

Blood Matters

Newsletter for the Haematology Association of Ireland Nurses' Group

APRIL/2009

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Letter from the Editor...

We hope you enjoy the latest edition of Blood Matters, and find it both educational and interesting. We would be delighted to receive feedback from you on any aspect of your newsletter, and as always appeal for contributions so we can all share the learning, innovations and developments within haematology.

In our previous October 2008 edition (which can be accessed from the HAI nurses website) we commenced a two part feature on Aplastic Anaemia. The second article inside will concentrate on supportive and definitive therapies for this condition. We also promised to continue our clinical trials update and include a report on paediatric clinical trials within Northern Ireland in this issue, however due to unforeseen circumstances we'll have to keep you in suspense until next time! This edition also introduces the first of a two article series on the pertinent topic of cancer cachexia.

Additionally, find out more about services for patients with Myeloproliferative neoplasms, both in the UK and Ireland and we'll also introduce you to two further recently appointed Haematology Clinical Nurse Specialists in Northern Ireland. Our regular features of poetry corner and a prize winning crossword can be found inside. This time the crossword concentrates on bleeding and clotting - but don't worry, not in too much depth!

Finally, if you were unable to attend the 2008 annual conference, catch up with what happened in Armagh, including changes to your HAI nurses committee. We look forward to seeing you at the Spring study day on Friday 24th April in the Hilton, Kilmainham, Dublin. For further details or registration please contact Sinead Cassidy, or alternatively check out our website (details below).

Yours on behalf of the HAI nurses' group
Caroline McCaughey
Newsletter Editor

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Newsletter for
the Haematology
Association of
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HAI Annual Conference 2008



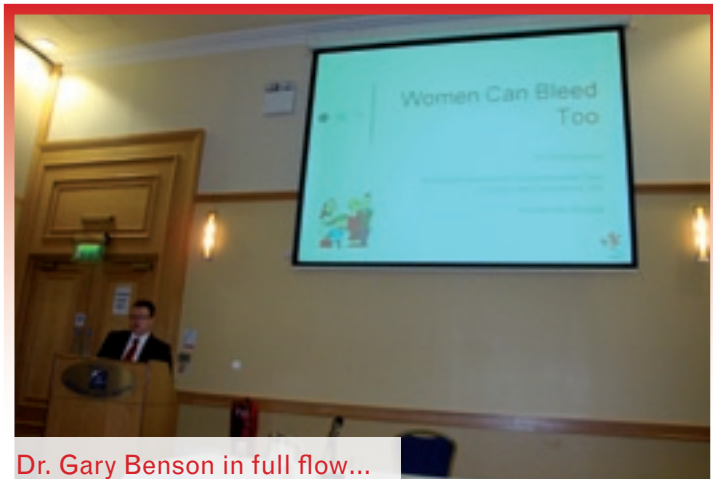
Mingling at registration



How clever am I?! Ger, after winning the quiz

The HAI nurses' group enjoyed both a very productive and social annual conference in the Armagh City Hotel on the 17th-18th October 2008. We were delighted to welcome Liz Henderson, Network Nurse Director, to open the conference by presenting the exciting developments in cancer nursing that the Northern Ireland Cancer Network have achieved to date, as well as future directions. Another highlight of the conference was Dr. Gary Benson, Consultant Haematologist, who managed to make the topic of female bleeding disorders not only educational but also memorably entertaining!

The afternoon sessions commenced with an excellent presentation by Dr. Scott McCloskey regarding haematological investigations; what could have been a tedious subject was clearly explained in an animated way, constructed around the theme of the "The Matrix"!



Dr. Gary Benson in full flow...



It's simple really...! Dr. Scott McCloskey unravels the mystery of PCR

Next Maxine Rudkin from Nottingham spoke regarding the pertinent complication of peripheral neuropathy; this generated lively debate from the floor including input from our occupational therapy and physiotherapy colleagues. Friday morning was concluded by our now traditional haematology nurses' quiz, which produced laughs and groans in equal measure! Congratulations to Ger Walpole, who defied the theory of "pregnancy brain" to score the top mark and win the €200 prize, kindly sponsored by Roche.

The diverse programme then turned to haematology clinical trials, with Tracey McGuigan, a research nurse from the Northern Ireland Cancer Clinical Trials Unit, outlining the ongoing studies specific to Myeloma. One of our external reviewers, Phil Larkin, subsequently led an insightful exploration into palliative care and haematology, prior to the five abstract presentations. Both the posters and these oral presentations were externally reviewed by Phil Larkin and Richard Henry, who represented the National University of Ireland, Galway and Queen's University, Belfast respectively. Congratulations to our prize winners, Finola Gill for her oral presentation on the experiences of general nurses caring for patients with cancer on non specialist wards, and Ruth Thompson, whose poster outlined the development of a nurse - led Myeloproliferative service. Thanks to Schering Plough who sponsored the €2300 travel bursary for best oral presentation and to Baxter for supporting the €1000 best poster bursary.

Friday ended on a high note with a banquet dinner and as you can see the Ulster Hospital contingent once again provided much of the entertainment!!



HAI wouldn't be HAI without Judy McCartney. She hasn't missed a conference since the nurses' group commenced.



Phew! That went well. Mary stepping down from the podium, congratulated by Dr Crotty

Theresa Meenaghan, despite the tough slot of first thing on Saturday morning, caught everyone's interest with her topical presentation on treating the elderly patient with AML. The next topic moved to the opposite end of the age spectrum as Dr Kevin Ryan portrayed the management of the child with ALL. The subsequent talk was also one not to miss - genetics in haematology made simple (well, as simple as it can be!) by Prof Mary Frances McMullin. This was both fascinating and well evaluated.

This year for the first time a nurse (our very own Mary Kelly, Chair of the Nurses' Group) was invited to present to our medical and scientific colleagues. Mary capably presented the findings of her MSc phenomenological study into the lived experience of Myeloma, a talk which incited much interest and discussion within the audience. Mary Cahill, President of HAI reciprocated by discussing the highlights of the medical programme with the nurses. As always this was a valuable and interesting session.

Mr Norman Campbell, previously a dentist within the Belfast City Hospital, made it worthwhile for those of us who stayed to the bitter end. Unfortunately he arrived earlier than expected, just as we dished out a bucket of sweets to munch on during our DVD viewing - to speak on dental health for haematology patients! By the time he had finished his highly informative talk we were all guilt ridden and determined to be much kinder to our teeth!

HAI 2008 was rounded off by Saturday night's dinner and prize presentations....and a bit of dodgy dancing by some!! After such a successful conference we're already looking forward to HAI 2009 in the Lyrath Estate, Kilkenny 16th-18th October - a venue not to be missed I hear!



Check out the killer heels!

Sickle Cell Anaemia

By Teresa Hayes, Clinical Nurse Manager -
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Sickle Cell Disease (SCD) occurs when a person inherits two genes for sickle haemoglobin (Hb S), one from each parent. It is caused by the substitution of the amino acid valine for the amino acid glutamic acid at a particular point in the chain (Bennett, 2005). The red cell becomes depleted in oxygen and causes 'sickling' of the red blood cells at particular times. This in turn causes them to become trapped in the slow flowing circulation. There is adhesion of the red blood cells to the endothelium in the veins causing inflammation and obstruction to the normal blood flow (vaso-occlusion). This in turn causes pain of varying intensity because of lack of oxygen and swelling. Once the red cells become oxygenated they unsickle again, unless permanently damaged; normal blood flow returns and the pain becomes less severe or ceases.

Factors triggering sickling of red cells

- Reduced level of oxygen
- Dehydration
- Infection
- Sudden change in temperatures
- Acidosis
- Smoking and alcohol

Deoxygenation of the sickle cell can also occur spontaneously with no precipitation factor. The spleen and the liver are more prone to the effects of sickling, as blood flow is slow in these organs.

Sequestration Crisis

This crisis occurs when there is a sudden life threatening drop in haemoglobin concentration, due to splenic sequestration (pooling of large volume of blood), or less commonly in the liver.

There is rapid painful swelling of the organ followed by worsening anaemia. Splenic sequestration usually affects children under the age of 5 and can result in circulatory collapse, anaemia and hypovolaemic shock followed by death. All new mothers should be taught to palpate their child's spleen and to recognise events which can trigger a crisis. Older adults with chronic hypersplenism should be considered for a splenectomy. Sequestration of the lungs is a form of acute chest syndrome.

Haemolytic crisis

Normal red cells survive approx 120 days but in the sickle cell, the red cell only survives about 19 days. This rapid breakdown causes an increase in the haemolytic rate resulting in rapid haemolysis, which leads to anaemia (Stuart & Nagel, 2004). During infection, the breakdown is rapid leading to exacerbation of anaemia.

Aplastic anaemia

Aplastic anaemia can occur at any age as a result of

reduction in or cessation of bone marrow activity. While it usually lasts only a few days it can cause a significant and dramatic fall in haemoglobin and be life threatening. The role of the human parvo virus B19 in sickle cell is thought to be a causative factor, however, it does not affect normal marrow.

Painful vaso-occlusive crises

Pain can occur anywhere in the body but the limbs, back and abdomen are the more common sites. Pain crises can vary in frequency, intensity and duration.

Nursing management

Initial management should be aimed at rapid assessment of pain, commencement of analgesia within 30 minutes with pain control achieved within 60 minutes (BCSH, 2003). Where possible, patients with known sickle cell disease should have rapid access to treatment. Patients should be encouraged to keep diaries of pain episodes and management so when presenting, the optimal regime can be commenced for the patient. Each patient is individual and the responses vary between people. Therefore personalised care plans should be available to on call staff, and ideally the choice of drug should be influenced by the individual's analgesic history. Assessment should distinguish between acute and chronic pain and once pain is controlled, the underlying cause should be assessed carefully.

First line analgesia

Paracetamol and non steroidal anti inflammatory drugs (NSAIDs) are appropriate for mild to moderate pain, unless there are contraindications in the patient's medical history. Dihydrocodeine is also suitable for children.

If pain is severe and non opioids not effective, opioids should be commenced. Opioids can be given orally, intravenously, intramuscularly or subcutaneously. Oral analgesia can be used to treat moderate pain after initial treatment with a single injection. Increasingly, children are managed solely with oral analgesia and as children mature, oral analgesia is the first choice in treatment of pain crisis.

Adjuvant medication

Sedatives and anxiolytics are required if the patient is agitated or frightened, but should not be used as a substitute for analgesia. It is important that laxatives be routinely prescribed if opioids are used and anti-emetics and anti-puritics may also be required, SCD is considered to be a prothrombotic state and painful crisis can result in prolonged periods of relative immobility thus anticoagulant therapy should be considered.

Fluid Replacement

Patients fluid balanced should be closely monitored and they must be encouraged to maintain an oral intake of 60ml/kg/24hours in adults or 100ml/kg/24hours in children. Intravenous supplementation of fluids can be initiated until the patient is drinking adequately and is painfree.

Patients should be nursed by staff who are skilled in the care of SCD and who can recognise and initiate treatment

promptly. Nurses also have a significant education role with the patient and their family, and can liaise with the community and hospital multidisciplinary team as necessary.

Optimal environmental conditions are important in minimising pain crises, for example, patients need to have consistent heat. Facilities such as central heating, access to a telephone and adequate nutrition are added requirements. These can incur expense and it is important

that the family have access to optimal community services to help support them in their needs.

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Your HAI Nurses' Committee



From left: Lorna Storey, Karena Maher, Liz O'Connell, Rosena Geoghegan, Susan Piggott, Kathleen Beston, Caroline McCaughey, Theresa Meenaghan, Ger Walpole and Mary Kelly. (Joy Lewis not present for photo)

We would like to thank Theresa Meenaghan and Emma Lane for their contribution to HAI as they step down from the committee. Following their nominations at the AGM, we are delighted to welcome Joy Lewis, Karena Maher and Kathleen Beston who are joining our ranks!



Sinead, HAI administrator, along with Elizabeth, relaxing after all the hard work!

Cancer Cachexia (Part 1)

By Dr. Joanne Reid, Lecturer in Cancer Nursing, Nursing and Midwifery Research Unit, Queen's University, Belfast

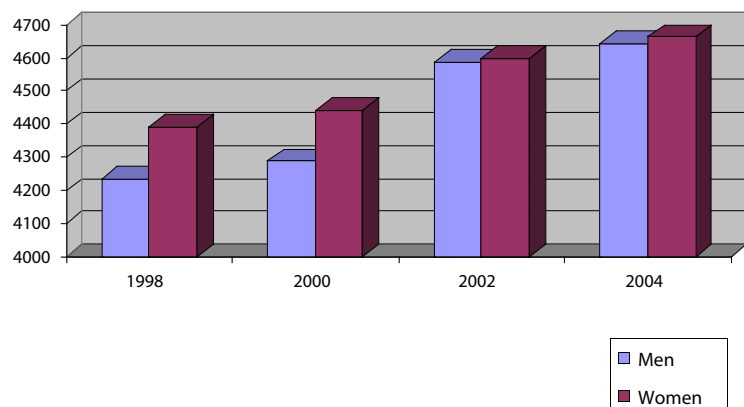
Introduction to cachexia.

Cachexia, although a frequent and devastating complication of advanced malignancy (DeWys et al, 1980), is not exclusive to cancer and has been reported to occur in many conditions. These include tuberculosis (Chavalittamrong et al, 1987), chronic obstructive pulmonary diseases (Schols, 2003), advanced organ failure (MacDonald et al, 2003), AIDS (Grinspoon and Mulligan, 2003), sepsis (Hasselgren et al, 2002), Chron's disease (Martignoni et al, 2003) and rheumatoid arthritis (Walsmith and Rubenhoff, 2002). Cachexia is a debilitating condition, which occurs in up to 80% of patients with advanced malignancy (Gordon et al, 2005). The incidence of cachexia is tumour dependent and ultimately this difference is thought to lie with the variations of tumour phenotype, which lead to the development of cachexia (Monitto et al, 2001). Patients presenting with the same tumour type and stage can experience differences in the degree of cachexia experienced. Similarly, tumour size bears little correlation to the degree of cachexia experienced and it can occur in patients when the total tumour mass is less than 0.01% of the host weight (Morrison, 1976). However, the degree of cachexia experienced correlates strongly with poor prognosis (Nixon et al, 1980); has a negative effect on quality of life and functional state (Barber, 2002); and is inversely proportional to the survival time of the affected patient (Barber et al, 2000).

Worldwide, it is estimated that approximately two million people die annually as a consequence of cancer cachexia (Muscaritoli et al, 2006). Locally, the occurrence of cancer in Northern Ireland continues to rise annually, as displayed in Figure 1 below.

Figure 1: Number of diagnoses of all cancers in Northern Ireland

(Northern Ireland Cancer Registry [NICR], 2006)



The frequency and mortality of cancer are reported as ever increasing (Cancer Facts And Figures - CFF, 1993). Indeed the latest figures show 9306 incidences of cancer in 2004 (NICR, 2006). The

Northern Ireland Cancer Registry recorded 3656 deaths from cancer in 2004 showing cancer to have taken over from heart disease [3237 deaths in 2004] as the main killer in Northern Ireland (Northern Ireland Statistics and Research Agency [NISRA], 2004). This was again re-emphasised in the Registrar General's Quarterly Report for Northern Ireland (Quarter 2, Department of Finance and Personnel Northern Ireland, 2005). In this report, deaths from cancer accounted for 25.6% of all deaths, compared to heart disease which accounted for 18.9% of all deaths, for this quarter. The latest figures available indicate 3656 deaths from cancer in Northern Ireland in 2004. Extrapolating the 80% incidence of cancer cachexia in advanced malignancy to this figure, would indicate that 2925 patients with advanced cancer were affected by cachexia in 2004 in Northern Ireland. Thus it is evident that cancer cachexia represents a considerable clinical problem regionally, nationally and internationally.

Aetiology and pharmacological approach to cancer cachexia

Cancer cachexia is typically characterised by non-intentional weight loss, muscle wasting, anorexia, early satiety, asthenia, anaemia and oedema (Dahele and Fearon, 2004). Weight loss is the symptom most commonly associated with cancer cachexia (Stewart et al, 2006). Importantly, not all weight loss experienced in people with cancer is the result of cachexia. However, the hallmark of weight loss associated with cancer cachexia is that it cannot be reversed with feeding (Barber, 2002). This difference that is thought to be due to metabolic alterations that are inherent in cachexia (Martignoni et al, 2003).

Research focusing on gaining insight and understanding into the metabolic anomalies associated with cancer cachexia has proliferated in the last number of years. In parallel to this, clinical research efforts have focused on the pharmacological management of cachexia aimed at reversing or ameliorating the associated weight loss (Barber et al, 1999; Gordon et al, 2005; Lopez et al, 2004; Fearon et al, 2003). However, preventative and therapeutic treatment options have been shown to be largely ineffective and currently there is no notable pharmacological agent that successfully halts and reverses the weight loss associated with cancer cachexia (Mattox, 2005). Furthermore, the optimal treatment for cancer cachexia in patients with advanced cancer has yet to be determined (Harle et al, 2005). In practice today, despite much time and investment into clinical research, standardised guidelines for the prevention, classification and treatment of cachexia have still not been developed (Muscaritoli et al, 2006). Ultimately this is because the nuances of the multi-factoral pathogenesis of cancer cachexia are still not understood. This only serves to compound the relevance of cachexia in negatively affecting the patients' surgical risk, response to chemo/radiotherapy and overall quality of life (Inui, 2002 and MacDonald et al, 2003). Hence, it remains a major cause of morbidity and mortality in advanced cancer patients today.

The impact of cancer cachexia

Cancer cachexia, along with pain, has been identified as one of the two most prevalent and devastating problems affecting individuals with advanced cancer. The wasted appearance of a cancer patient with cachexia is recognised as the most obvious sign of advanced illness and it is regarded as a colossal concern for not only patients but also their family members (McClement and Woodgate, 1997; MacDonald et al, 1995). The loss of skeletal muscle mass in cachexia leads to extreme physical debility (Cunliffe, 2006). It also has a psychological impact and has been shown to negatively affect the patients' body image (Bruera & Fainsinger, 1993). Additionally, it has been suggested that the negative psychological impact, due to alterations in body image, can reduce the ability of patients to socialize with friends and family at meal times (Higginson and Winget, 1996). While cancer cachexia has been shown to have a multidimensional impact on patients it is also suggested that it has distressing ramifications for the patients' family (Stroud, 2005). Indeed previous literature has highlighted both the emotional impact and existential concerns that cachexia causes for family members (Higginson and Winget, 1996; Thomas, 2006).

It is clear that the multifaceted dynamics of cachexia can impact on patients beyond that of a purely physical nature and can also affect the patients' family who care for them. Additionally, while the need for holistic interventions to treat cachexia has been stressed (Lesko, 1989), current management strategies focus primarily on pharmacological therapy aimed at reversing or ameliorating the associated weight loss. In this context it is important to recognise that even if a pharmacological agent were produced, which minimised the weight loss associated with cancer cachexia, it is unlikely that it would also solve the inherent psychosocial issues (Poole and Froggatt, 2002). It is vital that health care professionals understand the total illness experience of cancer cachexia in order to provide care that is responsive to patients' needs. Correspondingly, it has been suggested that research examining the holistic ramifications of cancer cachexia needs to be undertaken (Bruera & Fainsinger, 1993), in order to understand the totality of this illness experience and advance effective symptom management for this life limiting syndrome of advanced cancer.

The need for greater research activity into cachexia.

Despite its prevalence and prognostic impact (Andreyev et al, 1998; DeWys et al, 1980; Okusaka et al, 1998), there is evidence suggesting that cancer cachexia remains little understood and poorly managed in clinical practice (Delmore, 2000). There is also a contention that research into this phenomenon has been scant and as such it is recognised as an under researched area of advanced malignancy (Steer, 2005). It is argued that at a clinical level cancer cachexia is usually not treated until the affected individual suffers considerable weight loss and wasting (Argiles et al, 2005). Indeed, Brown and Radke (1998) concluded that, despite routine assessment showing adverse changes in weight and appetite, clinical intervention was not instigated for 40% of patients with cancer. One reason for this may be that cachexia

associated symptoms are 'silent' symptoms when compared with pain or shortness of breath and therefore their importance is often underestimated, under diagnosed (Costantini et al, 2000) and under treated (Tape, 2004). Additionally, Latham (2001) found that, health care professionals had insufficient knowledge of cancer cachexia. Latham (2001) concluded that this led to patients feeling pressurised to eat, thus highlighting a lack of knowledge with regard to the role of food and feeding in cachexia. A more recent study examining the experience of weight loss in patients with advanced cancer highlighted that nurse specialists perceived weight loss as 'taboo', because they felt there was little that could be done to rectify the patient's situation (Hopkinson et al, 2006).

A body of evidence exists to suggest that the management of cancer cachexia is under researched (Brown, 2002). Indeed, Cancer Research UK (2002) undertook a strategic review of issues facing cancer research, the results of which highlighted a lack of research activity in several areas, one of which was cancer cachexia (Steer, 2005). This may reflect the fact that our current understanding of the intricacies of the pathogenesis of cancer cachexia remains incomplete (Gordon et al, 2005 and Bing, 2005). As such it is argued that our current understanding of cancer cachexia is 'very limited' (Barber et al, 1999). It is further speculated, that although there have been vast advances in cancer research over the past thirty years, little progress has been made in the treatment of cancer cachexia (Gordon et al, 2005). Indeed, there is currently no clear diagnostic criterion for cancer cachexia. Furthermore, no therapy has produced adequate results to be considered a conventional standard of treatment (Barber, 2002; Muscaritoli et al, 2006), thus cancer cachexia remains a significant cause of morbidity and mortality in clinical practice (Muscaritoli et al, 2006; Barber et al, 2000).

It must be noted that cancer cachexia is known to be a multidimensional and multifactorial process (Bing, 2005). However, to date, this has not been reflected in either the design or reporting of research studies. Furthermore, much more understanding of what constitutes meaningful patient - focused end points is needed in cachexia research, in order to meet the needs of this client group. Thus, understanding the experience of cancer cachexia, from the perspective of patients and their significant others, is essential to develop plans of care, which recognise and meet the needs of this client group. It is suggested that if nurses are to understand, support and give the best care to their patients, they must understand the illness experience (Pearce and Lugton, 1999). This would enable nurses caring for patients with cachexia to work towards the NHS mandate of "achieving a high quality health service, which is responsive to peoples' needs" (NHS, 2007, p.2). Thus research which explores the complex and dynamic nature of cancer cachexia for both patients and their significant others is needed. An in-depth understanding of this phenomenon is essential if holistic patient-centred care, which is responsive to the needs of this client group is to be developed and if clinical practice is to improve.

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Developing a patient focused service for myeloproliferative disorders: the telephone follow-up clinic

**J Tonkin, Nurse Consultant
Haematology, Colchester**

Introduction

The NHS Modernisation Agency, through its work with thousands of NHS clinical teams, has identified ten high impact changes that organisations in health and social care can adopt to make significant, measurable improvements in the way they deliver care¹. The view of the Department of Health is that if these principles were adopted systematically by the whole NHS:

- The experience of patients would be greatly enhanced by more appropriate and timely care
- Clinician hours, hospital bed days and appointments in primary and secondary care would be saved
- Clinical quality and clinical outcomes would be tangibly improved
- Recruitment and retention of staff would significantly improve with enhanced job satisfaction

Each year in the NHS there are 37 million 'follow-up' appointments. A significant proportion of these follow-up visits are clinically unnecessary, create inconvenience and anxiety for patients and waste valuable resources. 75% of all outpatient 'Did Not Attends' (DNA) are for follow-up appointments¹. There are more than four million follow-up DNAs per annum, which costs the NHS more than £100 million a year.

Much of the current emphasis of redesign work is on the front end of the patient process examining demand management and avoiding initial hospital visits. Follow-up arrangements have not typically been the focus of attention. To date common practice has been to invite patients for a follow-up appointment 'just in case'. If practice is changed to that based upon, 'no follow-up unless there is a specific reason', i.e. clinical need or patient-led request, this would indisputably reduce the number of unnecessary follow-ups and DNAs. Reducing clinically needless and inconvenient follow-up visits frees up clinical resources for initial consultations.

The author of this paper, as a Haematology Nurse Consultant, has been in post since January 2005 working with the multidisciplinary team managing the care of patients with benign and malignant haematological disorders. The Nurse Consultant post was established in order to facilitate the development of services to provide improved outcomes for patients with haematological disease.

The nature of haematological disease means that some patients are followed up on a long term basis as outpatients at varying degrees of frequency ranging from monthly to annually. These patients often attend hospital for a review of current blood parameters and are then advised that their disease is stable and a further follow-

up appointment is scheduled. This frequently means that the patient has to travel to the hospital, pay to park, wait for a blood test, wait for the test result and then wait to see the doctor for a very short consultation. It became clear early on in the role that this group of patients could be more effectively and efficiently managed by the nurse consultant including the management of pharmacological interventions. The management of drug treatment for this group of patients by a nurse has been made easier with the advent of nurse prescribing. As a supplementary nurse prescriber, the Nurse Consultant was able to manage the patients independently through the use of clinical management plans. As from May 2006 however, as an independent prescriber, the Nurse Consultant has been able to freely prescribe according to protocol from the entire BNF.

The telephone clinic was established with the aim of enabling patients to receive appropriate healthcare without the burden of a hospital visit, improving the patient experience. In addition this would have the benefit of freeing up consultant capacity, enabling them to increase capacity for new patients, ensuring that they get the appropriate treatment at the earliest opportunity. It was anticipated that the benefits of the Telephone Follow-Up Clinic would include:

- Potential reduction of DNA rates.
- Increased patient choice
- Reduced waits
- Nurse led clinic offering the patients more time
- Enhanced patient satisfaction
- Manage capacity and demand more effectively
- Redirected Consultant time for other clinical priorities
- Reduce the burden on the hospital transport system

A protocol was devised outlining those patients eligible for this method of follow-up. This includes those who are deemed by their consultant to have stable disease, but require regular monitoring of their blood parameters every 3, 6 or 12 months. This includes those who require monitoring whilst receiving treatment with drugs such as Hydroxy-carbamide. Haematological disorders referred to the clinic include:

Myeloproliferative Disorder

- Essential Thrombocythaemia
- Chronic Lymphocytic Leukaemia (early)
- Polycythaemia not requiring venesection
- Smouldering myeloma
- Monoclonal Gammopathy of uncertain significance (MGUS)
- Stable ITP
- Myelofibrosis

The clinics run on a weekly basis for one full session and are held in the nurse consultant's office where there is access to PAS and pathology systems. Each slot is ten minutes long giving adequate

time for consultation, checking results, documentation and dictating letters to GPs. Patients are referred by the Consultant Haematologist by letter that specifies the patient's management plan in terms of frequency of follow-up, trigger blood parameters and drug regimen details where appropriate.

Conclusion

From its inception to March 2007 150 long term follow-up patients from the Consultant Haematologists lists have been transferred to the telephone clinic. This has saved in excess of 380 outpatient appointments for the period 06/07 creating greater capacity to cope with increasing demand. The service was recently evaluated by undertaking a patient survey. Questionnaires were sent to all 93 patients registered at that time to the clinic. A total of 65 questionnaires were returned for the evaluation - an excellent response rate of 70%. 90% of patients preferred the option of a telephone clinic to attending the outpatient department. As expected the majority of patients found the telephone clinic convenient because the system saved on transport and travel, time, parking and waiting in the outpatient department.

From this experience it would appear that telephone follow-up positively impacts on the patient and the service. Patients receive appropriate and timely care in the right

setting and this in turn creates additional capacity in the outpatient setting for those that require it. Clearly this must have an impact on non-attendance at clinics and consequently save the NHS resources. However to avoid potential pitfalls the selection of patients is clearly crucial alongside unambiguous criteria for referral and protocols for the management of this group of patients².

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Northern Ireland Update

The Myeloproliferative specialist nurse in the Belfast City Hospital, Ruth Thompson, who outlined her role in the April 2008 edition of Blood Matters, has also commenced telephone follow-up to support patients and families. Initially she is concentrating on elderly and vulnerable patients who find it physically challenging to attend hospital appointments. If, following formal evaluation, this is considered to be a valuable model for patient review, it could be expanded to minimise outpatient attendance and increase patient choice for a greater number of patients with MPD.

New Haematology Clinical Nurse Specialists in Northern Ireland

We are delighted to report that two new haematology Clinical Nurse Specialists (CNS) have recently been appointed in Northern Ireland. Joanne Ogborn has taken up this post in the Ulster Hospital, Dundonald, where she previously worked as both a Staff Nurse and Ward Manager within the Haematology / Oncology Unit. Louise Gribben, formerly the manager of the Haematology / Oncology outpatient department of Craigavon Hospital, has been employed as the Haematology CNS within the Southern Trust. Both Joanne and Louise are looking forward to the challenge of developing a CNS service that is patient focused, dynamic and promotes and delivers high standards of evidenced based care. Present priorities are to establish partnerships with key professionals, departments and organisations in order to provide and coordinate an effective support and information service to patients and their families.

The HAI nurses' group would like to congratulate both Louise and Joanne, who are long standing members of HAI, and wish

them all the best in their future careers. If you would like to network with either of the CNS's they can be contacted at the e mail addresses below.

joanne.ogborn@setrust.hscni.net
louise.gribben@southerntrust.hscni.net



Checking out the HAI posters in preparation for their new roles. Louise Gribben from Craigavon Hospital (left) and Joanne Ogborn from the Ulster Hospital (right)

Acquired Aplastic Anaemia (Part 2). Supportive and definitive therapy.

By Claire Jess, Staff Nurse, Bridgewater Suite, Belfast Trust, City Hospital.

Part one of this article, addressing the pathogenesis and diagnosis of Aplastic Anaemia, was published in Blood Matters October 2008. This may be viewed at our website www.ncnm.ie/haing/newsletter.asp

Supportive therapy for Aplastic Anaemia (AA)

Psychological support for the patient, family and close friends is of great significance, requiring careful explanation of aplastic anaemia, its prognosis and important issues such as pregnancy (BCSH, 2003). AA causes a range of difficult and complex emotional reactions in patients such as anxiety, depression, anger, uncertainty and fear (BCSH, 2003).

Supportive therapy is aimed at treating the consequences of pancytopenia and definitive therapy at restoring the function of the marrow, so that the patient becomes independent of blood transfusions and to minimise the risk from bleeding or infection. These approaches vary considerably in their applicability and their short-term and long-term risks. Brodsky and Jones, (2005) explain how Haematopoietic Stem Cell Transplantation (HSCT) eradicates the immune attack and is the only treatment that is curative for AA. The conditioning regime incorporating AntiThymocyte Globulin (ATG) along with high-dose cyclophosphamide, and subsequent ciclosporin therapy, primarily targets the autoimmune attack, allowing endogenous stem-cells to re-establish normal or near-normal haematopoiesis. The treatment plan for each patient will be determined by disease severity, age, availability of a suitable stem-cell donor and general health at the time of diagnosis.

AA patients are at risk of bacterial and fungal infections. Sepsis caused by bacteria or fungal infections are the most common cause of death from AA, where risk is determined by the patient's neutrophil and monocyte count (Howard and Hamilton, 2008; Gafter-Gvili et al, 2005). Aspergillus infections especially have a high mortality rate in patients with severe AA due to frequent prolonged periods of severe neutropenia and monocytopenia (BCSH, 2003). Patients will require prophylactic antibiotics and antifungals. Prophylactic antibiotics are given to prevent gram-negative sepsis and include quinolone antibiotics such as ciprofloxacin (Nirenberg et al, 2006a; Gafter-Gvili et al, 2005; BNF, 2008). However, there is concern about the emergence of quinolone resistant bacteria and an increase in gram-positive infections; additionally ciprofloxacin cannot be used to treat febrile neutropenia if it is used prophylactically (BCSH, 2003). Neutropenic patients with fever may require immediate hospitalisation and treatment. Patients should be treated promptly with broad-range antibiotics and antifungals (Marsh, 2006; BCSH, 2003). A short course of G-CSF may be considered for patients not responding to antibiotics and antifungals, although there have been no controlled studies evaluating their use in AA patients with infections (BCSH, 2003).

A common problem with multi-transfused patients is the development of alloimmunization to leucocytes present in blood and platelet transfusions, by generating human leucocyte antigen (HLA) or non-HLA antibodies (Brodsky and Jones, 2005; BCSH, 2003). A long-standing study

by Kaminsky et al (1990), found that alloimmunization results in platelet refractoriness, as well as an increased risk of graft rejection after allogeneic bone-marrow transplant (BMT). Additionally, other causes of platelet refractoriness include sepsis and drugs such as vancomycin and amphotericin (BCSH, 2003). Nevertheless blood and platelet transfusions should be administered to maintain a safe haemoglobin and platelet count and not be withheld for fear of sensitising the patient.

Brodsky and Jones (2005) found that if the patient is a potential candidate for HSCT and has negative or unknown cytomegalovirus (CMV) status, transmission of this virus should be avoided by the use of CMV negative products and leucocyte-depleted products. CMV is a herpes virus which in healthy people produces few symptoms, but poses a severe infection risk for immunocompromised patients. It is an important cause of mortality in HSCT patients (Marsh, 2007; BCSH, 2003; Fitzpatrick, 2002). Furthermore Brodsky and Jones (2005) emphasise that transfused patients with suspected AA should be administered irradiated products to prevent transfusion associated graft-versus host disease (TaGvHD). The BCSH (1996) advises that the routine use of leucocyte depleted blood and platelet transfusions are important in reducing the risk of alloimmunization. However, the absolute requirement for irradiated blood and platelet transfusions is from the beginning of the pretransplant conditioning regimen, applying to all patients undergoing allogeneic HSCT.

Blood and platelet donations from family members are not recommended by the BCSH (2003), to prevent alloimmunization to minor histocompatibility antigens from the potential bone marrow donor, therefore reducing the risk of graft rejection. After the patient has been stabilised, blood products should be prescribed judiciously as each transfusion carries a risk of reaction (Wilkins, 2000; BCSH, 1999).

As well as blood and platelet transfusional support, pharmacological agents may reduce blood loss. The use of oral tranexamic acid, an antifibrinolytic drug, can be introduced to help control gingival bleeding, and norethisterone to control heavy menses, both of which often affect individual's quality of life (BCSH, 2003). Haemoglobin concentration has been shown to have a considerable impact on the incidence of fatigue and subsequently quality of life (Foubert, 2006). Often patients can receive supportive care in their own home by community-based nursing services, promoting quality of life especially for those who may have to travel far to hospital or are simply too fatigued.

There are currently no safe and effective haematopoietic growth factors to support red cells and platelets in patients with AA (Marsh, 2007). Deficiency of haemopoietic growth factors, such as erythropoietin, granulocyte-colony stimulating factor (G-CSF), thrombopoietin, and granulocyte-monocyte-colony stimulating factor, is not the cause of the bone marrow failure in AA. Concentrations of haemopoietic growth factors are very high in patients in a compensatory attempt to increase blood production. Therefore they should not be implemented as an alternative to supportive or definitive therapy (BCSH, 2003). However, these growth factors are commonly used after immunosuppressive therapy or high-dose cyclophosphamide to accelerate

haemopoietic recovery; conversely their use has not been shown to improve survival (Brodsky and Jones, 2005). Additionally a concern of using erythropoietin is the possibility for inducing severe or sudden worsening of anaemia due to red cell aplasia from anti-erythropoietin antibodies as demonstrated by Casadevall et al, (2002). Furthermore, in combination with other drugs like ciclosporin, there is the potential for toxicity (Marsh, 2006). To date there has been no clinical research in the use of recombinant human thrombopoietin in AA. In addition there have been no controlled studies evaluating the use of G-CSF or other haemopoietic growth factors in the treatment of severe infection. Marsh (2007) suggests that haemopoietic growth factors should only be used to treat a severe infection that is not responding to antibiotics, although most patients with severe AA will predictably show no increase in neutrophil count.

Definitive therapy

Except for a minority of patients with mild AA, all patients will require some form of definitive therapy. The standard definitive treatment for a newly diagnosed patient with AA is either allogeneic stem-cell transplantation from an HLA-identical sibling, or immunosuppressive therapy with a combination of ATG and ciclosporin. Patients with severe AA, if they are <40 years old and have an HLA compatible sibling, should be transplanted up front, and should not receive prior immunosuppressive therapy (Marsh, 2007). Delay in transplantation will increase the risk of life-threatening infections and complications of multiple blood and platelet transfusions. However not all patients are eligible for stem-cell transplantation, nor have an appropriate donor and therefore can only be treated with immunosuppression.

Immunosuppression improves marrow function in up to 80% of patients with AA, with a 5 five year survival rate similar to those after BMT. However not all patients achieve normal blood counts and many relapse, become dependent on ciclosporin, or develop secondary clonal disease such as paroxysmal nocturnal haemoglobinuria (PNH) or myelodysplastic syndrome (MDS).

Antilymphocyte globulin (ALG) or antithymocyte globulin (ATG) combined with ciclosporin and corticosteroid is the recommended therapy for patients who lack a matched sibling donor, and has been especially effective in older adults and in patients with severe neutropenia. ATG is cytolytic towards T cells and inhibits T-cell formation, thereby rapidly reducing circulating lymphocytes.

ATG has many side effects, some serious and others very uncomfortable, as ATG is composed of foreign proteins. The most serious reaction is anaphylaxis. Patients often develop serum sickness due to their body's reaction to the foreign proteins forming 'protein complexes' (normally 7 days post ATG). This is manifested in symptoms such as fever, rigors, chills, joint pain, hives or skin rash and renal toxicity; it can be treated with analgesics and corticosteroids

Ciclosporin is normally commenced after completion of the ATG treatment and continued for at least six months. It is a specific T-cell inhibitor preventing the production of interleukin-2 and interferon, whilst allowing the production of colony-stimulating factors. However ciclosporin requires regular monitoring of blood levels so that the dose can be adjusted, allowing the maximum therapeutic effect without causing kidney or liver damage. Corticosteroids are usually started with ATG at a high dose and gradually reduced over four weeks. Toxicity can be problematic, including hypertension, hyperglycaemia and fluid retention;

they also have a masking effect on fever and infections due to their immunosuppressive affect.

The standard conditioning and GVHD prophylaxis regimen prior to bone-marrow transplantation (BMT) is a non-myeloablative, highly immunosuppressive regimen. The current standard regimen is 200mg/kg cyclophosphamide and ATG with ciclosporin and methylprednisolone. Conditioning further increases the risk of infection, hence the need for isolation. There is no indication for the role of using total body irradiation-based regimens as this can increase the risk of GVHD, pneumonitis, secondary tumours and infertility (Marsh, 2007). Fertility is usually well preserved or near normal after BMT for AA, where irradiation is not used. The degree and severity of the complications and side-effects of BMT depend upon the type of transplant, age, fitness, of the individual and the nature of the conditioning regimen. Advice may be given to patients regarding support groups such as the Aplastic Anaemia Support Group (www.lrf.org.uk).

Conclusion

Acquired Aplastic Anaemia is a rare haemopoietic stem-cell disorder resulting in pancytopenia and hypocellular bone marrow where haemopoietic cells are replaced by fat cells (Brodsky and Jones, 2005). AA is a disease that historically had an alarming reputation, but now thanks to the greatly improved means of support and treatment, many patients should recover.

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Forthcoming Events

29th March - 1st April 2009. European Blood and Marrow Transplantation (EBMT) Annual Conference. Goteborg, Sweden. <http://www.congex.ch/EBMT2009/>

24th April 2009. Haematology Association of Ireland Nurses' and AHP's Group, Spring Study Day. Hilton Kilmainham Hotel, Dublin. <http://www.ncnm.ie/haing/index.asp>. E mail s.cassidy@indigo.ie

27th - 29th April 2009. British Society of Haematology (BSH) Annual Meeting. Brighton. <http://www.bshconferences.co.uk/>

30th April - 3rd May 2009. Oncology Nursing Society Annual Congress. San Antonio. <http://ons.org/meetings/congress09/activities.shtml>

4 - 7th June 2009. European Haematology Association Congress. Berlin. <http://eha.eurocongres.com/14th/berlin.asp>

24th June 2009. European Blood and Marrow Transplantation (EBMT) UK Nurses Group. Reading. <http://www.ebmt.co.uk/>

15th October 2009. British Society of Blood and Marrow Transplantation (BSBMT) Education Day. London. http://www.bsbmt.org/cms_pages/10-Upcoming-Meetings

16th - 18th October 2009. Haematology Association of Ireland Annual Conference. Lyrath Estate, Kilkenny. <http://haematologyireland.org/> E mail s.cassidy@indigo.ie

3rd November 2009. UK Myeloma Forum Education Day. Royal Institute of British Architects, London. <http://www.ukmf.org.uk/meetings.htm>

4th November 2009. European Blood and Marrow Transplantation (EBMT) UK Nurses Group. Derby. <http://www.ebmt.co.uk/>

5th - 9th December 2009. American Society of Haematology (ASH). New Orleans. <http://www.hematology.org/meetings/2009/index.cfm>

This poem is written by the husband of a patient who successfully underwent bone marrow transplant a number of years ago. She continues to do well and credits this to the love and dedication shown by her husband.

Our Living Legend

Christmas coming, we're all full of delight,
She is worried, bruises give her a fright.
She's tearful, tired and feeling run-down
December, she fainted, Will she ever come round?
Shock, fear, anger and dread
Emotions set in, the bad news has spread.

White cells multiply, platelets are low
Hair falls out, will it ever grow?
Days are long, nights awake...
'Why me for heavens sake!'
Infusions, biopsies, drips on Ward 10
Will life ever be normal again?
Christmas passes, no-one cares
Priorities change - she's in our prayers.

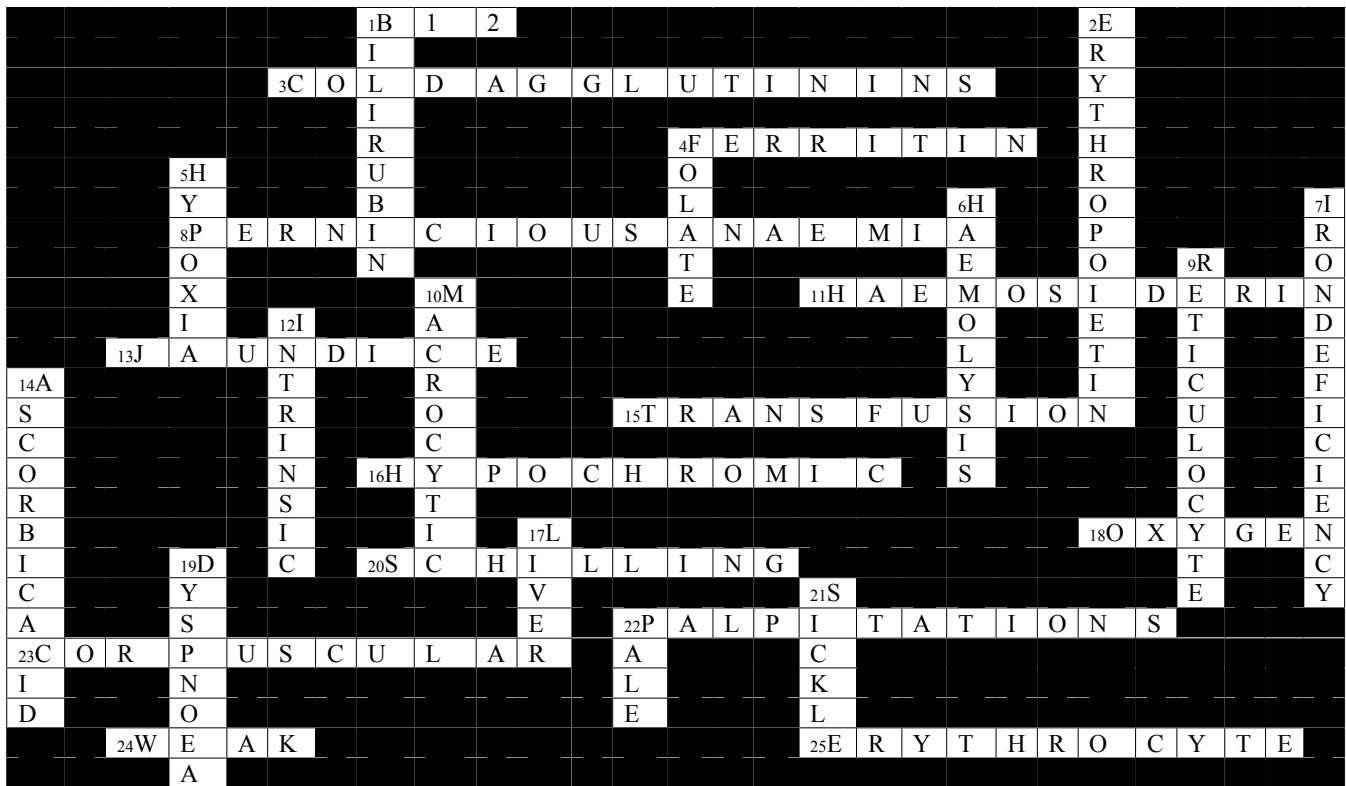
New Year comes, transplant in sight
3 siblings tested - Pray one is right.
Thank-you God for a perfect match,
A brother who's strong and an excellent catch!

More chemo, radio and very ill...
She stays positive and we are still.
July arrives and home is in sight...
A brave girl, after a courageous fight.
She's come through a time we can't even imagine..
She's our light, our living legend.

Poetry
Corner

Anaemia Crossword Solution

Congratulations to *Caitlin McCoy*, Manager of the MacDermott Unit, Ulster Hospital (and former chair of the HAI nurses' group) who was the first correct entry drawn to win the €50 prize. We were delighted with the number of correct entries submitted on this occasion - better luck next time!!



CLUES ACROSS

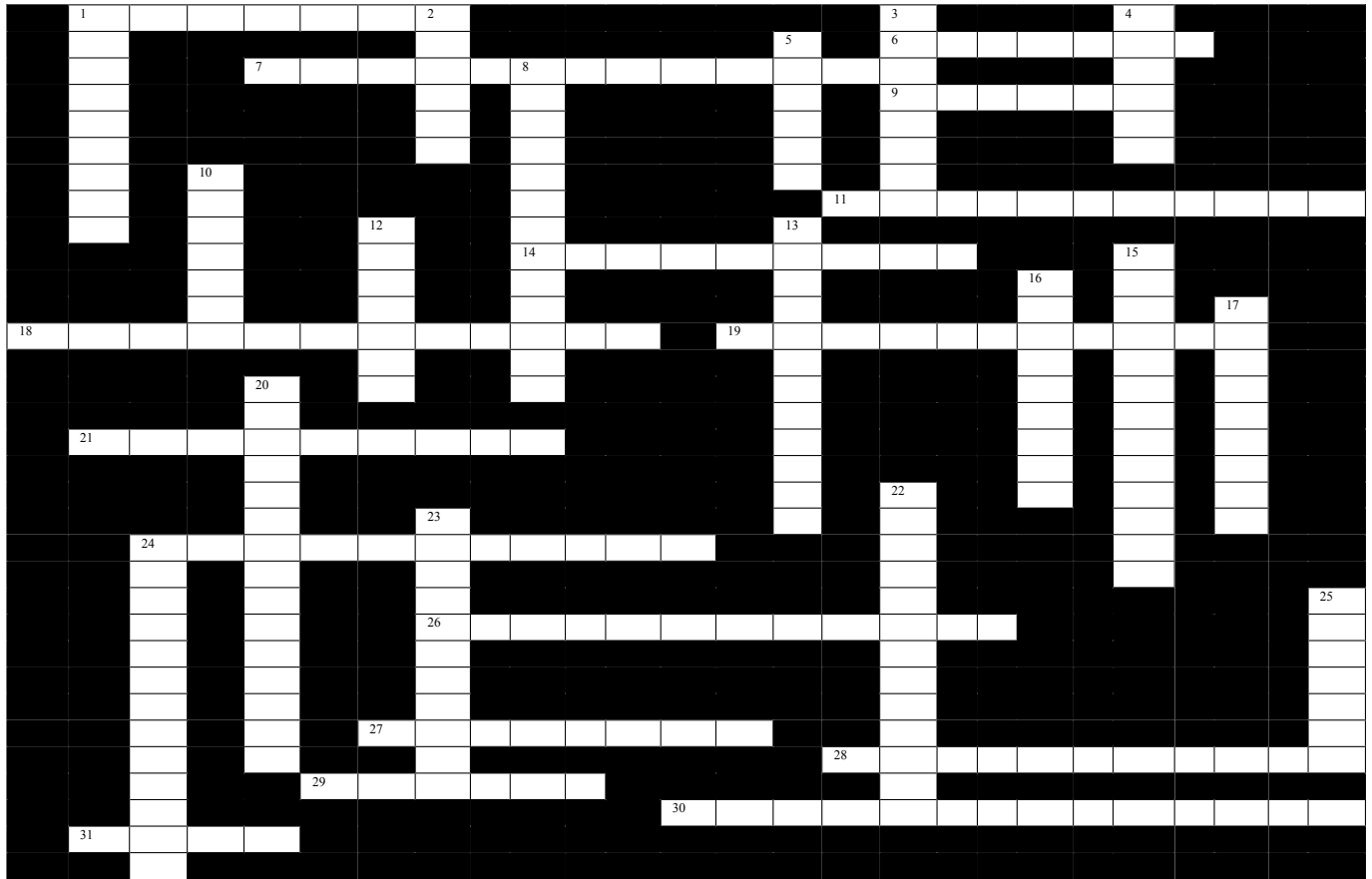
- 1 This nutrient is one short of unlucky
- 3 Cool clumps (2 words)
- 4 This wee weasel is in
- 8 Pale and spiteful (2 words)
- 11 Erin and Sidney with the haemo prefix
- 13 Stained sclera
- 15 Across the combination
- 16 This cell will dream of having haem!
- 18 A gaseous mobile network
- 20 A test of old British currency
- 22 Uncomfortable irregularity
- 23 The archaic middle word in MCV
- 24 52 of these P.A.
- 25 aka RBC

CLUES DOWN

- 1 Two boys names together
- 2 A three letter hormone
- 4 The wee horse was hungry
- 5 This may cause a shade of azure
- 6 I'm having a breakdown!
- 7 You'll have wrinkled clothes if this is the case (2 words)
- 9 When I grow up I want to be a red cell
- 10 Obese cell
- 12 Inherent factor
- 14 Helps assimilate iron (2 words)
- 17 Often served with onions
- 19 Leave me breathless...
- 21 Scythe
- 22 An old bucket

€50 Prize Winning Crossword

This crossword is specific to bleeding and clotting, so keep focused on that. Closing date for competition 31/07/09. The first randomly selected entry drawn after that date will be eligible for the prize. Good luck!



Cryptic clues

Across

1. Mass movement
6. Aspiring to drop a letter
7. In the midst of DIC
9. Have you got the X?
11. The end is at the beginning of helium
14. Outside pathway
18. Bleed out
19. Gush
21. Latin for look at Moses
24. Usually A or B
26. Kleenex component (2 words)
27. 11th Vitamin
28. Dissolve
29. Ship
30. Tell Ann it ends in Asia
31. The best of three in haemophilia

Down

1. Knows it can bleed
2. Use this partition to reveal your coag
3. The murder weapon for Roland
4. Delicate mesh
5. Yellow suspension
8. Shares the same initials as the Golf or Beetle (2 words)
10. Tarry waste
12. Contains two contented pussycats
13. It's all in your head
15. Pubertal platelet
16. Winter holiday
17. Going dotty
20. Starts with tears
22. The nation stuck together in the end
23. It just happened
24. Combined bleed
25. Can be light weight

NAME _____ JOBTITLE _____

WORKADDRESS _____

CONTACT NUMBER _____ **Entries should be sent to Caroline McCaughey, 50 Elmwood Ave, Belfast BT9 6AZ**