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Abstract

Myeloma is a challenging blood cancer characterized by bone destruction, hypercalcaemia, renal insufficiency and anaemia. Although myeloma remains incurable, recent advancements in treatments have resulted in significant improvements in morbidity. The use of immunomodulatory drugs, thalidomide, lenalidomide, pomalidomide (in clinical trials) and the proteasome inhibitor, bortezomib, in conjunction with conventional chemotherapy and supportive therapies have resulted in a major shift in approach to treatment and an improvement in patients' quality of life. Nurses need to keep up to date with current treatments for myeloma and their related side effects. In addition, nurses play a key role in the co-ordinating the multidisciplinary approach to care for myeloma patients.

Key words: myeloma, haematology, cancer

Introduction

Myeloma (often referred to as multiple myeloma) is the second most common cancer of the blood (Devenney and Erikson, 2004), and accounts for about 1% of all new cancers (Faiman, 2007). More specifically, myeloma is a B-cell malignancy which results in uncontrolled growth and division of plasma cells (Harrousseau, 2002). Myeloma is characterized by anaemia, renal dysfunction, lytic bone lesions and the presence of excess monoclonal immunoglobulin. Myeloma remains incurable but the development of newer treatments has improved survival rates (Faiman, 2007).

It is a cancer of older persons with the average age of diagnosis being between 65 and 68 years of age (Dvorak, 2006). However, myeloma can occur in younger persons; 2% of myeloma patients are aged under 40 years and 5% are aged under 50 years (King 2006). Recent data published by Tureson et al (2010) suggests that its incidence will continue to rise due to an ageing population. Tureson et al (2010) report that the incidence of myeloma among older people in Sweden with the disease aged 80 years or older doubled between 1950-1959 and 2000-2005.

The aetiology of myeloma is not known but certain factors have been associated with its cause. Radiation is a strong risk factor as is exposure to agricultural chemicals (Hussein, 1994). Some evidence exists to suggest that the risk of developing myeloma runs in some families, with 100 cases of familial myeloma already reported (Coleman et al, 2009). However, it is not known if familial myeloma is a result of genetic factors, environmental factors or both of these (Coleman et al, 2009).

There are also two forms of asymptomatic plasma disorders that may in time develop into myeloma. These are monoclonal gammopathy of uncertain significance (MGUS) and smouldering myeloma (SMM) (Rajkumar, 2005). All myeloma evolves from MGUS (Kumar et al, 2009), and patients with a diagnosis of MGUS or SMM require regular follow-up in view of their life-long risk of developing myeloma (Rajkumar, 2005).

Nursing management of myeloma is challenging and nursing goals may vary and change over time, requiring ongoing assessment, evaluation and review (Sheridan, 1996). However, in order to provide effective nursing management and understand the treatment regimes for myeloma patients, nurses must firstly understand the pathophysiology of this cancer.

What are the physiological events surrounding myeloma?

The first essential step in understanding myeloma is to understand what a plasma cell is and how it matures. The body's immune system has several types of cells that work together to fight infection and disease. All immune cells begin as stem cells which have the ability to mature into either lymphoid or myeloid cells. The myeloid cells further mature into platelets, neutrophils, eosinophils and basophils. The lymphoid stem cell divides into either T or B lymphocytes. When B-cell lymphocytes respond to an infection, they mature and change into plasma cells (Figure 1), which produce and release proteins called immunoglobulins (antibodies) which attack and help kill disease-causing germs. Normally mature plasma cells occupy less than 5% of the bone marrow (Mangan, 2005).

Each immunoglobulin consists of two polypeptide chains, two light chains and two heavy chains (Figure 2). The light chains are known as kappa and lambda while the heavy chains define the five classes of immunoglobulins: IgG, IgA, IgM, IgD and IgE (Sheridan, 1996). Each immunoglobulin has a particular role and function (Devenney and Erickson, 2004). Myeloma is an abnormal production of one of these immunoglobulins (Harousseau, 2002). This overproducing protein is known as the M protein or monoclonal protein. (Figure 3). In myeloma, patients produce more than 10% mature plasma cells in the bone marrow (Mangan, 2005).

How is myeloma diagnosed?

Diagnostic tests for myeloma include serum or urine protein electrophoresis and bone marrow aspirate analysis (Nau and Lewis, 2008). The diagnosis is usually confirmed by demonstration of a paraprotein in the serum or urine and/or lytic lesions on x-ray in conjunction with over 10% plasma cells in the bone marrow aspirate (Greipp, 1992). Moreover, although the presence of an M- Protein is the hallmark of myeloma, 1-2% of patients will have what is termed 'nonsecretory myeloma' with no M protein detectable in their serum or urine samples or evidence of light chains on serum analysis (Kumar et al, 2009). For the staging of myeloma, bone x-rays reveal any lytic lesions or vertebral compression fractures (Nau and Lewis, 2008). Magnetic resonance imaging and positron emission are also useful in patient evaluation (Nau and Lewis, 2008). The diagnosis of myeloma should be made using the International Myeloma Working Group (2003) criteria (Table 1).

What are the effects of myeloma on the body?

Bone destruction

Bone destruction is a hallmark of myeloma and up to 90 per cent of myeloma patients develop bone lesions (Roodman, 2010). Almost all myeloma patients develop osteolytic bone lesions that can cause hypercalcaemia, pathologic fractures, and severe bone pain and has major impacts on quality of life (Giuliani et al, 2006). Bone pain may also herald new evidence of disease (Sheridan, 1996).

The exact mechanisms involved in the pathogenesis of the bone disease associated with myeloma patients are not completely understood. According to Heider et al (2006), osteoclasts (cells responsible for bone resorption) are stimulated by Osteoclast Activating Factors (OAFs). OAFs are cytokines produced by myeloma cells and have been identified as the cytokines responsible for the bone lesions associated with myeloma (Sheridan, 1996). Interaction between myeloma cells and the bone environment leads to

increased bone reabsorption which is not matched with an increase in bone formation, resulting in bone disease (Kyle, 1999).

The diagnosis of bone lesions (lytic) is best observed on plain x-ray films rather than bone scan due to bone scans demonstrating abnormalities in bones where there is an increase osteoblast activity while myeloma lesions are a result of increased osteoclast activity (Sheridan, 1996).

Hypercalcaemia

The destruction of bone due to the osteoclast activity without matched bone formation leads to increased calcium secretion and subsequent hypercalcaemia. Hypercalcaemia normally presents itself with symptoms such as nausea, vomiting, lethargy and confusion. If left untreated hypercalcaemia leads to renal failure, progression of neurological symptoms, cardiac arrest or coma (Sheridan, 1996).

Renal insufficiency

Renal insufficiency occurs in approximately fifty percent of all individuals diagnosed with myeloma (Sheridan, 1996). A variety of renal insufficiencies occur with myeloma. The most common being hypercalcaemia, primary immunoglobulin light chain (AL) amyloidosis, myeloma kidney and cast nephropathy (Nozza et al, 2006). AL amyloidosis results in light chain portions of monoclonal immunoglobulins forming amyloid fibrils which are deposited in the kidney (King, 2006). Accumulation of these insoluble fibrils causes progressive disruption occlusion and subsequent damage to the structure and function of the kidney (Nakamoto et al, 1984). Myeloma kidney occurs as a result of the light chain component of the abnormal immunoglobulin depositing within the renal tubules. With cast nephropathy, renal inflammation results from an excess of filtered light chains that are transported to the kidney resulting in obstructive casts in the renal tubule (Clark and Garg, 2008).

Renal insufficiency among myeloma patients usually identifies itself as an elevation in creatinine, urea, and potassium or calcium level and can present at diagnosis or throughout the course of the disease. Presentation of renal insufficiencies is a poor prognostic factor (Blade et al, 1998; Eleutherakis-Papaiakovou et al, 2007). However, at diagnosis, if renal impairment is resolved, the patient's prognosis returns to that of a patient without renal impairment at presentation (Eleutherakis-Papaiakovou et al, 2007).

A central aspect of supportive care is adequate hydration, and although it is reported that a high fluid intake alone can reverse renal failure, Dimopoulos et al (2008) suggest that hydration alone will only reduce light chain concentrations and antimyeloma treatments (with agents not excreted by the kidneys) should also be given. In addition to this, other supportive measures include management of hypercalcaemia, with dose adjusted biphosphonates must be used cautiously and only when creatine levels improve (Dimopoulos et al, 2008). Moreover, supportive care in renal failure includes prompt treatment of infections, avoiding use of NSAIDs, aminoglycoside antibiotics and contrast media dyes (Durie et al, 2003).

Anaemia

Unlike other hematological malignancies the erythrocyte (red cells) line is most commonly the most effected in myeloma (Sherdian, 1996). Anaemia is a common presenting feature of myeloma and may be characterized by fatigue, weakness and dyspnea (Harousseau, 2002). The cause of anaemia is multifactorial and related to the replacement of erythrocyte progenitors in the bone marrow by plasma cells leading to decreased production of erythrocytes, low erythropoietin levels and increased erythrocyte destruction (Sherdian, 1996). Typically the degree of anaemia is related to the tumor mass (Mangan, 2005). In addition, the abnormal monoclonal protein can coat circulating peripheral erythrocytes to stack up like a roll of coins (rouleaux formation). When this occurs these stacks of red cells are unable to move through the capillary bed resulting in

increased erythrocyte destruction (Duffy, 1992). Erythropoietin is often given to patients whose anaemia persists after starting active treatment (Crotty, 2004).

Infection

As a consequence associated with plasma cell replacement in the bone marrow, immunosuppression can lead to neutropenia. Therefore recurrent infections are common in myeloma patients and many die as a result of bacterial infections (Devenney and Erikson, 2004). Respiratory and urinary infections are the most common. Recurrent infections in myeloma patients are as a result of decreased amounts of immunoglobulin (immune paresis) being produced along side the ineffectiveness of the overproduced immunoglobulin.

Treatment options for myeloma and nursing management

Early intervention with asymptomatic patients is not required, however these patients require close monitoring. Treatment in myeloma is indicated for patients with symptomatic disease defined by the presence of myeloma related organ impairment.

Treatment of myeloma is in four stages: initial treatment, stem cell transplantation (once suitable in terms of age and co-morbidities etc.), maintenance therapy and relapsed or refractory treatment (Wiley, 2007). The goals of initial treatment for myeloma are as follows: a) facilitate fast control of the disease and reverse any myeloma-related complications such as hypercalcaemia, renal dysfunction and anaemia; b) be well tolerated with minimal side effects; c) decrease the risk of early morbidity; and d) allow successful harvesting of stem cells when stem cell transplant is decided as a treatment option (Kumar et al, 2009). Improving the depth of response is becoming increasingly important as many studies in transplant and non-transplant settings have suggested an important link with maximal response attained and long term outcome after initial therapy and that increasing the complete remission rate after transplant results in prolonged progression free survival and overall survival (Lahuerta et al, 2008). Although high dose treatment is recommended where possible, many patients will not be

able to receive such therapy because of advanced age, co-morbidities or poor performance status. Treatment decisions should be reviewed in an MDT and should take into account individual factors and patient choice (BSCH guideline, 2010).

Chemotherapy induction

For patients older than sixty five years of age conventional treatment using melphalan and prednisone has been the treatment of choice since the 1960's. Combination chemotherapy was introduced in the early 1970s with regimes such as VAD (vincristine, doxorubicin, dexamethasone) (King, 2006). VAD was used widely in younger patients who had good performance status and who were eligible for stem cell collection and transplant. The introduction of novel agents described below usually in combination with steroids has lead to clear improvement in survival of patients with myeloma (Kumar et al, 2008). However, much work is needed to determine the best sequence and combinations of therapies. It is therefore essential wherever possible that patients are entered into clinical trials (BSCH 2010). Moreover, in the UK, the availability of drugs in the NHS is governed by National Institute for health and clinical excellence (NICE). Thus algorithms for myeloma treatment are difficult to present in the frontline setting. Nevertheless, the recently proposed algorithm for patients with relapsed myeloma by the BCSH which incorporates NICE recommendations (BSCH, 2010) is useful.

Novel immunomodulatory drugs

Thalidomide, lenalidomide and pomalidomide (both derivatives of thalidomide) are known as novel immunomodulatory drugs (IMiDs). Thalidomide was previously withdrawn for causing birth defects when used for morning sickness in the late 1950s and early 1960s (Quach et al, 2010). However, researchers at the University of Arkansas used Thalidomide to treat patients with relapsed and refractory myeloma on a compassionate basis in 1997 (Singhal et al, 1999). Surprisingly thalidomide was remarkably effective in these patients, most of whom had no other treatment options. The MRC Myeloma IX trial compared CVAD (cyclophosphamide, vincristine, doxorubicin and dexamethasone) with CTD (cyclophosphamide, thalidomide and dexamethasone). Preliminary results have

shown higher response rates in the CTD arm but information on the benefit for progression-free and overall survival is awaited. Other studies using thalidomide with myeloma patients also reported successful outcomes (Rajkumar et al, 2000; Kumar et al, 2003). This is now the most widely used combination in the UK. Stem cell mobilisation and harvesting are not adversely affected by the use of thalidomide- containing regimens (BCSH, 2010). NICE has recently recommended that Thalidomide should be available to newly diagnosed myeloma patients for whom high-dose chemotherapy and stem cell transplantation is unsuitable (NICE, 2010).

Thalidomide possesses the ability to inhibit blood supply to a tumour (antiangiogenesis), and is used in combination with steroids and newer IMiDs. The most common side effects of thalidomide include peripheral neuropathy, fatigue, sleepiness, constipation and thromboembolism (Dvorak, 2006). Peripheral neuropathy can affect patients' quality of life and compromise their treatment regime (Tariman et al, 2008). All myeloma patients receiving thalidomide therapy should be assessed before treatment starts for signs and symptoms of peripheral neuropathy, and re assessed throughout the treatment schedule (Tariman et al, 2008). For younger patients taking thalidomide, education regarding contraception and pregnancy avoidance is essential (Dvorak, 2006). Patients prescribed thalidomide are monitored on risk management programmes such as the System for Thalidomide Education and Prescribing Safety (S.T.E.P.S.) (Uhl et al, 2006) and the Thalidomide Education and Risk Management System (TERMS) (Ooba et al, 2010).

More recently, Lenalidomide, a derivative of thalidomide, is responsible for improving survival of myeloma patients (Zeldis et al, 2010). In combination with other agents, such as dexamethasone, lenalidomide is now used in newly diagnosed, relapsed/refractory, and high-risk smoldering myeloma (Zeldis et al, 2010). Moreover, current trials are investigating the use of lenalidomide as a maintenance therapy and as a preventive therapy in myeloma (Zeldis et al, 2010). This newer agent appears to have a greater anti myeloma effect than thalidomide but with less side effects for the patient (Wiley, 2007). However, lenalidomide does cause neutropenia and thrombocytopenia (Kettle et al,

2009). In the UK, NICE guidelines permit the use of Lenalidomide in combination with dexamethasone, within its licenced indication as an option for the treatment of myeloma only in patients who have received two or more therapies (NICE, 2009).

Although myeloma patients are at risk of thromboembolic events (deep vein thrombosis and pulmonary embolism) due to the nature of this disease and individual factors, the use of lenalidomide and thalidomide in the treatment of myeloma has been associated with an increased risk (Rome et al, 2008). However, this risk appears to be attributed to the use of either thalidomide or lenalidomide in combination with chemotherapy, steroids and erythropoietin (Kumar et al, 2009). Myeloma patients' risk of thromboembolic events can be monitored by nurses using the National Cancer Institute (NCI) Common Terminology Criteria for Adverse Events (CTCAE) and patients should receive prophylaxis for thromboembolic events if indicated (Rome et al, 2008).

A recent study from the Mayo clinic compared the efficacy and toxicity of lenalidomide plus dexamethasone (len/dex) versus thalidomide plus dexamethasone (thal/dex) as initial therapy for newly diagnosed myeloma patients. The study revealed that len/dex was better tolerated (the most common toxicity being neutropenia) when compared to thal/dex (most common toxicities being venous thromboembolism and peripheral neuropathy) (Gay et al, 2010). Gay et al (2010) also report that len/dex is more effective than thal/dex.

Lenalidomide and thalidomide also can cause serious gastrointestinal side effects, including constipation, diarrhea, nausea and vomiting (Smith et al, 2008). Nurses can use the National Cancer Institute Common Terminology Criteria for Adverse Events to determine the extent gastrointestinal side effects (Smith et al, 2008).

Pomalidomide, the newest of the immunomodulatory drugs has a combined chemical structure of thalidomide and lenalidomide (Quach et al, 2010). Clinical trials on the use

pomalidomide are ongoing but Lacy and Rajkumar (2010) report promising results in relapsed myeloma among patients who are refractory to other novel agents including lenalidomide, thalidomide and bortezomib.

Proteasome inhibitor (bortezomib)

Bortezomib was initially licensed in combination with melphalan and prednisone for elderly patients being treated for myeloma. This combination has shown to improve complete response, partial response rates and has improved time to progression (San Miguel et al, 2008). Moreover, bortezomib is also used in front-line therapy for younger patients prior to stem cell collection. Younger patients are deemed to be less than 65 years of age and eligible for stem cell transplant. However, these patients must also have good performance status and without other co-morbidities in order to be eligible for stem cell transplant (Harousseau et al, 2006). In the UK, bortezomib monotherapy is recommended as an option for the treatment of progressive myeloma in people who are at first relapse having received one prior therapy and who have undergone, or are unsuitable for bone marrow transplantation (NICE, 2007). In addition NICE has recently recommended that both Thalidomide and bortezomib should be available to newly diagnosed myeloma patients for whom high-dose chemotherapy and stem cell transplantation is unsuitable (NICE, 2010). .

Bortezomib requires frequent intravenous administration (usually twice a week, for two consecutive weeks with a 10 day rest period) and the actual administration only takes three to five seconds (Colson et al, 2008). However, myeloma patients often have to travel long distances to haematology oncology units for treatment which can have an adverse effect on their quality of life. In response to this, Meenaghan et al (2010) report on a consultant-led, nurse-coordinated service for the home administration of bortezomib in the West of Ireland. Nurses visit patients who live as far away as 100 miles from the hospital and assess for adverse side effects and administer bortezomib. This incentive is now also available to patients in Cork and Limerick .

Peripheral neuropathy is also a main side effect of Bortezomib treatment, with Corso et al (2010) reporting that increasing age represents the most relevant risk factor of this side effect (a risk increase of 6% per year of age). Similar to the approach taken with thalidomide therapy, all patients should be assessed regularly for this side effect and appropriate interventions used to manage peripheral neuropathy (Tariman et al, 2008).

Steroid therapy

Steroid therapy (dexamethasone, prednisone and prednisolone) has been used in the treatment of myeloma for over three decades. Steroids are used as single agents or in combination with other antimyeloma drugs, such as melphalan, lenalidomide, thalidomide and bortezomib (Faiman et al, 2008). High dose dexamethasone also plays a key role in managing renal dysfunction (Dimopoulos et al, 2008). However, steroids can affect many body systems and adversely affect myeloma patients' quality of life. Nurses therefore need to assess patients at baseline and regularly throughout treatment and should educate myeloma patients and their families about the potential side effects of steroid therapy (Faiman et al, 2008). A useful tool for nurses to use is the National Cancer Institution Common Terminology Criteria for adverse effects: Steroid related toxicity grades. These grades assess constitutional (e.g. flushing, insomnia), sexual dysfunction, psychiatric, immune, musculoskeletal, body image, ophthalmic, gastrointestinal, endocrine, cardiovascular and dermatologic effects of steroids on patients, and can be used by nurses to monitor toxicities and determine need for types of interventions (Faiman et al, 2008).

Biphosphonate therapy

Biphosphonate therapy is widely used for bone disease in myeloma. However, these agents may be associated with side effects, such as flu-like symptoms, renal toxicity and osteonecrosis of the jaw (ONJ) (Berenson et al, 2002). Nurses play an important role in

assessing patients' renal function by monitoring creatinine levels and fluid balance (Maxwell, 2007).

Osteonecrosis of the jaw usually presents as infection or necrotic bone in the mandible or maxilla (Lee, 2009). However, the risk appears to be with long-term therapy. It has been calculated that zoledronic acid is the most potent of the biphosphonate therapies with the mean time of bisphosphonate-associated osteonecrosis of the jaw (BONJ) after zoledronate treatment being 1.8 years and the minimum 10 months (Palaska et al, 2009). Nurses play a key role in educating patients on good oral hygiene. Maintaining good oral hygiene is central in preventing dental disease in patients taking bisphosphonate therapy (Ruggiero et al, 2009). Bisphosphonates also may have an anti myeloma effect (Avilés et al 2007).

Autologous stem cell transplantation (ASCT)

A major development in the management of myeloma was the first successful autologous stem cell transplantation in 1983 (McElwain and Powles, 1983). The ideal candidate for autologous stem cell transplantation was historically younger than 65 years of age with good performance status and without significant co-morbidities; many myeloma patients are therefore poor candidates for stem cell transplantation (Kettle et al, 2009).

Regimens with the use of novel agents are now used in the treatment of transplant eligible patients (Kettle et al, 2009). Generally, primary regimens for transplant eligible patients should not include alkylating agents (such as melphalan) because of their toxicity to stem cells and therefore their prohibitive effect on stem cell collection (Kettle et al, 2009). However, it is important to point out that because of the substantial responses achieved by newer chemotherapeutic agents, the role of transplant in the treatment of myeloma is now under scrutiny (Kettle et al, 2009). It is however likely that ASCT will further increase the rate and depth of responses achieved with induction therapy with a consequent improvement in progression free survival. There is therefore no current

evidence to support deferral of the first ASCT until the time of first relapse though prospective studies are underway to explore this possibility further (BSCH, 2010).

Living with a diagnosis of myeloma

Supportive therapies such as erythropoietin (EPO), granulocyte stimulating colony factors (GSCFs) and transfusions have contributed to patients' quality of life. Nevertheless, pain is a big issue for myeloma patients (e.g. Poulos et al, 2001) and analgesics play a key role in pain relief for patients with myeloma. NSAIDs should be avoided with myeloma patients, and stronger opioids such as morphine are needed if pain persists (Reich, 2003). Other interventions for pain include radiotherapy, bisphosphonate therapy and treating any identified underlying causes of pain. In addition, pain relief and a reduction in the disabling effects of myeloma pain is achieved following vertebroplasty for painful vertebral body fractures (Tancioni et al, 2010).

Patient education is essential in the management of myeloma. Patients need to be advised on adequate hydration to minimise renal dysfunction (Dimopoulos et al, 2008). Moreover, patients should be advised to attend hospital for admission and intravenous hydration if they are unable to take oral fluids due to an acute illness such as gastroenteritis (Crotty, 2004).

It is important to also give patients general advice on activity and back care. Immobility contributes to the development of hypercalcaemia and other complications of myeloma, such as infections, therefore patients should be encouraged to remain mobile (Crotty 2004). However, patients should be advised to avoid certain activities such as bending, lifting heavy items or undertaking other strenuous physical activities (Crotty, 2004).

There is a dearth of research which has explored myeloma patients' lived experience. Only three known qualitative studies have been published on this topic; Dahan and Auerbach (2006) (USA), Vlossak and Fitch (2008) (Canada) and Kelly and Dowling (in press) (Ireland). However, all three studies present an insight for nurses on what it is like

to live with this complex cancer. For instance, myeloma patients talk about their fear of recurrence (Kelly and Dowling in press) and an “obsession of how and when the end will come” (Vlossak and Fitch, 2008, p. 144). Moreover, myeloma patients have talked about living with a disease that they had never heard about before their diagnosis (Vlossak and Fitch, 2008), and their feelings of isolation because of the ‘unknown’ nature of their cancer (Kelly and Dowling, in press). Nurses play a pivotal role in continuing education of patients, families and the public on this rare disease.

Nurses play a key role in co-ordinating the multidisciplinary approach to care for myeloma patients. The specialist haematology nurse acts as patient advocate and helps orientate patients through the hospital system (Kelly, 2007). The haematology nurse’s role is also in educating myeloma patients on their disease and interventions to minimise risks of complications such as renal insufficiency, infections and spinal cord compression (Kelly, 2007). Myeloma patients can feel isolated and nurses should make patients aware of the support available to them by myeloma charitable organisations such as the International Myeloma Foundation (IMF), Myeloma UK, myeloma Ireland, myeloma euronet and The Multiple Myeloma Research Foundation in the USA. All raise awareness of myeloma for patients, their families and the general public. Moreover, support is provided through the IMF helpline, website, support groups and patient and family seminars.

Conclusion

Myeloma remains a complex disease to diagnose and treat. Promoting better quality of life through nurse co-ordinated multidisciplinary interventions is a central goal in the care of patients with myeloma.

As our understanding of the biology of myeloma continues to develop, increasing numbers of new potential therapies are emerging at a fascinating rate. Therefore nurses need to keep up to date with current treatments and their related side effects (Devenney

and Erikson, 2004). Although myeloma remains incurable, the emergence of new therapies has given much hope to patients living with this multifaceted blood cancer.

Key phrases

- Myeloma is a complex blood cancer involving plasma cells with many effects on the body including, bone destruction, hypercalcaemia, renal insufficiency and anaemia.
- The use of novel immunomodulatory drugs (thalidomide, lenalidomide and promalidomide) and the proteasome inhibitor (bortezomib), have resulted in better outcomes for myeloma patients.
- Nurses play a key role in education patients about the side effects of their treatments and monitoring their toxicity.
- Nurses need to educate myeloma patients on the importance of adequate hydration and appropriate activity and back care.
- Myeloma patients can feel isolated and nurses should make patients aware of various charitable myeloma organisations, which offer support.

References

Berenson J, Hillner BE, Kyle RA et al (2002) American society of clinical oncology clinical practice guidelines: The role of bisphosphonates in multiple myeloma. *Journal of Clinical Oncology* **20**(17): 3719-3736

Blade J, Fernandez-Llama P, Bosch F et al (1998) Renal failure in multiple myeloma: presenting features and predictors of outcomes in 94 patients from a single institution. *Archives of Internal Medicine* **158**(17):1889-1893

British Committee for Standards in Haematology in conjunction with UK Myeloma Forum (UKMF) (2010) Guidelines on the diagnosis and management of multiple myeloma.

Clark WF, Garg AX (2008) Plasma exchange for myeloma kidney: cast(s) away. *Kidney International* **73**(11): 1211-1213.

Coleman EA, Lynch H, Enderlin C et al (2009) Initial report of a family registry of multiple myeloma. *Cancer Nursing* **32**(6): 456-464

Colson K, Doss DS, Swift R, Tariman J (2008) Expanding role of bortezomib in multiple myeloma: Nursing implications. *Cancer Nursing* **31**(3):239-249

Corso ., Mangiacavalli S, Varettoni M, Pascutto C, Zappasodi,P, Lazzarino M (2010) Bortezomib-induced peripheral neuropathy in multiple myeloma: A comparison between previously treated and untreated patients. *Leukemia Research* **34**(4): 471-474

Crotty G (2004) Multiple myeloma- patient information and support and palliative therapy. *Irish Medical Times* November 19th: 50

Dahan JF, Auerbach CF (2006) A qualitative study of the trauma and posttraumatic growth of multiple myeloma patients treated with peripheral blood stem cell transplant. *Palliative & Supportive Care* **4**(4): 365-387

Devenney B, Erickson C (2004) Multiple myeloma, an overview. *Clinical Journal of Oncology Nursing* **8**(4): 401-405

Dimopoulos MA, Kastiris E, Rosinol L, Bladé J, Ludwig H (2008) Pathogenesis and treatment of renal failure in multiple myeloma. *Leukemia* **22**(8):1485-1493

Duffy TP (1992) The many pitfalls of diagnosis of myeloma. *New England Journal of Medicine* **326** (6): 394-396

Durie BGM, Kyle RA, Belch A et al (2003) Myeloma management guidelines: A consensus report from the Scientific Advisors of the International Myeloma Foundation. *Hematology Journal* **4**(6): 379-398

Dvorak C (2006) Common complaints, difficult diagnosis: Multiple myeloma. *Journal of the American Academy of Nurse Practitioners* **18**(5): 190-194

Eleutherakis-Papaiakovou V, Bamias A, Gika D et al (2007) Renal failure in multiple myeloma: Incidence, correlations, and prognostic significance. *Leukemia and Lymphoma*, **48**(2), 337-341.

Faiman B (2007) Clinical updates and nursing considerations for patients with multiple myeloma. *Clinical journal of oncology nursing* **11**(6): 831-840

Faiman B, Bilotti E, Mangan PA, Rogers K (2008) Steroid-associated side effects in patients with multiple myeloma: consensus statement of the IMF Nurse Leadership Board. *Clinical journal of oncology nursing* **12**(3 Suppl): 53-63

Gay F, Hayman SR, Lacy MQ et al (2010) Lenalidomide plus dexamethasone versus thalidomide plus dexamethasone in newly diagnosed multiple myeloma: a comparative analysis of 411 patients. *Blood* **115**(7): 1343-1350

Giuliani N, Rizzoli V, Roodman GD (2006) Multiple myeloma bone disease: pathophysiology of osteoblast inhibition. *Blood* **108**(13): 3992- 3996

Greipp PR (1992) Advances in the diagnosis and management of myeloma. *Seminars in Haematology* **29**: 24-25

Harousseau JL (2002) Management of multiple myeloma. *Reviews in Clinical and Experimental Hematology* **6**(3): 253-275

Harousseau JL, Attal M, Leleu X et al (2006) Bortezomib plus dexamethasone as induction treatment prior to autologous stem cell transplantation in patients with newly diagnosed multiple myeloma: Results of an IFM phase II study. *Haematologica* **91**(11): 1498-1505

Heider U, Fleissner C, Zavrski I, Kaiser M, Hecht M (2006) Bone markers in multiple myeloma. *European Journal of Cancer* **42**(11): 1544-1553

<http://www.nice.org/uk/guidance>

Hussein M (1994) Multiple myeloma: an overview of diagnosis and management. *Cleveland Clinic Journal of Medicine* **61**: 285-298

International Myeloma Working Group (2003) Criteria for the classification of monoclonal gammopathies, multiple myeloma and related disorders: a report of the International Myeloma Working Group. *British Journal of Haematology* **121**:749-757

Kelly M (2007) The role of the haematology nurse in caring for patients with myeloma. *Oncologynews* **1**(4): 20-21 www.oncologynews.biz

Kelly M, Dowling M (In press) Living with a diagnosis of myeloma: an ‘unknown’ cancer. *Nursing Standard*

Kettle JK, Finkbiner KL, Klenke SE, Baker RD, Henry DW, Williams CB (2009) Initial therapy in multiple myeloma: Investigating the new treatment paradigm. *Journal of Oncology Pharmacy Practice* **15**(3): 131-141

King T (2006) Myeloma. In: Grundy M, ed. *Nursing in Haematological oncology*. Bailliere Tindall, Edinburgh: 85-110

Kumar S, Gertz MA, Dispenzieri A et al (2003) Response rate, durability of response and survival after thalidomide therapy for relapsed multiple myeloma. *Mayo Clinic Proceedings* **78**(1): 34-39

Kumar SK, Mikhael JR, Buadi FK et al (2009) Management of newly diagnosed symptomatic multiple myeloma: Updated Mayo Stratification of Myeloma and Risk-Adapted Therapy (mSMART) consensus guidelines. *Mayo Clinic Proceedings* **84**(12): 1095-1110

Kyle RA (1999) Maintenance therapy and supportive care for patients with multiple myeloma. *Seminars in Oncology* **26**(5): 35-42

Lacy MQ, Rajkumar SV (2010) Pomalidomide: A new IMiD with remarkable activity in both multiple myeloma and myelofibrosis. *American Journal of Hematology* **85**(2): 95-96

Lahuert JJ, Mateos MV, Martinez-Lopez J, Rosinol L et al (2008) Influence of pre and post transplantation responses on outcome of patients with multiple myeloma: sequential improvement of response and achievement of complete response are associated with longer survival. *Journal of clinical Oncology* **26**, 5775-5782.

Lee J (2009) Complication related to bisphosphonate therapy: Osteonecrosis of the Jaw. *Journal of Infusion Nursing* **32**(6):330-335

Mangan P (2005) Recognizing multiple myeloma. *The Nurse Practitioner* **30**(3): 14-27

Maxwell C (2007) Role of the nurse in preserving patients' independence. *European Journal of Oncology Nursing* **11**(SUPPL. 2): S38-S41

McElwain TJ, Powles RL (1983) High-dose intravenous melphalan for plasma-cell leukaemia and myeloma. *Lancet* **2**(8354): 822-824

Meenaghan T, O'Dwyer M, Hayden P, Hayat A, Murray M, Dowling M (2010) Home administration of bortezomib: Making a difference to myeloma patients' lives. *European Journal of Oncology Nursing* **14**(2):134-136

Nakamoto Y, Hamanaka S, Akihama T, Miura AB, Uesaka Y (1984) Renal involvement patterns of amyloid nephropathy: A comparison with diabetic nephropathy. *Clinical Nephrology* **22**(4): 188-194

Nau KC, Lewis WD (2008) Multiple myeloma: Diagnosis and treatment. *American Family Physician* **78**(7): 853-859+860

Nozza A, Siracusano L, Armando S (2006) Bortezomib-dexamethasone combination in a patient with refractory multiple myeloma and impaired renal function. *Clinical Therapeutics* **28**(6): 953-959

Ooba N, Sato T, Watanabe H, Kubota K (2010) Resolving a double standard for risk management of Thalidomide: An evaluation of two different risk management programmes in Japan. *Drug Safety* **33**(1): 35-45

Palaska PK, Cartsos V, Zavras AI (2009) Bisphosphonates and time to osteonecrosis development. *Oncologist* **14**(11): 1154-1166

Poulos AR, Morie A, Gertz MD, Pankratz VS, White JP (2001) Pain, mood disturbance and quality of life in patients with multiple myeloma. *Oncology Nursing Forum* **28**(7): 1163-1171

Quach H, Ritchie D, Stewart AK et al (2010) Mechanism of action of immunomodulatory drugs (IMiDS) in multiple myeloma. *Leukemia* **24**(1): 22-32

Rajkumar SV, Fonseca, R, Dispenzieri A et al (2000) Thalidomide in the treatment of relapsed multiple myeloma. *Mayo Clinic Proceedings* **75**(9): 897-901

Rajkumar SJ (2005) MGUS and smoldering multiple myeloma: update on Pathogenesis, Natural history, and Management. *Hematology*: 340-345.

Reich CD (2003) Advances in the treatment of bone metastases. *Clinical journal of oncology nursing* **7**(6): 641-646

Roodman GD (2010) Pathogenesis of myeloma bone disease. *Journal of Cellular Biochemistry*, **109** (2): 293-291

Rome S, Doss D, Miller K, Westphal J (2008) Thromboembolic events associated with novel therapies in patients with multiple myeloma: consensus statement of the IMF Nurse Leadership Board. *Clinical journal of oncology nursing* **12**(3 Suppl): 21-28

Ruggiero SL, Dodson TB, Assael LA, Landesberg R, Marx RE, Mehrotra B (2009) American Association of Oral and Maxillofacial Surgeons position paper on bisphosphonate-related osteonecrosis of the jaw - 2009 update. *Australian endodontic journal : the journal of the Australian Society of Endodontology Inc* **35**(3): 119-130

San Miguel JF, Schlag R, Khuageva NK et al (2008) Bortezomib plus melphalan and prednisone for initial treatment of multiple myeloma. *New England Journal of Medicine* **359**(9): 906-917

Sheridan CA (1996) Multiple myeloma. *Seminars in Oncology Nursing* **12**(1): 59-69

Singal S, Mehta J, Desikan R et al (1999) Antitumor activity of thalidomide in refractory multiple myeloma. *New England Journal of Medicine* **341**(21): 1565-1571

Smith LC, Bertolotti P, Curran K, Jenkins B (2008) Gastrointestinal side effects associated with novel therapies in patients with multiple myeloma: consensus statement of the IMF Nurse Leadership Board. *Clinical journal of oncology nursing* **12**(3 Suppl): 37-52.

Tancioni F, Lorenzetti M, Navarria P et al (2010) Vertebroplasty for pain relief and spinal stabilization in multiple myeloma. *Neurological Sciences*: 1-7

Tariman JD, Love G, McCullagh E, Sandifer S (2008) Peripheral neuropathy associated with novel therapies in patients with multiple myeloma: consensus statement of the IMF Nurse Leadership Board. *Clinical journal of oncology nursing* **12**(3 Suppl): 29-36

Turesson I, Velez R, Kristinsson SY, Landgren OLA (2010) Patterns of multiple myeloma during the past 5 decades: Stable incidence rates for all age groups in the population but rapidly changing age distribution in the clinic. *Mayo Clinic Proceedings* **85**(3): 225-230.

Uhl K, Cox E, Rogan R et al (2006) Thalidomide use in the US: Experience with pregnancy testing in the S.T.E.P.S.® programme. *Drug Safety* **29**(4): 321-329

Vlossak D, Fitch MI (2008) Multiple myeloma: the patient's perspective. *Canadian Oncology Nursing Journal* **18**(3): 141-151

Wiley KE (2007) Multiple myeloma and treatment-related thromboembolism: oncology nurses' role in prevention, assessment, and diagnosis. *Clinical journal of oncology nursing* **11**(6): 847-851

Zeldis JB, Knight RD, Jacques C, Tozer A, Bizzari JP (2010) Lenalidomide in multiple myeloma: Current role and future directions. *Expert Opinion on Pharmacotherapy* **11**(5): 829-842

Figure 1

Plasma cell development

Source: National Cancer Institute

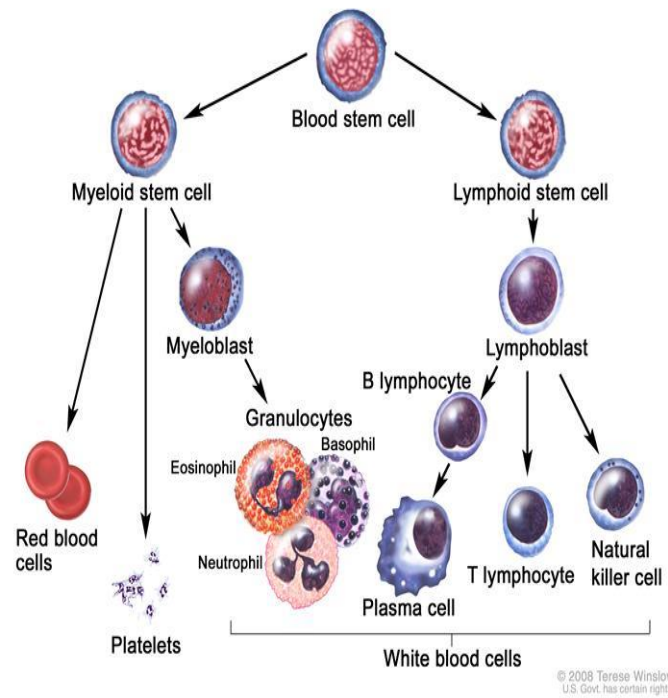


Figure 2

Immunoglobulin structure

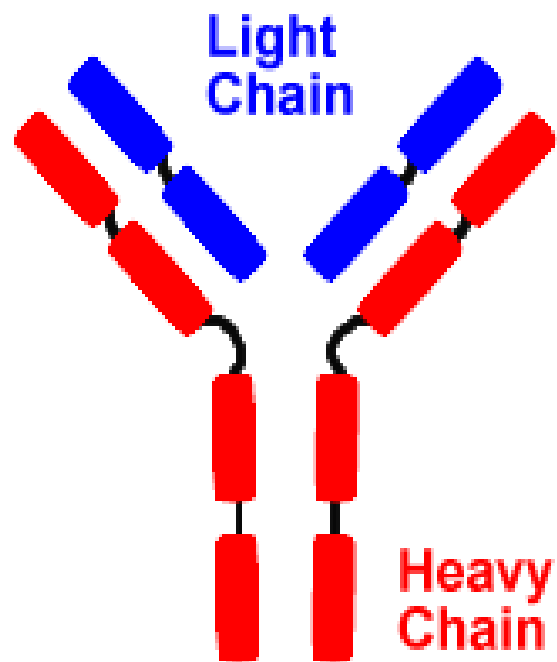


Figure 3

Myeloma cells (abnormal plasma cells) making M proteins. M proteins are antibodies created by a Myeloma cell.

Source: National Cancer Institute (NCI)

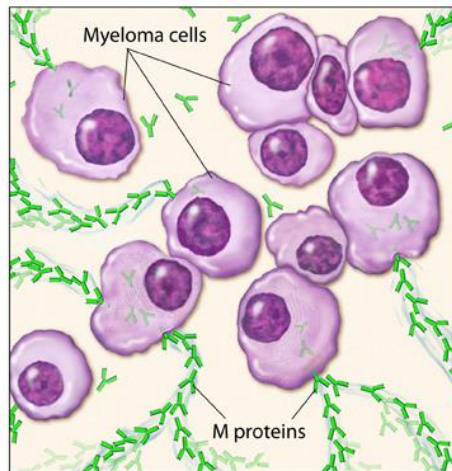


Table 1. Diagnostic criteria for MGUS, asymptomatic myeloma and symptomatic myeloma (adapted from the International Myeloma Working Group 2003).

MGUS	Asymptomatic myeloma	Symptomatic myeloma
<ul style="list-style-type: none"> • M-protein in serum <30 g/l 	<ul style="list-style-type: none"> • M-protein in serum >30 g/l and/or Bone marrow clonal plasma cells >10% 	<ul style="list-style-type: none"> • M-protein in serum and/or urine
<ul style="list-style-type: none"> • Bone marrow clonal plasma cells <10% and low level of plasma cell infiltration in a trephine biopsy (if done) 		<ul style="list-style-type: none"> • Bone marrow (clonal) plasma cells or biopsy proven plasmacytoma
<ul style="list-style-type: none"> • No ROTI (including bone lesions) • No evidence of other B-cell disease or light-chain amyloidosis or other light-chain, heavy-chain or immunoglobulin-associated tissue damage 	<ul style="list-style-type: none"> • No myeloma-related organ or tissue impairment (including bone lesions) or symptoms 	<ul style="list-style-type: none"> • Any myeloma-related organ or tissue impairment (including bone lesions)