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Editorial

Editor - Vicki Evans

Concussion in Sport has taken on a life of it's own following investigation of a series of increased episodes of depression and suicide amongst professional football players in the USA's NFL. These players are being diagnosed with dementia following years of repetitive concussions and improper respect of this traumatic brain injury (TBI).

These investigations are having an impact around the globe—looking at elite sports as well as school sports, where children and their developing brain are the targets of an increased public awareness program highlighting the dangers of concussion and new management strategies for this TBI.

March 2013, saw the first-ever Concussion in Sport Conference held in Australia at Melbourne's Etihad Stadium. Australia's four major football codes were well represented at this event, co-sponsored by the AFL and NRL with the ARU and FFA participating as well. The Players' Association and Referees were also represented. It was pleasing to be involved with this Conference, where under the umbrella of all the football codes, people gathered to discuss the way forward to minimise the risk and manage concussion in elite sport.

Long term effects of concussion were discussed, including dementia and chronic traumatic encephalopathy (CTE); putting concussion research into practice; priorities of concussion research into the future; implementation of outcomes from the 2012 Zurich International Conference on Concussion in Sport.
Issues discussed were:

1. Laws and penalty changes to protect the head and neck.
2. Revised guidelines including a more conservative management of concussion—in line with international best practice.
3. Educational awareness-raising amongst community and school level competitions.
4. Building knowledge by working together with concussion experts and through long-term research projects.
5. Introduction of the Pocket Concussion Recognition Tool and SCAT3.

Due to the media saturation of this type of injury, the schooling system has initiated discussions on how to protect growing brains. Hopefully with this heightened awareness, issues surrounding second impact syndrome should eventually be a thing of the past and concussion and its treatment, will have gained the respect that it deserves.

Once the guidelines are implemented in elite sport, the flow-on effect down to school sport and grass-roots level will be more effective.

From this conference, the **Pocket Concussion Recognition Tool** was introduced as a means to help identify concussion in children, youth and adults. This is a pocket tool for the sideline coaches to use. The SCAT3 was also discussed as a means to identify the severity of injury for use by medically trained staff. They can be downloaded from

- **bjsm.bmj.com/content/47/5/267.full.pdf**
- **www.neurosurgery.net.au/concussion.html**

*Concussion: "*If In Doubt, Sit It Out"
"Recognise—Remove from Play—Refer"

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~ Cheers, *Vicki*

Apology ~ In the October 2012 edition of the AJoN, the manuscript by Bernice Appiah, "A pilot study of post discharge needs of people who had removal of a primary brain tumour", inadvertently omitted the second author—Dr Imke Fisher (Australian Catholic University, North Sydney). Apologies to both authors.

Stimulation: What's your technique?

Danielle Wheelwright and Stephanie Gilmore
St Vincent's Hospital, Sydney

Background

Sternal rub has been shown to cause marked bruising and skin breakdown and should be used with extreme caution (1). Similarly, supra orbital ridge pressure can cause harm to patients if the patient moves during the examination (2). The trapezius pinch appears to be the safest technique for healthcare workers to use.

Aim

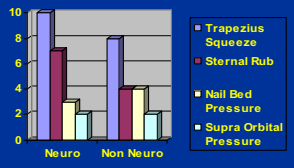
To compare knowledge and practice of nurses working in the neurosciences versus non neurosciences areas in regards to painful stimulation administered during the Glasgow Coma Scale assessment.

Method

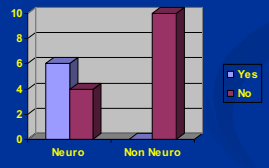
20 nurses- 10 working in the neuroscience specialty and 10 working in other clinical areas of the hospital were given a survey to complete. Questions asked were:

1. What types of painful stimuli do you commonly use as part of the GCS assessment?
2. Do you know the difference between central and peripheral painful stimuli?
3. Do you feel that St Vincent's policy and procedure on neurological assessment provides clear guidance of best techniques to use?

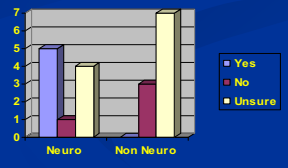
Results



What types of painful stimulation do you commonly use?



Do you know the difference between central and peripheral stimulation?



Do you feel that SVH policy and procedure provide clear guidance of best technique to use?

Discussion/Conclusion

When conducting the GCS assessment, a high percentage of both neuroscience nurses and non neuroscience nurses are still using techniques of painful stimulation that are not in line with current evidence based practice. Nurses need to be educated about the difference between central and peripheral painful stimulation and its use in the GCS. It is clear that there needs to be more education and clearer guidelines surrounding this in all areas of the hospital.

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St Vincent's Hospital

The Volunteer Feeding Program in patients prescribed a minced diet on the Neurosurgical Unit of Royal North Shore Hospital, Sydney.

Ella J Murray, Kirilee M Matters, Annie L Dent, Nicola A Pereira

Abstract

Malnutrition is a serious problem in hospitals. Up to 40% of patients are estimated to suffer malnutrition on hospital admission and many experience decline in nutritional status during their hospital stay. Causes of malnutrition may include poor appetite, disinterest in food, feeding difficulties, dysphagia and lack of feeding assistance. Malnutrition can result in dehydration, increased risk of infection, delayed wound healing and reduced quality of life. Subsequently clinical outcome is affected leading to extended length of stay and increased costs to the healthcare system.
The Volunteer Feeding Program was developed for the neurosurgical ward at Royal North Shore Hospital, Sydney in 2007. Volunteers were trained to assist with meal setup and feeding to improve oral intake and optimise nutritional status. The program was implemented in patients on a full diet that were identified as being safe for feeding. Evaluation has shown positive outcomes from the program and it has received executive endorsement. Dysphagia is common amongst the neurosurgical population and may be treated with texture modified diets such as minced diets. Patients on minced diets often need specific strategies and supervision at mealtimes to facilitate safe and adequate oral intake. This paper illustrates one way to safely and effectively extend volunteer feeding to patients on minced diets using a small pilot study in the neurosurgical population. Initial results show volunteer assistance improves energy intake by up to 189% and reduces waiting times significantly. Meal consumption improved from 13% to 75% of patients consuming all of their meal with volunteer assistance.

Key Words: *Malnutrition, Volunteer, Assisted feeding, Dysphagia, Minced moist, Neurosurgery.*

Introduction

With estimates indicating up to 40% of all patients admitted to hospital are already suffering from malnutrition and a possible 60% are 'at risk' of becoming malnourished it is clear that malnutrition in hospitals is a serious and widespread problem (Walton, Williams et al. 2008). Many patients will experience a decrease in oral intake during their admission secondary to poor appetite, disinterest in food, lack of variety, poor dentition, difficulty utilising cutlery and accessing food, difficulty with excessive food packaging, lack of feeding assistance and encouragement, difficulties chewing or swallowing, gastrointestinal upsets, malabsorption, depression or dementia which can lead to continued decline in nutritional status (Chima, Barco et al. 1997; Kowanko 1997; Kowanko, Simon et al. 1999; Hall, Whiting et al. 2000; Hickson 2006; Walton, Williams et al. 2008). Decline in nutritional status has been shown to cause a range of serious consequences including dehydration, increased risk of infection, delayed wound healing, weakened respiratory system, decreased mobility, reduced quality of life and depression leading to significantly increased costs to the healthcare system due to prolonged hospital length of stay, bed block and increased rates of readmission (Jordan, Snow et al. 2003; Stratton, Hackston et al. 2004; Kyle, Genton et al. 2005; Adams, Bowie et al. 2008).

Royal North Shore Hospital (RNSH) is 550 bed hospital located on the lower north shore of Sydney. RNSH provides level 6 tertiary neurosurgery services and facilities include a dedicated neurosurgery ward supported by neurosur-

Questions or comments about this article should be directed to Kirilee Matters, Senior Clinical Dietitian, Royal North Shore Hospital, Sydney at KMatters@nsccahs.health.nsw.gov.au

gical ICU. The neurosurgical trauma referral for the hospital includes the catchment area of NSW's North Coast (Northern Sydney Local Health District, 2012). The neurosurgical unit is a 26 bed ward which includes four step-down beds. Stroke, traumatic brain injury, brain tumours and neurovascular disorders are among the most common patient diagnoses. In 2007 a project known as the Volunteer Feeding Program was developed for the neurosurgical ward at RNSH. Volunteers were recruited and trained to assist with meal setup and feeding to improve oral intake and optimise nutritional status. The program was implemented in patients on a full diet that were identified as being safe for feeding by the nursing and allied health teams. On initial evaluation, adequate provision of feeding assistance increased from 75% to 91% and the number of patients consuming greater than 3/4 of their meals increased from 55% to 76% with volunteer assistance. In 2010 a follow-up evaluation was conducted which continued to show positive outcomes with a total of 1171 hours of volunteer assisted feeding and volunteers available at the majority of meal services 7 days a week. The percentage of patients waiting longer than 10-15 minutes for feeding assistance reduced from 27% to 8% and meal consumption improved from 27% to 67% of patients consuming more than 3/4 of their meals with volunteer assistance. Since its development and implementation, the program has achieved great success and has won several awards including the NSW Volunteer Service of the year 2011. It has executive endorsement and is a key initiative for the local health district. The program is a multidisciplinary partnership with nursing, nutrition and speech pathology (Kleiner and Friedman 2011).

The neurosurgical population experience a broad range of neurological diagnoses impeding their capacity to eat and often putting them at higher risk of malnutrition. This may include incoordination, muscle weakness, visual, cognitive and behavioural disturbances (Holmes 2006) and dysphagia (Mackay, Morgan et al. 1999; Ward, Green et al. 2007; Hansen, Engberg et al. 2008; Takahata, Tsutsumi et al. 2011) Dysphagia can contribute to an extended hospital stay or death due to dehydration, aspiration pneumonia, malnutrition or permanent disability impacting the clinical outcome (Mackay, Morgan et al. 1999; Mann, Hankey et al. 2000; Paciaroni, Mazzotta et al. 2004). Dysphagia can be treated with texture modified diets such as minced diets which can lead to further reduced oral intake increasing the risk of malnutrition. Indications for a minced diet include swallowing difficulties, poor dentition and painful mouth. Minced moist foods may be naturally soft (e.g. ripe banana), or cooked or minced to alter texture. Patients use the tongue, rather than teeth, to break the small lumps in this texture . Texture modified diets are nutritionally adequate for all nutrients except dietary fibre but intake of these diets tend to be suboptimal and therefore need close monitoring (Agency of clinical Innovation, 2011). Additionally, nutritional requirements are estimated to be higher in patients suffering from brain injuries as tissue needs to be repaired, increasing the likelihood of the diet being inadequate to meet the patients nutritional needs (Kleiner and Friedman 2011). Decline in nutritional status may exacerbate the extent of dysphagia therefore increasing the risk of morbidity and mortality (Veldee and Peth 1992).

A study conducted by Siebins et al. (1986) found that patients who required assistance at meal times also had a higher prevalence of swallowing difficulties suggesting many of the people who would benefit from the volunteer feeding program were not identified as suitable for inclusion. Demands on hospital nursing staff are high with mealtimes being particularly busy. A study conducted by Xia and McCutcheon (2006) found that during mealtimes nurses were busy with documentation and medication, rather than assisting with eating. Kowanko et al. (1999) reported that pressure of time and perceived urgency of other tasks were major reasons for nurses not enjoying the feeding of patients. Nurses estimated that it takes half an hour to feed some patients so when several patients need assistance it causes significant problems with time management. Volunteer assistance with feeding may be an appropriate solution to the problem with feeding being the volunteer's sole priori-

ty and adequate time able to be spent with each individual patient. Patients on modified diets including minced diets often need specific strategies and supervision at meal times to facilitate safe and adequate oral intake and reduce the risk of aspiration suggesting that comprehensive training must be given to volunteers to enable them to safely feed patients on minced diets.

Limited literature was found demonstrating that volunteer feeding programs have been carried out involving neurosurgical patients on minced diets; as such this project is an innovative and flexible way to improve nutrition in this patient group. The results of this study will contribute to the research base and improve quality of life of neurosurgical patients on minced diets.

Aim

To educate volunteers enabling the extension of the Volunteer Feeding Program to neurosurgical patients prescribed minced diets. The objective is to optimise nutritional intake whilst maintaining safe feeding and swallowing practices.

Literature review

A literature search was conducted on malnutrition in patients with dysphagia and strategies for improvement (subtopics including, but not limited to; malnutrition, dysphagia, minced diet, volunteer, assisted feeding, risk factors/ indicators for aspiration in neurologically impaired patients). The reference lists of several similar articles were also reviewed to find relevant articles. The literature review identified possible issues associated with feeding patients with dysphagia, higher risk of malnutrition in patients with dysphagia and the detrimental effects malnutrition can have on this patient group. The literature review further identified similar programs that have been implemented in elderly populations and their strengths and weaknesses. Results of this literature review are critiqued in the discussion section of this paper.

Methods

Baseline data collection

A baseline quantitative study was conducted in July 2012, a convenience sample of eight inpatients from the neurosurgery ward at RNSH was used and data was collected using an assisted feeding survey that was modified from the survey used by Kleiner and Freedman (2011). This work was deemed a quality project by the Human Research Ethics Committee and therefore did not require ethical approval.

Inclusion criteria for the study included all patients on a minced moist diet as prescribed by the speech pathologist that were considered safe to be fed by a volunteer and verbal consent to be part of the study, if the participant was unable to consent the next of kin was asked for consent.

Participants were excluded if they were on thickened fluids and/or puree diet as only registered nursing staff are able to feed these patients. Patients with significant behavioural difficulties or who had a code black in the past 48 hours were also excluded to ensure volunteer safety. Code black is defined as a personal threat (armed or unarmed persons threatening injury to others or themselves). The survey included questions relating to proportion of patients requiring assistance, level of assistance required (identified using a descriptive numerical scale), persons providing assistance, length of waiting times for feeding assistance, amount of time to finish the meal, amount of the meal consumed and the nutritional analysis of the diet. This data was collected by the researchers and intake information was collected using standardised food record charts endorsed by the local health district and tray tickets. Intake was analysed using recently updated ready reckoners provided by RNSH Department of Nutrition Services.

Ready reckoners are a condensed version of a nutrition panel and they list key nutrients and values.

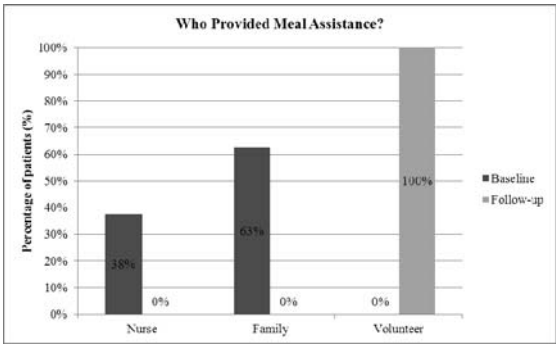

Figure 1. *Who provided meal assitance (%) before and after volunteer intervention.*

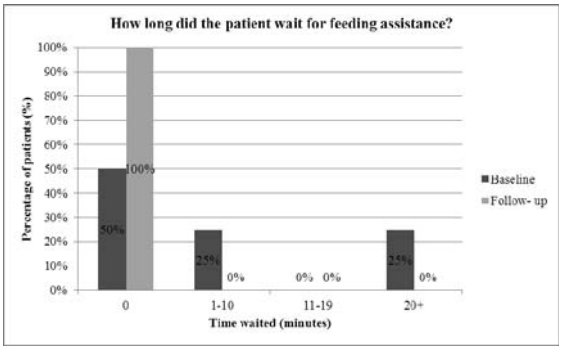

Figure 2. *Amount of time patients waited for feeding assistance before and after intervention.*

Training package

Following collection of baseline data a training program was developed and implemented for three established volunteers. The three volunteers are all long standing volunteers on the Volunteer Feeding Program at RNSH; they have established themselves, proved to be diligent on the ward, excellent with the volunteer feeding procedures, documentation and motivated to receive extended roles on the ward. The training tool was adapted from Greenslopes Private Hospital's 'Patient feeding self-directed learning package and competency'. The training was conducted with the assistance of the clinical nurse educator, dietitian and speech pathologist and aimed to extend the volunteers current scope of practice. Training included information relating to dysphagia, possible complications when feeding, consequences of dysphagia, identifying swallowing difficulties, management of dysphagia, the importance of nutrition in patients with brain injuries and encouraging high energy and high protein foods. Duties were outlined and the volunteers were made aware of when they must call for nursing assistance (e.g. patient positioning/ inserting dentures/ signs of aspiration). A volunteer folder was also developed containing a flowchart of this new process, referral sheets and a volunteer sign in form. Within this folder volunteers receive all relevant information on referred patients from the speech pathologist and have room to provide feedback. This was kept separate from the volunteer folder for patients on a full diet to avoid confusion and prevent volunteers who have not received specialised training from feeding patients on minced diets.

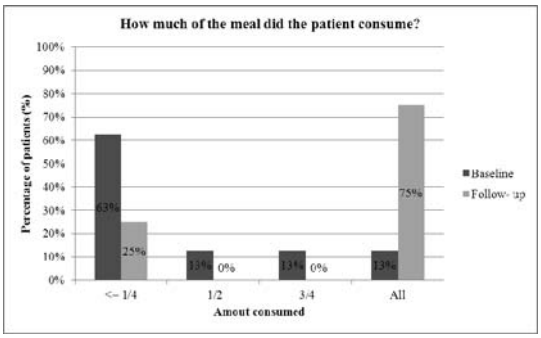

Figure 3. *Amount of meal consumed by patients before and after intervention.*

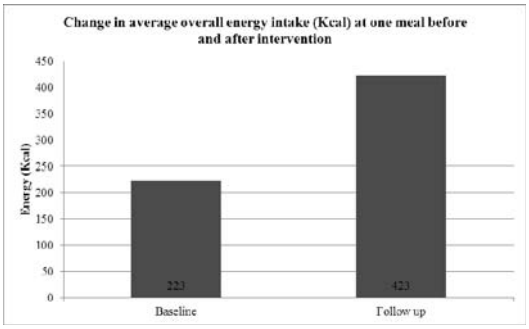

Figure 4. *Change in average overall energy intake (Kcal) at one eating occasion before and after intervention.*

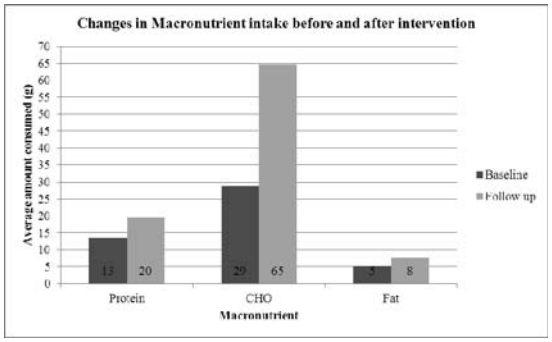

Figure 5. *Changes in average overall macronutrient intake before and after intervention.*

Competency

The volunteers were assessed while feeding a patient (who was prescribed a minced moist diet) by the speech pathologist. A competency document was designed and utilised for competency. Aspects such as obtaining all relevant information, awareness of patient positioning, seeking assistance where suitable, suitable bolus size, identifying signs of aspiration, communication with patient, completion of food chart and relevant feedback to appropriate member of staff were detailed in this document.

Intervention

Follow-up data was collected on days when both a trained volunteer (Wednesday and Friday) and a suitable patient on a minced diet were available. Proportion of patients requiring assistance, level of assistance required, persons providing assistance, length of waiting times for feeding assistance, amount of time to finish the meal and amount of the meal consumed were all recorded for the follow up patients. The amount of food con-

sumed was recorded using tray tickets and standardised food record charts endorsed by the local health district and was analysed using the same ready reckoners used at baseline.

Data Analysis
The data collected at baseline and follow up was analysed using Microsoft Excel 2007. The intakes were not compared to individual patient's nutritional requirements but rather overall improvement in nutrient intake was looked at. Energy, protein, fat and carbohydrate were focused on as full data was available for these nutrients while some data was missing from the ready reckoners for sodium, potassium and fibre. Comparisons were made between pre and post intervention results.

Staff Education
An inservice presentation was prepared and two sessions were run educating nursing staff on the existing volunteer feeding program, changes to the program, what this meant for nursing staff and the process' required to ensure safe operation. The referral process for patients was discussed and nursing staff were made aware that not all patients on minced diets are suitable for feeding and not all volunteers are suitable to feed these patients. They were made aware that only the speech pathologist could refer a patient and that those patients who had a code black in the last 48 hours were not to be fed by a volunteer.

Safety Flow chart
A flow chart was developed outlining safe feeding procedure to reinforce the new methods implemented in the extended Volunteer Feeding Program. This aimed to communicate best practice and reduce risk associated with the program.

Results
All volunteers passed the competency for feeding following the training provided and no adverse events were reported throughout the pilot period. Both groups were matched for level of assistance required (Average=2.75).

Before volunteers were trained to safely feed patients on minced diets the majority of patients (63%) were fed by family members and the remaining patients were fed by nursing staff (38%). Following the implementation of the pilot 100% of patients on minced diets deemed appropriate by speech pathologist were fed by trained volunteers.

Before the implementation of volunteer feeding 50% of patients had to wait for feeding assistance with 25% waiting for 1-10 minutes and 25% waiting for 20 minutes or more. One of these patients waited 20 minutes due to nursing staff taking observations and the patient experiencing high levels of pain and discomfort which were attended to. The other patient had no attention in the 20 minutes from when the meal tray had arrived and attention was brought to the nursing staff by the observing researcher at 20 minutes at which point they attended to the patient. Following intervention 100% of the patients were fed immediately when the meal arrived.
Figure 3. Amount of meal consumed by patients before and after intervention.

At baseline the majority (63%) of the patients were consuming one quarter or less of their meal with only 13% managing to eat all of the meal. Following the implementation of volunteer assistance this improved to just 25% of patients consuming one quarter or less of the meal and 75% of patients consuming all of the meal.

Overall energy intake of patients improved by 189% after the introduction of the volunteer feeding program to patients on minced diets.

Protein intake slightly improved in patients following implementation of the volunteer feeding with average protein intake increasing from 13g at baseline to 20g at follow up. Carbohydrate intake significantly increased with patients consuming an average of 29g at baseline and 65g at follow up. Fat intake remained quite stable with a modest increase of 3g post intervention.

Discussion
The pilot study was able to demonstrate good outcomes in overall energy consumption with a 189% increase after volunteer feeding had been implemented. Informal volunteer feedback was positive with all volunteers reporting that they enjoyed the added responsibility of feeding patients on a minced diet. The volunteers said that the training had prepared them well for feeding and all volunteers commented that they had enjoyed learning more about swallowing disorders and nutrition in neurosurgery. At baseline 63% of patients were being fed by family. While family feeding promotes social interaction, patients on minced diets may require extra care during feeding to minimise risks, particularly aspiration risk. Post intervention all patients on minced diets, considered suitable by speech pathology

were being fed by volunteers. This combines the social aspect of feeding while ensuring risk is minimised. Volunteers include family when they are present and encourage them to stay at meal times while they are feeding to help create a relaxed social and enriching environment for the patient. Reduced patient waiting times in the follow up may have contributed to improved intakes as all patients were fed immediately and therefore the meals would have been closer to the desired temperature and more appetising for the patients.

The results of this pilot are consistent with those found by Musson et al. (1990) who performed a quality assurance problem-focused study on the 'Silver Spoons' program implemented in Miami, USA. The program uses volunteers to provide eating assistance to patients with feeding and swallowing difficulties in a Nursing Home Care Unit (Musson, Kincaid et al. 1990; Musson, Frye et al. 1997). Volunteers undergo an orientation session involving presentations from the associate chief nurse of extended care, the dysphagia team speech pathologist, the dietitian and the 'Silver Spoons' volunteer coordinator. They were provided with practical training which includes proper positioning, feeding rate, volume of food to be fed, recognition of signs of aspiration, maintenance of an appropriate environment and verbal and physical cueing techniques. The nursing home also implements a second seating in the dining room for people with feeding and swallowing difficulties and 'Happy hour' where residents gather daily to partake in social activities such as singing or bingo as well as eating and drinking together. The results showed that when residents were included in all three initiatives from the initial month of data collection they gained an average of 1.9 kg over 3 months while those patients not participating in any of the initiatives lost an average of 0.6 kg over 3 months indicating that volunteer feeding along with cresting a social environment for feeding reduced the risk of malnutrition.

A 333 bed community hospital located in Sydney piloted a similar study in a 28 bed aged care ward in 2005 (Walton, Williams et al. 2008). The hospital had 25 trained volunteers who were available at lunchtimes on weekdays with around 8-10 patients referred daily. Patients were referred by the clinical care coordinator or the Nurse Unit Manager if they require feeding assistance, encouragement, social assistance or assistance opening packaging. Each volunteer had approximately 45 minutes to help 2-3 patients with their lunch. Patient's levels of independence varied greatly with some requiring just encouragement and others needing full assistance. Volunteers were educated to encourage high energy, high protein foods first and are aware of their role and when they must call for assistance. Evaluation of the program in August 2006 used a convenience sample of nine elderly patients. It involved two weekdays (Thursday and Friday) and the following Saturday and Sunday for each patient. Researchers observed volunteers (during lunch on weekdays only), patients and staff at each main meal, leftover food was weighed and demographic details for each patient were recorded. Patients were asked about their mid meal intakes and appetite. Data on diet type, age, reason for admission, weight and height (if available) were collected. Meal orders from the tray ticket were recorded. Each patient's estimated daily requirements for protein and energy was calculated. Observational data was collected at each main meal and focused on when and how the food was served, the time before patients started to eat, the time patients took to eat, the assistance provided, any socialisation aspects and any interruptions during mealtimes. The evaluation was able to conclude that a volunteer feeding assistance program can improve protein intakes in longer stay, aged care hospital patients. They also found higher intakes of energy at lunch when volunteers were present which is in line with the findings from this pilot study (Walton, Williams et al. 2008).

A study conducted by Wright et al. (2008) demonstrated volunteer feeding in patients with dysphagia in Charing Cross Hospital, London between August and December 2005. This study considered all patients over 65 years with diagnosed dysphagia for intervention. Only dysphagic subjects prescribed a texture modified diet, and/or thickened fluids were included. Exclusion criteria included if their family or carers were available to provide assistance at mealtimes; if they were being totally or partly fed via a tube; or if they were for palliative care only. Before assisting patients, the volunteers attended a one week training program provided by a dietitian, speech pathologist and nurse. Food intake data for the intervention group was collected using food charts which were completed by the volunteers. Each patient was assisted for three days including breakfast, lunch, snacks and supplements. There were no apparent problems, such as signs of aspiration with the volunteers feeding the intervention group.

The findings of the study indicated that food intake at meals and nutritional supplements both independently increased with feeding assistance to older patients with dysphagia in hospital. Dysphagic patients with targeted feeding assistance had a higher consumption of energy and protein than those without individualized feeding assistance and no problems with safe feeding were reported. These findings are indistinguishable from the results of this study.

Limitations

This study had many limitations, in particular the small sample size used in the pilot. This was due to the inconsistency in numbers of patients requiring a minced diet at any one time and that it is a relatively small patient group we are working with. The small number of trained volunteers also meant that volunteer assistance for patients on a minced moist diet was only available on Wednesdays and Fridays so any patients on a minced diet on other days of the week could not be included in the follow up data. Only four patients met all the inclusion criteria on Wednesdays or Fridays during the follow up data collection period so fewer patients were included in follow up. Eight patients were included in the baseline data collection. Care was taken during data collection to ensure similar patient types were used in order to draw reasonable conclusions, while level of assistance required was matched for each group (average level of assistance = 2.75 in both groups). Patient diagnoses and detailed patient statistics were not recorded. No age, gender, reason for admission, weight or height data was recorded for the patients in this study for practical reasons; therefore variation between the baseline and follow up group statistics could confound the results. Due to this data not being collected no nutritional requirements were calculated for the patients. Patient oral intakes were not compared to requirements but rather analysed for overall increase in nutritional intake between baseline and follow up groups. Therefore one cannot conclude if the follow up group was closer to meeting individual nutritional requirements than the baseline group. In addition only the lunch meal was observed so it is unclear if improved intakes at lunchtime are associated with smaller intakes at breakfast or dinner therefore negating the benefit. A study conducted by Walton et al. (2008) was able to show that patients were not eating significantly less energy and protein at breakfast or dinner in response to higher lunch intakes when volunteer feeding was implemented. This indicates that improved lunch intakes may improve intake across the whole day. The nature of neurosurgical patients also made the study difficult with not all patients suitable for volunteer feeding secondary to the behavioural nature of the patients. Some patients display aggressive behaviour and others may behave inappropriately due to the area of the brain that has been affected. Many patients are in post traumatic amnesia and need to be assessed on a case to case basis for suitability for volunteer feeding. Safety of the patients and the volunteers is an important issue and all measures were implemented to ensure this was not compromised. This was an overt study and therefore behaviours and resultant intakes may have been influenced. Researchers remained as inconspicuous as possible in order to allow natural eating and feeding patterns to occur in an attempt to minimise this bias. The influence external factors such as number of visitors, medical condition and state of recovery had on oral intakes were not explored in this study and may have influenced results. This study was conducted across a period of 4 months and during this time participant numbers were limited due to leave issues. Those absences limited the number of days available to collect data. In order to draw clearer conclusions this study would need to be conducted in a larger sample group. Continued data collection would be beneficial.

Conclusion

This pilot study suggests that energy intake at mealtimes can be significantly improved and protein intakes can be modestly improved for neurosurgical patients on a minced diet as prescribed by a speech pathologist when feeding assistance is provided. Volunteer feeding assistants, once trained and with adequate support networks, can potentially provide safe and effective feeding assistance to patients on minced diets. Patients under the neurosurgical speciality with dysphagia have a high risk of aspiration and malnutrition. It is imperative that any volunteer feeding program developed for patients on modified diets is done with a multidisciplinary approach including cooperation from dietetic, speech pathology and nursing teams in order to minimise risk and provide coordinated and individualised programs for this at risk population. Patients on a minced diet were assessed by speech pathology pre referral for feeding by a volunteer. Patients on puree diets and/or thickened fluids were excluded from being fed by the trained volunteers because of the high risks associated with feeding these patients and

the special training that is required. Recommendations for further study include surveying nursing staff, volunteers and patients regarding the extension of the Volunteer Feeding Program to gain formal feedback as well as continuing data collection to get a larger sample size. This would be beneficial for this project in order to draw stronger conclusions and ensure quality control.

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Boswellia serrata as an alternative to Dexamethasone to treat peritumoural oedema

Kylie Wright

Abstract

One of the most common complications of brain tumour growth is peritumoral oedema. Such oedema and its inflammatory processes are major contributors to neurological symptoms and morbidity in brain tumour patients and the treatment of these phenomena has always been of high importance.

The first choice treatment of symptomatic cerebral oedema is the corticosteroid dexamethasone. Despite significant adverse side effects such as cushingoid habitus, immunosuppression, hyperglycaemia and proximal myopathy, this drug is widely used and considered a necessary evil in neuro-oncology management. There is evidence that dexamethasone influences cancer therapies through stabilisation of blood-brain and blood-tumour barriers and reduction of tumour perfusion (Kotsarini, Griffiths, Wilkinson & Hoggard, 2010) It has also been shown to interfere with the efficacy of chemotherapy by directly inhibiting apoptosis in malignant glioma cells (Smith, Simpson & Sekhon, 2005; Hou, Veeravagu, Hsu & Tse, 2006).

Boswellia serrata, a traditional herbal extract of the Indian frankincense tree, could be considered as a promising steroid sparing agent for the treatment of cerebral oedema with less adverse effects and additional induction of apoptosis (Kotsarini, et al., 2010). Boswellia serrata is virtually unknown however clinical research has shown its use may reduce cerebral oedema.

Throughout this paper peritumoral oedema and the indications for corticosteroid use will be explored. The challenging side effects of dexamethasone therapy will be highlighted through the use of a case study and the literature surrounding the effectiveness of Boswellia serrata and its potential use as a way to reduce steroid use in treating cerebral oedema will be reviewed.

Key Words: *Peritumoural oedema, dexamethasone, Boswellia serrata, Neuro-oncology*

Introduction

High grade brain tumours are associated with significant morbidity including cognitive, neurological, and behavioural symptoms affecting both the patient and their caregiver. One of the most common complications of brain tumour growth is peritumoral oedema. Such oedema and its inflammatory processes are major contributors to neurological symptoms and morbidity in brain tumour patients and the treatment of these phenomena has always been of high importance. The effective management of cerebral oedema is crucial to keeping the patient alive in order to treat the underlying ailment as it impacts directly on the survival of these patients.

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Peritumoral oedema

Gliomas account for more than 70% of all primary brain tumours. In this patient group, raised intracranial pressure (ICP) due to peritumoral oedema is a highly prevalent and critical clinical problem (Kotsarini, et al., 2010). The majority of patients with high grade glioma (HGG) will have symptoms of raised intracranial pressure at some point during their illness. In adult patients with supratentorial tumours, headache and signs of ICP at diagnosis occur in at least 19-34% of cases, often in conjunction with cognitive dysfunction and other focal neurological signs including hemiparesis (14-41%) and seizures (17-31%) (Smith, et al., 2005). These symptoms may persist, recur and worsen in severity (Hou, Veeravagu, Hsu & Tse, 2006; Osaba, Brada, Yung & Prados, 2000) due to the high likelihood of recurrence of the tumour and poor prognosis despite advances in adjuvant thera-

pies (Chamberlain, 2010). Peritumoral oedema can often be the cause of death in neuro-oncology patients.

The pathogenesis of peritumoral oedema is not fully understood but it is believed that oedema forms as a result of excess fluid build-up in the extravascular space surrounding the tumour and an inability of the brain to clear this fluid due to a deficit in the blood brain barrier (BBB). The BBB within a brain tumour and in the vessels surrounding the brain tumour is disrupted due to the loss of tight junctions between endothelial cells, increased pinocytosis in the endothelium and an increase in endothelial fenestrations. The increased capillary permeability is also mediated by the release of vasoactive cytokines and mediators of tumour associated angiogenesis. (Sarin & Murthy, 2003; Kaal & Vecht, 2004; Australian Cancer Network Adult Brain Tumour Guidelines Working Party, 2009; Hildebrand, 2003)

Peritumoral oedema is vasogenic, the BBB is disrupted, protein (serum) leaks out of the vascular system and enhances on imaging. The accumulation of fluid mainly occurs within the brain parenchyma (the white matter) causing an increase in intracranial pressure and neurological dysfunction, presumably because of ischaemia from the cerebral mass effect. Associated symptoms include headache, nausea and vomiting, and a decreased level of consciousness.

Patients with symptomatic cerebral oedema will almost always be considered for corticosteroid treatment. Indications for such treatment include progressive neurological deficits and symptoms of raised ICP (Sarin & Murthy, 2003). Treatment is not usually indicated if patients are asymptomatic with oedema identified on imaging only.

Dexamethasone

No randomised trials have compared different corticosteroid agents in patients with brain tumours. However, the first choice treatment and routine management of symptomatic cerebral oedema is the administration of the corticosteroid 'Dexamethasone' (Kotsarini, et al., 2010; Australian Cancer Network Adult Brain Tumour Guidelines Working Party, 2009). Dexamethasone is generally the corticosteroid of choice, due to association with less salt retention and less inhibition of leucocyte migration (Sarin & Murthy, 2003). Dexamethasone has less mineralcorticoid activity and high glucocorticoid potency compared to other corticosteroids (Hildebrand,2003; Wen, Schiff, Kesari, Drappatz, Gigas & Doherty,2006).

The vast majority of patients with high grade brain tumours are prescribed Dexamethasone at several points along their disease journey from diagnosis, during the perioperative period, during adjuvant radio/chemotherapy, and at tumour recurrence or progression, with many patients needing to remain on Dexamethasone continuously (Kotsarini, et al., 2010) and indefinitely.

It is thought corticosteroids reduce cerebral oedema by stabilising the disrupted blood brain barrier and neurological recovery has been related to reversal of microscopic features of cerebral oedema, axonal disruption and myelin disruption (Weissman, Dufer, Vogel & Abeloff,1987; Van Roost, Hartmann & Quade, 2001; a) Long, Hartmann & French, 1966; b) Long, Hartmann & French,1966; c) Long, Hartmann & French,1966; Weinstein, Toy, Jaffe & Goldberg,1973; Chumas, Condon, Oluoch-Olunya, Griffiths, Hadley & Teasdale,1997).

The benefit of corticosteroids is generally accepted but there are gaps in the scientific literature. The optimum dose of Dexamethasone is unknown, as there have been no randomised trials comparing different doses of Dexamethasone in patients with malignant glioma. Clinically, the usual starting dose is 16mg per day (Wen, et al., 2006). Furthermore, treatment with corticosteroids has not been evaluated in a randomised clinical trial and the absolute efficacy cited in the literature of the ability of corticosteroids to reduce raised intracranial pressure is highly variable (Kotsarini, et al., 2010; Sarin & Murthy, 2003). The proportion of patients with partial or complete symptom relief has been reported to range between 33 – 80% (Sarin & Murthy, 2003). These results come from patient populations that were undergoing or recently completed cranial irradiation with difficulty determining response from steroids versus radiation (or contribution of radiation to worsening oedema) and in patients with brain metastases (Sarin & Murthy, 2003). The neurological response also depends on the nature, severity and chronicity of symptomswith recent focal deficits due to vasogenic oedema responding better (Sarin & Murthy, 2003). Further to this, a dose-response effect of corticosteroids has not been clearly established

in relation to neurological improvement (Sarin & Murthy, 2003).

Clinical challenges of dexamethasone use
Dexamethasone is widely used and currently the only available ongoing therapy to treat raised ICP secondary to peritumoral oedema but causes significant morbidity of its own. Sometimes dexamethasone toxicity is unavoidable as recommencement or increased dosing of corticosteroids is often the only strategy available to maintain neurological and cognitive function. Despite the potential benefits of corticosteroids the associated adverse events can be severe and add to the tumour and treatment related morbidity (Sarin & Murthy, 2003). Ironically, the steroids themselves can worsen the exact problems they aim to reduce. The prolonged use of steroids must be balanced against the potential side effects.

Significant adverse side effects include cushingoid habitus including a moon face and weight gain, muscle wasting and proximal myopathy (severe enough for some people to be bed bound) and psychological disturbances such as restlessness, agitation, anxiety, sleep disturbance and personality or mood changes including suicidal thoughts, profound euphoria or psychosis. Furthermore hyperglycaemia , peripheral oedema, gastrointestinal toxicity, immunosuppression causing increased susceptibility to infections, skin changes such as acne, bruising and poor wound healing and osteoporosis are all adverse effects of corticosteroids.

Studies of brain metastasis treated with dexamethasone report cushingoid features present in 32% - 69% of patients depending on the dose (Vecht, Hovestadt, Verbiest, van Vliet & van Putten, 1994). Most patients treated with conventional doses of steroids (for example 16mg / day for more than 2-3 weeks) develop some degree of myopathy (Batchelor & Byrne, 2006) with rates of 10 – 60% reported for patients on corticosteroids for more than three weeks (Sarin & Murthy, 2003; Vecht, et al., 1994). The incidence of severe psychiatric illness is uncommon at low doses but increases to almost 20% for patients treated with more than 12mg/day of dexamathasone (Brown & Chandler, 2001). Steroid induced hyperglycaemia is reported in 18 – 25% of patients on corticosteroids (Sarin & Murthy, 2003; Vecht, et al., 1994) with approximately one-half of patients treated with steroids over a prolonged period developing disturbed glucose metabolism that may persist following withdrawal of the drug (Meyer & Badenhoop, 2003).

The longer-term toxicity of corticosteroids when used for weeks to months in patients with high grade tumours has not been formally documented in the published literature and in particular not from the patient and caregivers perspective. However clinical experience and patient/caregiver feedback suggests the adverse events can be multiple, protracted and severe, especially in the setting of prolonged dexamethasone use. In particular, patients and caregivers report significant impact on function and need for care from myopathy and weight gain which significantly hinders mobility and the ability to care for patients at home. In addition, personality or mood change (ranging from depression, mood swings, hypomania) and changes in physical body image due to cushingoid side effects leads to psychological distress. Furthermore insomnia and restlessness leads to lack of sleep which causes fatigue in both patients and caregivers.

Further challenging aspects of dexamethasone treatment is that of 'tapering' the drug dose which needs to be scheduled with extreme care to avoid clinical deterioration and corticosteroid withdrawal syndrome. Further to this it is also thought that steroid drugs may protect brain tumour cells and influence cancer therapies through stabilisation of blood brain barriers and reduction of tumour perfusion, (Meyer & Badenhoop, 2003). They influence vascular response to radiation (Glaser, Winter, Groscurth, Safayhi, Sailer, Ammon, Schabet & Weller, 1999) and directly inhibit apoptosis in human malignant glioma cells (Weller, Schmidt, Roth & Dichgans, 1997; Gorman, Hirt, Orrenius & Ceccatelli, 2000).

Case Study
The complexities of long term dexamethasone use can be highlighted through the following neuro-oncology case study. The patient was a 55 year old male who presented with two week history of dizziness, decreased mobility and a mild hemiparesis. A CT scan showed a left parietal enhancing mass with vasogenic oedema and mass effect. The patient was started on the routine 4mg QID dexamethasone and hence his neuro-oncology journey started. After surgery the tumour was histologically confirmed as a Glioblastoma Mulitiforme (GBM).

The patient recovered well from the surgery but experienced ongoing headaches lasting more than half a day on most occasions. The dexamethasone dose was weaned but continued through concurrent radio and chemotherapy. One month post surgery the patient's wife reported he was experiencing mood swings and that he became irritable and short tempered with his family over minor things, which was completely out of character for him. Two weeks post radiotherapy attempts were made to wean the dexamethasone dose and the patient was managed on 2 mg dexamethasone daily with only minor headaches.

After his diagnosis the patient attended a session at his hospital's Brain Tumour Support Group entitled "Diet and Nutrition after a diagnosis of brain tumour". He attended alone without his family as he was ashamed of his weight gain and he was very self conscious about his body image. At four months after diagnosis he had gained 14kg in weight. After attending the support group he self referred to the hospital dietician regarding his increased appetite and weight gain and was notably distressed at the consultation, asking if he could take meal replacement shakes. Six months post diagnosis the patient had gained 20kg and this weight gain was his biggest concern. He had developed large fat deposits around his neck and a change in his facial appearance that was consistent with the typical cushingoid appearance. His daughter reported that he had refused to get into family photographs at Christmas time because of his change in appearance and associated embarrassment. He was 'extreme dieting' and had self weaned his Dexamethasone down to 1mg per day leading to a return of his headaches and decline in his neurological function.

Eight months after diagnosis the patient developed severe peripheral oedema. He was having difficulty walking and could not wear his usual footwear. He had no cardiac history and an electrocardiogram (ECG) was normal hence the oncologist determined the symptoms were related to the dexamethasone. Unsuccessful attempts were made again at this point to wean his dexamethasone dose with severe headaches returning as the dose was decreased.

Within a few weeks the patient was having difficulty walking up his front house steps and transferring with notable weakness in his legs. He was also developing symptoms of myopathy in his left hand.

Eleven months post diagnosis the patient underwent a second debulking surgery for recurrence of his tumour. This led to an increase in his dexamethasone dose back up to 4mg QID. Post surgery he underwent adjuvant chemotherapy. It was at this point where his blood glucose readings became as high at 21mmol/L and insulin was required.

Numerous more unsuccessful attempts were made to taper the dexamethsone dosages over the following months and to get the patient's blood glucose under control. He had a grand mal seizure 14 months post diagnosis, suffered bouts of hypertension and had electrolyte imbalances.

The patient's irritable behaviour was challenging for his family, his hyperglycaemia was difficult to manage and he suffered from peripheral myopathy. He suffered headaches most days and was very cushingoid with his weight increasing from 90kg – 119.5kg (29.9kg). His family were very aware of the effects of dexamethasone and asked regularly if there was a suitable alternative. At 16 months post diagnosis the patient died having never come off the dexamethasone his whole journey and suffering many adverse effects.

Despite dexamethasone having relatively poor efficacy and its associated toxicity, currently no accepted alternatives are available. There is a clinical need for an alternative agent to reduce ICP or to use as an adjuvant treatment to Dexamethasone to allow reduction in corticosteroid dose and duration of use.

Boswellia serrata, a traditional herbal extract, could be considered as a promising alternative to corticosteroids in the treatment of cerebral oedema with a better safety profile than corticosteroids and less adverse effects.

Boswellia serrate

Boswellia serrata is a traditional herbal extract of the Indian frankincense tree that grows abundantly in the dry hilly parts of India. The herb has been used for hundreds of years in traditional medicine with its major use in contemporary medicine as an anti-arthritic and anti-inflammatory pharmacological agent.

It is reported that Boswellia serrata can stop the production of certain substances called leukotrienes which help regulate the state of blood vessels and airways, and influence the activities of white blood cells. Leukotrienes seem to be produced in large amounts by gliomas and contribute to the production of peritumoral oedema in the brain (Hildebrand, 2003). It is for this reason that Boswellia serrata can be referred to in the literature as a lipoxygenase inhibitor. There is evidence that Boswellic acids can cross the blood brain barrier based on in-vivo animal studies (Kruger, Daneshfar, Eckert, Klein, Volmer, Bahr, Muller, Karas, Schubert-Zsilavecz & Abdel-Tawab, 2008) and it has been shown to have significant effects on the invasiveness of GBM cells in-vitro (Federation of American Societies for Experimental Biology, 2008).

Clinical trials have demonstrated promising benefits from Boswelic acids in rheumatoid arthritis, chronic colitis, ulcerative colitis, Crohn's disease, and bronchial asthma, in addition to benefits for brain tumour patients (Ammon, 2002). Burning Boswellia resin has also shown to have anti-depressive and anti-anxiety affects (Federation of American Societies for Experimental Biology, 2008).

Literature review

There is minimal published data about the effects of Boswellia serrata on brain oedema and brain tumours. There are two prospective clinical studies, a retrospective and a prospective case series, all involving patients with brain tumours or metastases, as well as a case report of a patient with brain metastases.

The most recent and promising clinical trial is a randomised, placebo controlled, double blind study involving 44 patients investigating the efficacy of Boswellia serrata on cerebral oedema in patients irradiated for brain tumours (Kirste, Treier, Wehrle, Becker, Abdel-Tawab, Gerbeth, Johannes Hug, Lubrich, Grosu & Momm, 2011). Patients receiving irradiation of the brain for primary brain tumours or brain metastasis of solid tumours were administered either Boswellia during radiotherapy or a placebo. In patients with brain metastases, a reduction in brain oedema (evaluated by MRI scans) of >75% was seen in 67% in the Boswellia group and 31% in the placebo group at the end of radiotherapy. This result was reported as statistically significant.

The tumour/oedema volume ratio decreased only in the Boswellia group, suggesting and anti-tumour effect in addition to the anti-oedema activity. Re-evaluation at 4 weeks after radiotherapy showed no differences between the Boswellia and placebo groups, which authors thought might be attributable to the termination of Boswellia intake at the end of radiotherapy. Common adverse events associated with radiotherapy were similar in both groups, although gastrointestinal discomfort was higher in the Boswellia group.

In another prospective clinical study, 29 glioma patients were non-randomly allocated to receive three different doses of Boswellia prior to surgical intervention (Heldt, Winking & Simmet, 1996). After seven days of intervention, the size of perifocal oedema was reduced by 33% in the CT scans of participants that consumed the highest dose of Boswellia, and to a lower degree the middle dose. Improvement in clinical symptoms were found in the group receiving the highest daily dose.

A further case series study evaluated the use of Boswellia retrospectively in 17 children and adolescents with different progressive or relapsed brain tumours (Janssen, Bode, Breu, Dohrn, Engelbrecht & Gobel, 2000). The Boswellia was administered as a palliative therapy for up to 26 months with or without concomitant conventional therapy. Six patients reported an improvement of their clinical condition and subjective relief of symptoms. In two of these patients, and in two additional patients with no subjective changes, regression of prior neurological symptoms like pareses and ataxia was reported. Regression of the peritumoral oedema in one case and a reduction of a tumour cyst in another case were reported by MRI. Taking Boswellia acids, four children with malignant brain tumours in progression remained in stable disease over 3-8 months. Another study included a prospective case series of 12 adult GBM patients with progressive cerebral oedema with or without tumour progression (Streffer, Blizler, and Schabet, Dichgans & Weller, 2001). Boswellic extract was administered for four weeks resulting in eight patients reporting a clinical improvement with two of these having a reduction in perifocal odema seen on MRI. This study concluded that Boswelllic acid could be a possible surrogate for corticosteroids in patients with mild to moderate (but not with severe) brain oedema.

In a case study report the favourable course of a 39 year old patient with breast cancer multiple metastases to the brain was described (Flavin, 2007). After 10 weeks of Boswellia treatment in combination with radiation and chemotherapy treatment the brain metastasis could no longer been seen on the CT scan. The patient was maintained on Boswellia serrata for another four years without signs of recurrent cerebral metastases but later developed bone metastases. The authors concluded that it was not possible to attribute the long term remission of her brain metastases to Boswellia extracts because both radiotherapy and chemotherapy treatment have reportedly induced remission of central nervous system (CNS) metastases and may have been the active treatment in this case. Nevertheless, a long term remission of multiple CNS metastases is rare and all interventions, including boswellia extracts deserve consideration in future investigations.

Further to these clinical trials and case series/ study reports, a systematic review looked at all the published data reported from randomised clinical trials about the effectiveness of Boswellia serrata as a treatment for any human medical condition (Ernst, 2008). The trials related to asthma, rheumatoid arthritis, chron's disease, osteoarthritis and collagenous colitis. Results of all trials indicated that Boswellia serrata extracts were clinically effective and no serious safety issues were noted.

Further research

Boswellia appears to be a possible supplement providing therapeutic benefits for several symptoms in the neuro-oncology patient population. The combination of an anti oedema effect with cytotoxic activity against the tumour, combined with Boswellia's extremely low toxicity warrants consideration for neuro-oncology patients. More research is required to support Boswellia's use as a steroid sparing agent.

The long term effects of Boswellia on humans are unknown. The dosages of Boswellia are a largely unexplored area. It is known relatively high doses are needed to be of therapeutic benefit and the product is not available intravenously. The serum levels of Boswellia are also influenced by the patient's diet. Higher serum levels and hence more therapeutic levels are evident on a high fat diet. Furthermore the supplement is difficult to obtain. The German brand of boswellia called H15, it is sold over the counter as a dietary supplement in Germany, for example, yet is reported to be difficult to obtain outside Europe.

Other therapies showing promise as corticosteroid-sparing agents include Acetazolamide and the synthetic coricotrophin releasing factor Corticorelin acetate with Phase III trials showing a 50% reduction in Dexamethasone use with associated stabilisation or improvement in neurological function.

Suggested areas for future research to address the problem of corticosteroid toxicity in neuro-oncology patients could include a longitudinal documentation of the real burden of corticosteroid toxicity from patients living with HGG and their caregivers and an evaluation of corticosteroid treatment. Furthermore a phase III trial to build on the findings by Kriste et al (2011) to consider the effect of Boswellia serrata on cerebral oedema with possible reduction of the necessary dexamethasone dose and anti-tumour effect as an endpoint. Furthermore research using of higher concentrations of Boswellia and additional measurements of quality of life may add to the body of knowledge on this issue.

Conclusion

A diagnosis of a high grade glioma brings sudden change in health, dependence on a caregiver, and the need to rapidly adapt to cognitive and functional changes.
Raised ICP due to peritumoral oedema is a highly prevalent and critical clinical problem in this patient group with associated symptoms of headache, nausea and vomiting, cognitive decline, hemiparesis, seizures and/ or a decreased level of consciousness. Any intervention which can minimise the degree of symptom burden can make a significant impact.

The corticosteroid dexamethasone is widely used and currently the only available ongoing therapy to treat raised ICP secondary to peritumoral oedema. Despite the potential benefits of corticosteroids, the associated adverse events can be severe and add to the tumour and treatment related morbidity. A safe and simple to administer alternative or supplementary treatment to corticosteroids in the management of peritumoral oedema would be welcomed by patients, caregivers and health professionals.

Clinical trials have demonstrated promising benefits from Boswelic acids in rheumatoid

arthritis, chronic colitis, ulcerative colitis, Crohn's disease, bronchial asthma, and brain tumours, and may be a possible supplement providing therapeutic benefits for several symptoms in the neuro-oncology patient population. More research is required to support Boswellia's use as a steroid sparing agent.

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Louie Blundell, was born in England, and although she wanted to be a nurse she had to wait until after World War II to start her training as a mature student in her late twenties. Later she and her family moved to Western Australia in 1959. She worked for a General Practice surgery in Perth until a move to the Eastern Goldfields in 1963. Subsequently, she worked at Southern Cross Hospital and then Meriden Hospital. During this time she undertook post basic education to maintain her currency of knowledge and practice, especially in coronary care.

Louie was also active in the community. She joined the Country Women's Association and over the years held branch, division and state executive positions until shortly before her death in 2007. She was especially involved in supporting the welfare of students at secondary school, serving on a high school hostel board for some time.

She felt strongly that education was important for women and was a strong supporter and advocate of the move of nursing education to the tertiary sector, of post graduate study in nursing and the development of nursing scholarship and research, strongly defending this view to others over the years.

For further details and criteria guidelines please visit the ANNA website at www.anna.asn.au

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Vasospasm in the neuroscience patient is not all it's cracked up to be!

Elizabeth O'Brien

Abstract

Cerebral vasospasm is usually thought to be associated with subarachnoid haemorrhage and aneurysms. A rare diagnosis, reversible cerebral vasoconstriction/ vasospasm syndrome is precipitated by thunderclap or severe headache, and fluctuating neurological deficits, it commonly affects women of child bearing age. There is no evidence of aneurysm rupture or subarachnoid haemorrhage on CT scanning however an MRA may indicate vasospasm. Precipitating factors have been attributed to postpartum state, exposure to vasoactive substances and possibly in combination with binge drinking. Treatments are varied but include the use of calcium channel blockers. This case presentation will follow the journey of a 50 year old female presenting with stroke and the unfolding of therapies and treatments normally reserved for aneurysmal cases.

Key Words: *Stroke, cerebral vasospasm, reversible cerebral vasospasm, neuroscience nursing, thunderclap headache.*

Introduction

The purpose of this article is to explore a rare condition of reversible cerebral vasospasm causing ischaemic stroke. To gain a complete appreciation of this condition it is important to revisit vasospasm in the Neurosciences patient. To enhance the understanding of this condition a case study will highlight a patient who does not fit in the usual 'box' of vasospasm. This article endeavours to provide an understanding of Reversible Cerebral Vasospasm, current treatments and its relevance to Neuroscience Nursing. It is also a means by which we need to remember that stroke is not always black and white!

Cerebral Vasospasm

Hickey (2009) notes that vasospasm occurs in approximately thirty percent of patients who present with aneurysmal subarachnoid haemorrhage (SAH) with fourteen to thirty six percent of patients suffering disability and death. Cerebral vasospasm can develop from three to fourteen days post SAH and can occur out to twenty one days. By definition cerebral vasospasm is narrowing of a cerebral blood vessel causing reduced blood flow distally, which then may lead to delayed ischaemic deficit and cerebral infarction if left untreated (Hickey 2009).

Questions or comments about this article should be directed to Elizabeth O'Brien, Clinical Nurse Consultant Early Supportive Program—Stroke at EObrien@nsccahs.health.nsw.gov.au

It is noted by Hickey (2009) that the aetiology of vasospasm is still hypothesis based. Hickey (2009) explains that spasmogenic agents are released as the clot breaks down; an endothelium relaxing factor such as nitric oxide is somehow inhibited from exerting its relaxing effect because of changes in the endothelial cells that produce nitric oxide. These cellular changes may be induced by the release of blood into the extra vascular compartment. There are two types of vasospasm - *angiographic vasospasm* with no clinical symptoms found incidentally on formal four vessel cerebral angiography and *symptomatic vasospasm*.

The signs and symptoms of cerebral vasospasm can include a gradual neurological deterioration; usually focal signs are demonstrated however confusion, decreased level of consciousness and coma are also seen. Deficits exhibited may be specific to a cerebral vascular territory or multifocal and diffuse. Patients can present with paralysis, cranial nerve deficits and aphasia. Cerebral infarction may develop as an end result of the vasospasm. (Hickey 2009).

Case Study

A fifty year old female presented to a smaller peripheral hospital with a one week history of severe bi-frontal headache after a coughing fit. On examination her neck was sore but no stiffness was elicited. She had little relief with analgesia and had trialed Panadeine Forte

and Nurofen.

The initial cerebral computed tomography (CT) one week previous was reported as normal. The twenty four hours prior to this presentation the patient had experienced two episodes of collapse, no loss of consciousness; however, some confusion was noted. On re-presentation another CT scan was again reported as normal.

A lumbar puncture was performed, cerebrospinal fluid (CSF) was reported - protein and glucose normal, red cell count 146, polymorphs nil, monocytes 2, gram stain negative, bilirubin and oxyhaemoglobin negative.

The patient was transferred to the tertiary referral hospital for CT Angiography (CTA) and Magnetic Resonance Angiography (MRA).

A review of her history was carried out on arrival; there was no prior history of headache or migraine. She had a history of hypertension treated with Candesartan (angiotensin II antagonist receptor) and Verapamil (calcium channel blocker). Asthma was managed using Symbicort and Salbutamol. In 2009 she had an aortic valve replacement complicated by aortic dissection requiring graft and stent; she was on Aspirin for antiplatelet coverage. A history of depression was managed with Citalopram (selective seritonin reuptake inhibitor – SSRI) and Premarin for hormone replacement.

The patient was neurologically intact, power to all limbs was equal with 5/5 power, sensation was intact to all modalities; no ataxia or in-coordination was demonstrated. Her blood pressure (BP) was normotensive at 130/70, she was afebrile.

Further Investigations
A CTA/ CT venogram (CTV) was performed, there was no evidence of dissection, no venous sinus thrombosis, no haemorrhage or aneurysm.

All blood investigations were normal; her echocardiogram (ECG) showed sinus rhythm (SR).

A magnetic resonance image (MRI) /MRA was organised for the next day. It was reported showing bilateral parietal infarcts and a right occipital infarct. There was reduced calibre of the superior sagittal sinus, possibly indicating a venous sinus thrombosis. Possible tiny right carotid aneurysm in region of the carotid cave was reported.

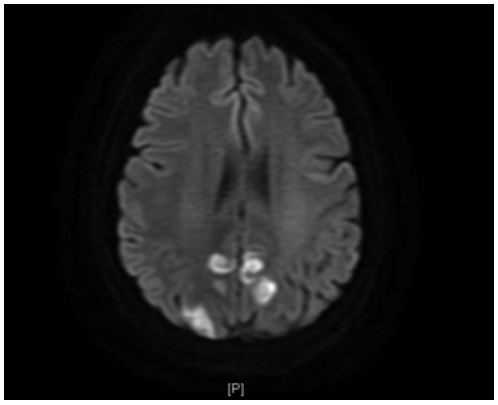

Figure 1 (above): *MRI Brain*

Treatment
Due to the multi territory cerebral infarcts on the MRI she was commenced on therapeutic dose Enoxaparin (low molecular anticoagulation) to prevent further possible cardio-embolic causes. A five day course of steroids, Methylprednisolone was commenced. An angiogram was planned.

The first cerebral angiogram demonstrated areas of focal segmental narrowing. Most pronounced was left anterior cerebral artery (ACA) vessel narrowing. Less pronounced was distal narrowing within the ACA's, posterior divisions of the middle cerebral arteries (MCA) and distal branches of posterior cerebral arteries (PCA). Venous flow was normal, the superior sagittal sinus was normal in appearance. The report concluded this "May be consistent with primary reversible cerebral vasoconstriction syndrome".

A second angiogram the next day was planned. That morning the patient demonstrated a new left leg weakness; a repeat CT scan was unchanged.

The angiogram continued and showed the left internal carotid artery (ICA) demonstrating more severe narrowing than the previous day – suggestive of reversible vasospasm syndrome. 15milligrams (mg) of Verapamil (calcium channel blocker) was administered directly to the left ICA. The right ICA also demonstrated severe vasospasm affecting the right ACA. 20mgs of Verapamil was administered. During this instillation the patient experienced a seizure this was controlled by the anaesthetist, the patient was ultimately intubated. Follow on angiography, on the day after, the left and right ICA showed near total

resolution of vasospasm. A further 10mg of Verapamil was instilled into the right vertebral artery (VA). On completion of the angiogram she was transferred to the intensive care unit (ICU) for SAH vasospasm protocol (bedrest and Nimodipine (calcium channel blocker) infusion).

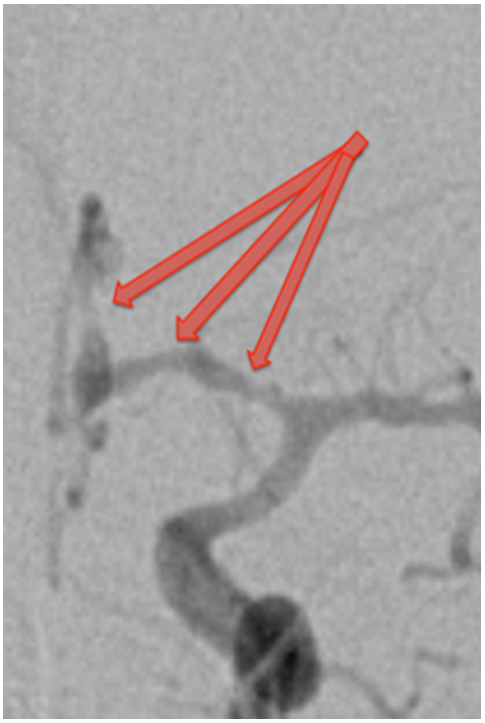

Figure 2 (Above): *Segments of vessel narrowing throughout the circulation, here seen in the proximal portion of the left anterior cerebral artery – prior to Verapamil.*

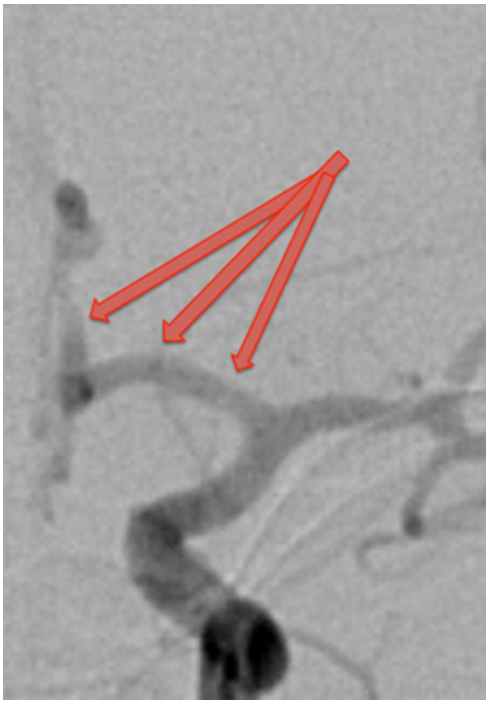

Figure 3 (Above): *Proximal portion of the left anterior cerebral artery – post Verapamil.*

A third angiogram showed marked improvement, with little if any vasospasm. A further 10mg of Verapamil was instilled into the left and right ICA and right vertebral arteries.

There was no neurological deficit once extubated. The patient remained on the SAH vasospasm protocol of Nimodipine for 21 days, intravenous (IV) then oral. A visual field deficit was noted in ICU; an ophthalmology review noted a bilateral paracentral scotoma consistent with the posterior occipital lesion. Her headache improved, she was discharged fifteen days post intra- arterial Verapamil infusion.

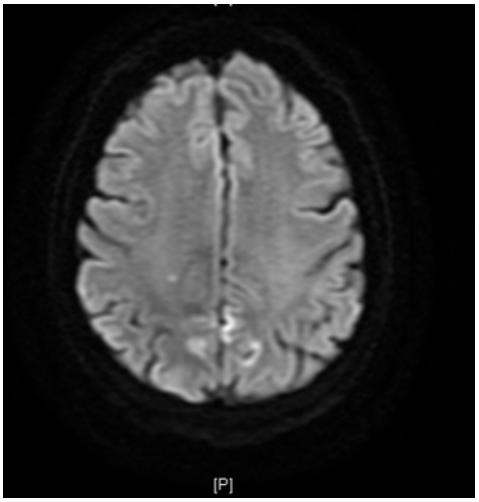

Figure 4 (Above): *10 Days post Verapamil*

This patient was discharged on oral Nimodipine, Amitriptyline, Pantoprazole, Phenytoin, Aspirin, Symbicort. Ceased medications were Citalopram, Verapamil SR and Premarin.

Discussion

Reversible Cerebral Vasoconstriction Syndrome (RCVS) was first described in 1988 by Call and Fleming (Call, Fleming, Sealfon, Levine, Kistler and Fisher 1988). It is known as “Call- Fleming syndrome”, “benign angiopathy of the nervous system”, “postpartum angiopathy” and “drug induced vasospasm”. (Neil, Dechant and Urtecho. 2011). It describes a clinical syndrome characterised by sudden - thunderclap severe headache, fluctuating neurological deficits and fully reversible segmental vasoconstriction of the medium and large cerebral arteries (Neil.et al 2011).

The presentation is usually a thunderclap or severe headache. Altered mental status, sei-

zures and visual disturbance have also been reported. Alterations in haemodynamics can lead to haemorrhage or ischaemic stroke. (Ducros, Boukobza, Porcher, Sarov, Valade and Bousser 2007; Neil.et al 2011; Koopman, Teuneter, Laan, Uyttenboogaart, Vroomen, Keyser, and Luijckx 2008) Cortical subarachnoid haemorrhage (SAH) has also been reported as a complication (Ducros, et al 2007). RCVS commonly affects young healthy women of child bearing age (Ducros, et al 2007, Neil.et al 2011). Precipitating factors have been identified as eclampsia, the postpartum period and exposure to vaso-active substances including nasal decongestants, triptans and selective seritonin reuptake inhibitors. (Ducros, et al 2007; Neil.et al 2011; Koopman et al 2008; Singhal, Caviness, Begleiter, Mark, Rordorf, and Koroshetz. 2002).

Pathophysiology of RCVS is poorly understood. Transient alterations in vascular tone modulated by the sympathetic nervous system are suspected to play a role (Neil.et al 2011). The known occurrence of reversible vasoconstriction following SAH, sympathomimetic drug abuse, surgical manipulation and closed head injury (CHI) suggest various forms of chemical and mechanical stimuli can precipitate the process (Call, et.al.1988). It is important to differentiate between other syndromes, usually RCVS is a diagnosis of exclusion but thunderclap headache is a hall mark symptom (Koopman, Uyttenboogaart, Luijckx, Keyser, and Vroomen. 2007; Neil.et al 2011; Schwedt, Mathuru, and Dodick, 2006). In comparison Cerebral Angiitis is often a gradual onset rarely presenting with headache, the CSF is abnormal with a large percentage of abnormalities with raised protein. CSF in RCVS is usually normal (Koopman, et al. 2007; Schwedt, et al. 2006).

Treatment

The treatment of RCVS remains unclear however non-invasive management of hypervolaemia, hypertension and haemodilution are used as first line measures. Endovascular treatment with intra-arterial infusions of calcium channel blockers such as verapamil appear to exert a prolonged effect on arterial dilation without significantly affecting haemodynamic stability or intracranial pressure (ICP) during the procedure (Stuart, Helbok, Kurtz, Schmidt, Fernandez, Lee, Badjatia, Mayer, Lavine, Meyers, Connolly and Claasen 2011).

Cessation of vaso-active drugs such as nasal sprays, SSRIs, sympathomimetic drugs (cocaine, amphetamine, ergot) is essential (Neil.et al 2011; Singhal, Caviness, Begleiter, Mark, Rordorf, and Koroshetz, 2002; Stuart, et al 2011). The use of steroids has been trialled but not proven (Koopman, et al. 2007). Currently the use of Nimodipine is to provide a cerebral vasodilator action and hence increase the cerebral blood flow (iMIMs 2011).

Progress

The MRI and MRA was repeated 2 monthly, this did not show any further ischaemia or vessel spasm. Review in the eye clinic showed an improvement in the visual deficit. This patient returned to work as a nurse at a rehabilitation hospital.

Reflections

This case study highlights that not everything in Neuroscience Nursing and Stroke is black and white. Something that had always been associated with Neurosurgery came across the corridor to meet me in our Acute Stroke Unit. This case of RCVS was another learning curve to explore and discover that all is not as it seems – the first question I asked in the unit was, had she had a SAH?

There is still debate as to the validity of this syndrome; many Neurologists are sceptical of it being a 'real' syndrome. Real or not, this syndrome has now come across my path three times in a year, a hat trick!

It is vital in Neuroscience Nursing to remember, there is always something waiting around the corner to pique your interest!

Acknowledgements

Dr Ferdinand Miteff, Neurologist, Interventional Neuroradiology Fellow, RNSH.

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