance for patients and their families. The Leukaemia Foundation also funds research into cures and better treatments for leukaemias, lymphomas, myeloma and related blood disorders.

The Leukaemia Foundation receives no direct ongoing government funding, and relies on the continuous support of individuals and corporate partners to expand its services.

To find out more about the work of the Leukaemia Foundation of Queensland and how you can help, phone 1800 620 420 or visit the Foundation's website: www.leukaemia.org.au

Disclaimer: No person should rely on the contents of this publication without first obtaining advice from their treating specialist.

If you do not wish to receive future editions of this publication please contact the Leukaemia Foundation Support Services Division on 07 3840 3844.



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