Ulster Med J 2012;**81(1):**59-64

Abstracts

Spring & Autumn Meetings of The Ulster Society of Internal Medicine

Ulster Society of Internal Medicine Spring meeting: 13th May 2011

CAUSEWAY HOSPITAL, COLERAINE

Rare Cause of Lymphocytic Colitis.

CG Harrington, South Eastern HSC Trust, Belfast, UK

This case report demonstrates an unusual cause of lymphocytic colitis.

A 56 year old male presented to a gastroenterology outpatient clinic with diarrhoea. It was a relatively mild presentation with 2-3 semiformed motions per day. He had no systemic upset, no weight loss and was still able to work as a dentist. He also complained of frequent coughs and colds. Respiratory, cardiovascular, abdominal and neurological examinations revealed no abnormalities. Bloods revealed normal inflammatory markers and a normal albumin. His total white cell count was just below normal at 3.3 (neutrophils 2.09, lymphocytes 0.62, monocytes 0.48). Most significantly, he had reduced immunoglobulins (IgG 4.39, IgA 0.7, IgM 0.22). Colonoscopy appeared normal apart from some minor erythema in the rectosigmoid mucosa. Colonic biopsies revealed lymphocytic colitis. An OGD was normal and duodenal biopsies were normal.

The patient was referred to an immunologist. Further investigations, including measurement of immunological response to vaccination were carried out. Subsequently, a diagnosis of Common Variable Immunodeficiency (CVID) was reached.

CVID is the most common form of severe antibody deficiency affecting both adults and children. The hallmark immunological defect is impaired B cell differentiation with resultant defective synthesis of immunoglobulin. The diagnosis should be considered in patients presenting with recurrent infections who have low immunoglobulin levels.[1]

This case provides some good teaching points. It illustrates the insidious and nonspecific nature of CVID. It also demonstrated a rare cause of lymphocytic colitis.

REFERENCES

1 Ahn S, Mayer L, Cunningham-Rundles C. Evaluation and diagnosis of common variable immunodeficiency. *Up to date*

Assessment of Cardiac CT Ca scores by allied health professionals.

P McKavanagh[1], PM Donnelly[1], P Ball[2],

Departments of Cardiology[1] and Radiology[2], Ulster Hospital, South Eastern Trust, Belfast, UK.

The 2010 NICE guidance[1] for the assessment of chest pain suggested an important "gatekeeper" role for coronary artery calcification (CAC) assessment. In the NICE algorithm calcium scores (CS) determine which further cardiac imaging techniques are required in low risk patients. The aim of this study was to determine the feasibility of radiographer's CS assessment. 63 patients with chest pain had Agatston scores prospectively determined by two experienced cardiac radiographers. All CS examinations were performed on a Philips Brilliance 64 detector system, using a standard non-contrast enhanced prospective gated protocol (120kV, 3mm slice thickness). All images were anonymised and transferred to an off-line workstation for interpretation. A semi-automated algorithm identified areas of CAC. The radiographers' assessments were compared to the scores obtained by an experienced consultant cardiologist.

The mean Agatston score was 376 (range 1.4 – 3900). A high degree of reliability was found between Calcium measurements by three observers, the average measure ICC was 0.988 and the 95% confidence interval was from 0.982 to 0.992%. There was a non-significant trend to score variance with increased CS, for those with scores >1000 ($p = 0.45$). Further analysis revealed that image noise contributed to 98% of this absolute score variance. Interestingly no significant difference was found in the calcium score quartile allocated to subjects. Radiographer assessment of coronary calcium scores is feasible and accurate. If the NICE guidance is to be implemented it is likely that there could be an extended role for radiographers.

Cooper A, Calvert N, Skinner J, et al.Chest pain of recent onset: Assessment and diagnosis of recentonset chest pain or discomfort of suspected cardiac origin. London: National

Clinical Guideline Centre for Acute and Chronic Conditions. 2010. Available online at http://www.nice.org.uk/nicemedia/live/12947/47931/47931.pdf. Last accessed 12th April 2011.

The role of placental p38-MAPKa, ERK and JNK nitration and antioxidant supplementation in the pathogenesis of type 1 diabetic pre-eclampsia in a sub-cohort of the Diabetes and Pre-eclampsia Intervention Trial (DAPIT)

PC Johnston[1], LA Powell[2], DR McCance[1], K Pogue[2], C McMaster[2], S Gilchrist[2], VA Holmes[3], IS Young[2], A McGinty[2]

1 Regional Centre for Endocrinology and Diabetes, Royal Victoria Hospital, Belfast, UK,

2 Nutrition and Metabolism Group, Centre for Public Health, Queen's University Belfast, UK,

3 Nursing and Midwifery Research Unit, School of Nursing and Midwifery, Queen's University Belfast, UK

Aim: To examine the role of placental p38-Mitogen-Activated Protein Kinase α (p38-MAPKa), Extra Cellular-Signal Regulated Kinase (ERK) and c-Jun NH2-Terminal Kinase (JNK) nitration in the pathogenesis of type 1 diabetic pre-eclampsia, and their putative modulation by vitamin C and E supplementation.

Methods: Placental samples were obtained from a sub-cohort of the DAPIT study[1]: a randomised placebo-controlled trial of antioxidant supplementation to reduce pre-eclampsia in type 1 diabetic pregnancy. DAPIT placentae: placebo-treated normotensive (n=17), pregnancy induced hypertension (n=7) and pre-eclamptic (n=6): vitamin-treated, normotensive (n=20), pregnancy induced hypertension (n=4) and pre-eclamptic (n=3) were analysed. Protein tyrosine nitration was assessed by means of immunohistochemistry in paraffin-embedded tissue. Catalytic activity of placental MAPK's was measured by enzyme-linked immunosorbent assay (ELISA).

Results: Nitrotyrosine immunostaining was present in placebo-supplemented normotensive, pregnancy induced hypertension and pre-eclamptic placentae, with no significant difference observed between the groups. Vitamin supplementation did not change nitrotyrosine formation in normotensive or pre-eclamptic placentae. There was a non-significant trend towards decreased p38-MAPKa activity in pre-eclamptic vs normotensive placentae, but this was not augmented by vitamin supplementation in either group. ERK and JNK did not significantly differ amongst the three outcome groups and vitamin supplementation did not significantly alter their activity.

Conclusion: Nitrotyrosine immunopositivity in normotensive diabetic placentae indicates some degree of tyrosine nitration in uncomplicated diabetic pregnancy, possibly due to inherent oxidative stress and peroxynitrite production. Our results suggest that p38-MAPKa, ERK and JNK nitration is not directly involved in the pathogenesis of type 1 diabetic pre-eclampsia and is not modulated by vitamin-supplementation.

REFERENCES

1 Mc Cance DR, Holmes VA, Maresh MJA, Patterson CC, Walker JD, Pearson DWM, Young IS on behalf of the DAPIT study group. Vitamin C and vitamin E for the prevention of pre-eclampsia in women with type 1 diabetes (DAPIT): A multicentre randomized placebo-controlled trial. *The Lancet* 2010; **376(9737)**:259-266.

Ulster Society of Internal Medicine Autumn meeting: 21st October 2011

CAUSEWAY HOSPITAL, COLERAINE

On-site percutaneous coronary intervention (PCI) at Altnagelvin Area Hospital has led to a significant reduction in time to PCI in patients presenting with NSTEMI and meets the ESC 2010 guidelines.

M Monaghan, C Small, C Hutchinson, E Armstrong, P Smart, S Hughes, JA Purvis, AJ McNeill, P McGlinchey & MJ Moore.

Cardiology Department, Altnagelvin Hospital, WHSCT.

The European Society of Cardiology (ESC) published guidelines in 2010 on the management of patients presenting with non-ST elevation MI (NSTEMI). They recommend that coronary angiography and revascularisation should be performed during the same hospital stay and preferably within 72 hours of admission.[1]

A dedicated on-site PCI service was introduced in the Western Health and Social Care Trust at Altnagelvin Area Hospital in February 2010 with emergency cardiac surgery support provided by the Belfast Trust. Prior to 2010, patients diagnosed with NSTEMI requiring PCI were transferred to Belfast.

The aim of this pilot study was to investigate if introduction of an on-site PCI service (1) is safe (2) decreases time spent in hospital (3) reduces time to revascularisation as recommended by the ESC.

A retrospective study was made comparing length of hospital stay and time to revascularisation between a randomly selected cohort of patients (n=23) who presented with NSTEMI in 2009 prior to introduction off on-site PCI and patients (n=45) who presented post on-site PCI in 2010. The results are tabulated (Table 1). A significant reduction in time to PCI from 7.54 days (+/- 5.46) to 2.1 days (+/-1.4) p=0.009 was shown with a concomitant reduction in hospital stay from 5.71 (+/-1.98) to 4.29 (2.62) p=0.1. Survival to discharge and 90 days was 100%.

The establishment of on-site PCI is safe and has significantly increased the proportion of patients receiving coronary artery revascularisation within the target 72 hours recommended by the ESC.

REFERENCES

1 Guidelines on myocardial revascularization. The Task Force on Myocardial Revascularisation of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). European Heart Journal (2010) 31, 2501–2555.

Table 1

	Pre on-site PCI	Post on-site PCI	p-value
Patients (n)	23	45	
Age (Years) +/- SD	61 +/- 11	62 +/- 13	
Male (n) (%)	15(65%)	40 (89%)	
LOS (Nights) +/- SD (Excluding inpatient CABG (n=2))	8.73 +/- 10.28 (5.71 +/- 1.98)	4.29 +/- 2.62	0.0526 0.1
Time to Catheterisation (Days) +/- SD	3.22 +/- 2.79	2.16 +/- 1.20	0.0934
PCI performed (n) (%)	11 (48%)	28 (62%)	
Referred for CABG (n) (%)	3 (13%)	2 (4%)	
Referred for Medical Management (n) (%)	9 (40%)	15 (33%)	
Days to PCI +/- SD	7.54 +/- 5.46	2.1 +/- 1.4	0.0090
DES (n) (%)	7/11(64%)	19/28 (68%)	
Radial Access (n) (%)	7/11 (64%)	15/45 (33%)	
Alive to Discharge (n) (%)	22 (96%)	45 (100%)	
Alive at 90 Days (n) (%)	22 (96%)	45 (100%)	

Is it safe to send very low risk patients away? An assessment of NICE chest pain guidance.

A Inaba, M Callaghan, J Purvis.

Department of Cardiology, Altnagelvin Hospital, Western HSC Trust, Londonderry.

Recent National Institute of Clinical Excellence (NICE) guidance on the management of chest pain recommends risk stratification for coronary artery disease (CAD) prior to diagnostic testing. If risk is calculated at <10%, causes other than angina should be sought. The aim of this study was to assess whether this strategy is safe and accurate in clinical use. Secondly, to assess potential reduction in workload from EST referrals to rapid access chest pain clinics (RACPC).

The study prospectively assessed 132 consecutive patients referred to our RACPC in summer 2011. Subjects whose clinical data were incomplete were excluded. 101 patients were eligible. Risk stratification as per NICE guidelines was undertaken. Sometimes cholesterol level was not available but otherwise, the strictest interpretation of the strategy was employed.

Mean age of patients was 51±13 years. 46% were female. Mean risk score was 40%. All 23 patients (23%) who had a CAD risk <10% had negative ESTs. In contrast, of the 78 patients who had a risk ≥10%, 23 had either positive or inconclusive results.

The specificity of NICE's risk stratification is 1.00 (95% CI: 0.82-1.00) in this sample. Estimated annual cost-saving from sending very low risk patients away is £54,069 (£66 per EST) at this hospital.

Therefore, NICE guidelines seem to provide a safe means of risk-stratifying patients. General practitioners (GPs) should be asked to calculate CAD risk and refer only those patients with risk ≥10%. Introduction of an electronic referral form province-wide will facilitate this.

An audit of Acute Kidney Injury (AKI) following contrast coronary angiography.

M Connolly, D Mc Eneaney, M Harbinson, N Morgan.

Cardiology Research Unit, Craigavon Area Hospital (CAH), Southern HSC Trust.

3720 coronary angiograms were performed in Northern Ireland in 2010. Coronary angiography and Primary Coronary Intervention (PCI) depend on the use iodinated contrast. Patients undergoing such intervention are at risk of contrast induced nephrotoxicity (CIN). CIN induced Acute Kidney Injury (AKI), defined as a creatinine rise >25% from baseline, begins within 24 hours of contrast administration. The strongest risk factor for CIN is the presence of pre-existent chronic kidney disease (CKD), the UK incidence of which is 5-8%. Other risk factors include increasing age, diabetes, hypertension and peripheral vascular disease[1]. CIN is associated with increased morbidity, mortality and duration of hospital stay. The rising incidence of atherosclerotic disease and requirement for angiography/PCI positions CIN as a major healthcare problem.

Patients undergoing coronary angiogram at CAH between 01/08/2010-01/02/2011 were selected. Renal function was recorded prior to angiogram. Patients identified with significant CKD (eGFR <60mls/min) had a follow up eGFR 48 hours post contrast to assess for AKI. 634 angiograms were performed. 77 patients (12.1%) had an eGFR<60mls/min. 9 patients (11.6%) developed AKI. Mean age was 77.1 years (range 69-85 years). Affected patients demographics and risk factors were recorded (figure 1).

Figure 1	No of patients eGFR<60 (%)
Developed AKI	9 (11.6)
CRF Stage 3	8 (88.8)
CRF Stage 4	1 (11.1)

Hypertension	6 (66.6)
Diabetes	1 (11.1)
Mean contrast dose (range)	132mls (100-190mls)
ACE –I / ARB	7 (77.7)
Diuretic	4 (44.4)
MACE at 6 months	2 Deceased (22.2)

CIN is common post angiography, exceeding 10% in CKD patients. The clinical consequences can be severe and there is no specific treatment once established. Unfortunately serum creatinine is a delayed marker of GFR decline[2], novel biomarkers to detect early CIN are urgently required. Such markers will be the focus of further study.

REFERENCES

1. Lautin EM, Freeman NJ, Schoenfeld AH *et al.* Radiocontrast-associated renal dysfunction: incidence and risk factors. *AJR Am J Roentgenol*. 1991;157(1):49.
2. Nash K, Hafeez A, Hou S. Hospital-acquired renal insufficiency. *Am J Kidney Dis.* 2002;39:930–6.

Placental antioxidant enzyme analysis (glutathione peroxidase, glutathione reductase, superoxide dismutase and catalase) and antioxidant supplementation in the pathogenesis of Type 1 diabetic pre-eclampsia in a sub-cohort from the Diabetes and Pre-eclampsia Intervention Trial [DAPIT]

PC Johnston[1], DR McCance1, L Powell[2], C Mercer[2], K Pogue[2], S Gilchrist[2], VA Holmes[3], IS Young[2], A McGinty[2]

1 Regional Centre for Endocrinology and Diabetes, Royal Victoria Hospital, Belfast, UK,

2 Nutrition and Metabolism Group, Centre for Public Health, Queen's University Belfast, UK,

3 Nursing and Midwifery Research Unit, School of Nursing and Midwifery, Queen's University Belfast, UK

Aim: To examine the role of placental antioxidant enzyme activities [glutathione peroxidase (GPX), glutathione reductase (GRED), superoxide dismutase (SOD) and catalase] in the pathogenesis of Type 1 diabetic pre-eclampsia, and their putative modulation by antioxidant supplementation.

Methods: Stored placental samples (central and peripheral tissue) were obtained from a sub-cohort of the DAPIT study: a randomised placebo-controlled trial of antioxidant supplementation to reduce pre-eclampsia in type 1 diabetic pregnancy. DAPIT placentae: placebo-treated normotensive (n=17), pregnancy induced hypertension (n=7) and pre-eclamptic (n=6): vitamin-treated, normotensive (n=20), pregnancy induced hypertension (n=4) and pre-eclamptic (n=3) were analysed. GPX, GRED and SOD activities were assessed by kinetic analysis using commercially available kits by clinical analyser ILAB 600. Catalase activity was measured by enzyme-linked immunosorbent assay (ELISA).

Results: There was no statistical differences in any of the antioxidant enzyme activities according to DAPIT outcomes in either central or peripheral placentae in placebo-supplemented subjects Vitamin-supplemented placental GPX was significantly lower in normotensive placentae (p=0.031) and significantly higher in peripheral pre-eclamptic placentae in comparison to placebo (p=0.022). Significantly lower levels of placental peripheral catalase was observed in vitamin-supplemented PIH placentae in comparison to placebo.

Conclusions: Our results, suggest that GPX, GRED, SOD and catalase are not directly involved in the pathogenesis of type 1 diabetic pre-eclampsia, however although antioxidant-supplementation did modulate some levels of antioxidant enzymes (GPX, catalase) overt evidence to discriminate antioxidant enzyme activity between any of the DAPIT outcome groups was not found.

Detection of Acute Coronary Occlusion in patients with Acute Coronary Syndromes presenting with Isolated ST-segment Depression.

Daly MJ[1], Finlay DD[2], Guldenring D[2], Nugent CD[2], Tomlin A[1], Smith B[1], Adgey AAJ[1], Harbinson MT[3]

1. The Heart Centre, Royal Victoria Hospital, Grosvenor Road, Belfast, Northern Ireland UK
2. School of Computing and Mathematics and Computer Science Research Institute, University of Ulster, Northern Ireland, UK
3. Centre for Vision and Vascular Sciences, Queen's University, Whitla Medical Building, 97 Lisburn Road, Belfast, Northern Ireland UK

Aim: We hypothesized that 80-lead body surface potential mapping (BSPM) would improve detection of acute myocardial infarction (AMI) and occluded culprit artery in patients presenting with ST-segment depression (STD) only on 12-lead ECG.

Methods and Results: Consecutive patients presenting pre- and in-hospital between 2000-6 with acute ischaemic-type chest pain and an initial 12-lead ECG with STD only of ≥0.05mV in ≥2 contiguous leads were analyzed. Patients with ST-segment elevation (STE) or 12-lead ECG confounders were excluded. AMI was defined as cardiac troponin T (cTnT) ≥0.03μg/L. Flow in the culprit artery at angiography was graded using the TIMI flow grade (TFG) criteria.

Enrolled were 410 patients: of these, 240 (59%) had an occluded culprit artery (TFG 0/1) with AMI, 80 (19%) had a patent culprit artery (TFG 2/3) with AMI, 67 (16%) had TFG 2/3 with cTnT<0.03μg/L and 23 (6%) had TFG 0/1 with cTnT<0.03μg/L. BSPM STE occurred in 267 (65%) patients. For the diagnosis of TFG 0/1 in the culprit artery and AMI, BSPM STE had sensitivity 91% and specificity 72% with STE occurring most commonly in the posterior territory (60%). Patients with TFG 0/1 and AMI were significantly more likely to suffer death or nonfatal MI at 30-days than those with TFG 2/3 and cTnT<0.03μg/L (Adjusted Hazard Ratio 4.12, 95% CI 1.67 to 8.56, p = 0.003).

Conclusion: Among ACS patients presenting with only STD, BSPM identifies STE beyond the territory of the 12-lead ECG with sensitivity 91% and specificity 72% for diagnosis of occluded culprit artery with AMI.

Association of low serum concentrations of sex hormone binding globulin and insulin resistance is stronger in females and independent of body mass index.

Wallace IR[1,2], McEvoy CT[2], Hamill LL[2], Woodside JV[2], Ennis CN[1,2], Bell PM[1], Young IS[2], McKinley MC[2],Hunter SJ[1].

1 Regional Centre for Endocrinology and Diabetes, Royal Victoria Hospital, Belfast, UK. BT12 6BA.

2 Nutrition and Metabolism Group, Centre for Public Health, Queen's University of Belfast, Belfast, UK. BT12 6BJ.

Introduction: Low circulating levels of sex hormone binding globulin (SHBG) have been shown to be a strong predictor of the risk of developing type 2 diabetes. Genetic studies suggest that this may be a primary causal abnormality and the mechanism might relate to effects on insulin resistance.

Methods: Insulin resistance was assessed using a two-step euglycaemic hyperinsulinaemic clamp in 92 (59 male and 33 female) overweight individuals (BMI = 27-35 kg/m^2) at elevated risk of cardiovascular disease (>20% ten-year risk as assessed by the Joint British Societies 2 guidelines). SHBG concentrations were measured using a solidphase, two-site chemiluminescent immunometric technique. Statistical analysis was performed using Pearson's correlation coefficients and linear regression.

Results: For the total group, a statistically significant positive correlation was apparent between SHBG and glucose infusion rate (GIR) ($r=0.421$, $p<0.001$). When analysed by gender, this correlation was stronger in females ($r=0.631$, $p<0.001$), than in males ($r=0.248$, $p=0.059$). Using a linear regression model, including potential anthropometric confounders (body mass index, waist circumference, waist:hip ratio) a doubling of SHBG was associated with a 21% (95%CI: 11%-32%) increase in GIR ($p<0.001$) in the total group; a 15% (95% CI: 1%-32%) increase ($p=0.04$) in males and a 27% (95%CI: 14%-42%) increase in GIR ($p<0.001$) in females.

Conclusions: Low serum SHBG concentration is associated with insulin resistance. This relationship is stronger in females and is unaffected by body mass index.

The case for routine HIV testing in internal medicine

EJ McCarty, Quah SP, Emerson C, Dinsmore WW

In the past there have been many misconceptions about offering HIV testing. Routine testing has been successfully established in many areas of medicine including antenatal screening. We wish to demonstrate the advantages of routine HIV testing using 5 examples in the last year.

1. 56 year old male with severe worsening pneumonia was diagnosed with HIV following a needle-stick injury to member of staff. Recovered following appropriate medical treatment in ICU.
2. 28 year old male was under investigation for respiratory sarcoid and receiving high dose steroids. He was admitted with acute respiratory failure and died in ICU shortly after HIV testing.
3. 21 year old male with sudden onset right sided weakness was treated with methylprednisolone for demyelination. He was subsequently diagnosed with HIV and progressive multifocal leukoencephalopathy.
4. 50 year old female was investigated for 6 stone weight loss with no cause identified. She was admitted with pneumonia and after prolonged hospital admission tested positive for HIV.
5. 23 year old female was under investigation for thrombocytopenia for several years. She was eventually admitted with pneumonia and subsequently diagnosed with HIV.

These cases highlight the problems of attempting to select "at-risk" individuals. The case in favour of routine testing in an era of highly effective treatment is now unanswerable. The consequences of delayed diagnosis are often fatal.

Kernohan's notch phenomenon.

E Nelson, E Kerr, P McCaffrey

Stroke Unit, Southern HSC Trust, Craigavon, UK

Kernohan's notch phenomenon is a rare presentation in patients with a supratentorial mass lesion and herniation. This occurs in approximately five percent of subdural haematomas[1].

We report on a forty-four year old woman who presented unresponsive with left sided hemiparesis and left pupil mydriasis.

An immediate computed tomography (CT) scan of her brain confirmed a five centimetre left frontal subdural haematoma with significant mass effect and midline shift. Her condition deteriorated with a reduction in Glasgow Coma Scale (GCS) and vomiting. She was accepted by neurosurgery and had a craniotomy with evacuation of the haematoma the next day.

A post-operative magnetic resonance imaging (MRI) brain showed abnormal signal intensity in the right cerebral peduncle. This was caused by lateral pressure from the left hemisphere compressing the contralateral cerebral peduncle against the tentorium causing it to notch[2]. This is known as Kernohan's notch. This compression stretches motor tracts and veins leading to the ipsilateral signs, known as Kernohan's notch phenomenon.

This diagnosis should be considered when patients present with motor deficit ipsilateral to a supratentorial mass lesion. Diagnosis is confirmed by MRI which may show a deformity or abnormal signal intensity in the contralateral cerebral peduncle.

Our patient's swallow improved and she regained some motor function. She continued her rehabilitation in the regional acute brain injury unit.

REFERENCES

1. Meagher RJ, Lutsep HL. Subdural haematoma. In: emedicine, 2009; article 1137207.
2. McKenna C, Fellus J, Barrett, AM. False localizing signs in traumatic brain injury. Brain Inj. 2009 July: 23(7): 597-610

Swallow assessment in stroke patients.

E Nelson, P McCaffrey,

Stroke Unit, Southern HSC Trust, Craigavon, UK.

Dysphagia is common after stroke and associated with poorer outcomes. Following national institute for health and clinical excellence (NICE) guidance[1] all stroke patients should have their swallow assessed on admission. Those with problems need specialist assessment within seventy-two hours but

preferably within twenty-four hours of admission.

This audit was a random selection of thirty-two patients from Craigavon and Lurgan stroke units. Criteria were selected from the national sentinel stroke audit[2]. We also recorded Glasgow Coma Scale (GCS), the admitting doctors plan regarding swallow and when referral to speech and language therapy (SALT) was made.

Twenty patients had a GCS of fourteen or more on admission. Of these only three were documented as having a safe swallow by the admitting doctor. Eleven had no comment made on their swallow and the remaining six were made nil by mouth (NBM).

Only one patient had their swallow assessed within four hours, twelve within twenty-four hours and fifteen within seventy-two hours. Five patients did not have their swallow assessed within seventy-two hours having been admitted before a weekend or bank holiday. Of the twenty-one patients who were referred to SALT only seven had been referred within twenty-four hours.

We conclude that doctors need to assess swallow using the water swallow test. Drowsy patients should be NBM and referred to SALT immediately. All stroke nurses should be trained in swallow assessment. Patients not receiving nutrition need a definitive plan before a weekend or bank holiday. All patients should receive nutrition within twenty-four hours.

REFERENCES

1. Stroke: diagnosis and initial management of acute stroke and transientischaemic attack (TIA), NICE clinical guideline 68.
2. The national sentinel stroke audit 2010, Royal College of Physicians.

PFO with stroke – Aspirin vs warfarin vs closure the best management?

V Krishnaswami[1], P Gordon[1], S Sarup[2]

Geriatrics, Belfast HSC Trust, Belfast

Geriatrics, Altnagelvin hospital,Western HSC trust,

Case1 - 73 year old male was admitted with left arm and left leg weakness. He had a past medical history of Hypertension, atrial fibrillation, previous Ischaemic stroke with full neurologic recovery and hypercholestolemia. His current list of medication included warfarin with target INR of two to three, ezemtibe 10 mg od, lisinopril 10 mg od, amlodipine 5 mg od, atorvostatin 20 mg od.On examination he had left pronator drift and rest of the neurologic examination and other systemic examination did not reveal any abnormalities. Investigations findings were CT brain (Image 1,2) with right frontal infarct and echocardiogram showing PFO with ASA(Image 3, 4).Case2 - 38 year old male was admitted with history of transient slurred speech resolving in twelve hours. He had no significant past medical history and not on any regular medications. No significant family history .His Investigations findings were CT head no abnormality, MRI had showed left temporal parietal small infract, echo showed PFO and no ASA . Management - Case 1 : PFO with ASA – PFO closure[1,2] Case 2 : PFO without ASA – warfarin[1,2]

Current evidence, based on PFO in Cryptogenic stroke study(PICSS)[1] and French PFO – ASA study[2] suggest that PFO without ASA treatment options suggest no clinical difference with aspirin and warfarin . PFO with ASA warfarin especially when stroke is a suitable treatment options .The device closure has no superior effect than warfarin but advantage over warfarin is significant bleeding risk reduction.

REFERENCES

1. *Circulation* 2002 Jun 4;**105(22):**2625-31 (PICSS)
2. *N Engl J Med* 2001; **345**:1740-1746 December 13, 2001 (French PFO – ASA Study)

ABBREVIATIONS:

PFO – Patent foramen ovale; ASA – Atrial septal aneurysm.

Warfarin to treat Hemorrhagic stroke

Dr V Krishnaswami, Dr P Gordon

Geriatrics, Altnagelvin Hospital,Western health and social care trust.

A 65 year old lady was admitted with history right temporal headache, generalized tonic clonic seizures. Her medical history includes hypertension, migraine, chronic kidney disease .Medications - Lisinopril 10 mg, Ibuprofen PRN.

On neurological examination the Glasgow coma scale was 15/15, cranial nerve examination including fundus was normal, motor system examination showed increased tone in the right upper and lower limb with pronator drift, power of 4/5 in MRC scale and upgoing plantars.

Investigations revealed normal blood count, liver function, inflammatory markers and coagulation. CT scan (Image 1) revealed hemorrhage and infarct to both sides cerebral hemisphere and reverse delta sign on contrast CT (image 2) . MRI /MR Venogram which showed filling defect in superior saggittal sinus and hypoplastic right transverse sinus (image 5, 6).

Management of patient involved lifelong anticoagulation warfarin with target INR of two to three .She showed elevated IgM kappa paraproteinemia and negative urine for Bence Jones protein.. The final diagnosis is MGUS causing superior saggittal sinus thrombosis[1,2].

Mortality in untreated casesis13.8-48%, in treated cases 82% recovered completely . CT brain scan show infarctions often associated with haemorrhage bilaterally. The presence of both the delta(non contrast CT) and reverse delta(contrast CT) signs increases the likelihood of the diagnosis. MRI/ MRV is the best method of diagnosing cerebral venous thrombosis. MGUS of IgM serotypes are associated with risk of venous thromboembolism. We could not find any previous case reports of saggittal sinus thrombosis due to MGUS[1,2].

1. Ann Oncol (October 2004) 15 (10): 1490-1494. doi: 10.1093/annonc/mdh385
2. Blood, 17 June 2010, Vol. 115, No. 24, pp. 4975-976

