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Abstracts

Ulster Society of Internal Medicine
95th (Spring) Meeting
Friday 27th May 2016

Altnagelvin Hospital

PROGRAMME:

2.00 pm **Pneumocystis jiroveci pneumonia; The who and the where; identifying the population at risk.**

L McCorry. Dept of Microbiology, Kelvin Laboratory, RVH, Belfast HSC Trust, Belfast, UK.

2.15 pm **Pulmonary Embolism: A case requiring rescue percutaneous intervention.**

A.P. Gray, D. Flannery.

Department of Cardiology, Craigavon Area Hospital, Southern Trust, Northern Ireland.

2.30 pm **Pyoderma Gangrenosum in a Critically Ill Patient.**

P Collins, C Devereux, P Windrum.

Dermatology Department, Northern HSC Trust.

2.45 pm **Guest Lecture: "PCSK 9 inhibitors."**

Professor Maurice O'Kane, Clinical Chemistry, Altnagelvin Hospital.

3.15 pm **Afternoon Tea** and **Poster Viewing**

Refreshments sponsored by **Sanofi.**

Poster 1 **Dysplastic Brain.**

D Cousins, E Kerr

Stroke Team, RVH, Belfast Trust.

Poster 2 **Complications after baby number three!**

L Kayes, D Comer, R Sharkey, MG Kelly, M McCloskey.

Respiratory Department, Altnagelvin Area Hospital, WHSCT.

Poster 3 **A case of voriconazole induced adrenal suppression in a patient with polyarteritis nodosa.**

S McDonald, N McKee, G Wright.

Musgrave Park Hospital, Belfast, N. Ireland

3.40 pm **Grand Rounds: Cases from Altnagelvin Hospital.**

4.10 pm **And the band played on! Hypersensitivity pneumonitis from wind instruments.**

G Gamble, E Toner, N Chapman & R Convery.

Respiratory Medicine, Craigavon Hospital. SHSCT.

4.25 pm **Granulomatosis with polyangiitis – cerebral involvement.**

McKnight J, Burns J, McCann S .

Rheumatology department. Northern HSC Trust, Antrim Area Hospital, UK.

5.10 pm Presentation of prize for the best abstract.

2PM ORAL

PNEUMOCYSTIS JIROVECI PNEUMONIA; THE WHO AND THE WHERE; IDENTIFYING THE POPULATION AT RISK.

L McCorry, Department of Microbiology, Kelvin Laboratory, Royal Victoria Hospital, Belfast HSC Trust, Belfast, UK

Pneumocystis jivoreci is an opportunistic pathogen which can lead to life threatening respiratory failure. It has a documented mortality of between 5-20%[1]. Historically it was almost exclusive to HIV patients however; an increase in immunosuppressive therapies has led to a reciprocal increase in the prevalence of Pneumocystis pneumonia (PCP) in this non-HIV population[2]. Despite clear guidelines for PCP prophylaxis in HIV, there is a haphazard approach in other immunosuppressed populations[1].

A growing body of evidence suggests that immunosuppressed patients are at an increased risk of PCP, but to what extent, or to whom that risk is greatest is not certain2.

Given this uncertainty we felt it prudent to review regional rates of PCP to develop a clearer understanding of the potential at risk population. We believe this is the first study of its kind.

We audited a random cohort of 103 patients who tested positive for Pneumocystis jiroveci over a 5-year period in Northern Ireland, who had documented pneumonia. We collated information on potential risk factors, morbidity and mortality.

The highest proportion of patients were cancer patients, however the most at risk population were rheumatology patients, with a 73% mortality rate in those who tested positive for PCP. Admission to ICU was 38% and mortality was 32%. Mortality was highest when prescribed three modalities of immunosuppression in combination, however prednisolone alone carries a mortality rate of 62%.

We feel that prophylaxis guidelines should be considered in these identified high risk groups.

REFERENCES

1. Centres for Disease Control, Guidelines for Prophylaxis Against Pneumocystis carinii Pneumonia for Persons Infected with Human Immunodeficiency Virus, *MMWR*, June 16, 1989 / **38(S-5)**;1-9

2. Kovac, J A. and Masur, H "Evolving health effects of Pneumocystis one hundred years of progress in Diagnosis and treatment", *JAMA* 2009; **301(24)**; 2578-2585

215PM ORAL

PULMONARY EMBOLISM: A CASE REQUIRING RESCUE PERCUTANEOUS INTERVENTION

A.P. Gray, D. Flannery

Department of Cardiology, Craigavon Area Hospital, Southern Trust, Northern Ireland

We present the case of a 35-year-old female with no past medical history who presented with a one-week history of exertional shortness of breath. She denied any additional symptoms suggestive of venous thromboembolism. She was haemodynamically stable with oxygen saturations of 99% on room air. Admission biochemistry was unremarkable except a D-Dimer elevated at >2 (reference range <0.5). ECG and chest x-ray were equally unremarkable. She was admitted under the medical team and treated with therapeutic enoxaparin for presumed pulmonary embolism. Her risk factors constituted raised body mass index and oral contraceptive use.

CT pulmonary angiogram confirmed large bilateral pulmonary emboli. Despite this she remained stable during admission however whilst attending for cardiac echocardiogram suffered a respiratory arrest. She was successfully resuscitated but subsequently arrested again twice. She received thrombolyis with Actilyase but remained haemodynamically unstable with oxygen saturations 70%, despite intubation and ventilation, and required inotropic support. She was profoundly acidotic (pH6.9) with a lactate of 20. On discussion with the tertiary referral centre it was felt that without intervention she was unlikely to survive. She was transferred to the cardiac catheterisation suite two and a half hours post arrest, remaining acidotic, hypotensive and hypoxic. Catheterisation of her pulmonary artery using a balloon tipped 6f swan ganz catheter was successful at restoring flow in both lung fields. Her haemodynamic status immediately improved with normalisation of oxygen saturations, pH and lactate. She was extubated the following day and was found to have GCS 15 with appropriate verbalisation.

230PM Oral

Pyoderma Gangrenosum in a Critically Ill Patient

P Collins, C Devereux, P Windrum.

Dermatology Department, Northern HSC Trust.

Pyoderma gangrenosum is a rare autoimmune condition which can lead to devastating skin loss if it is not diagnosed and managed promptly and appropriately.

A 72-year-old male was admitted to our intensive care unit with septic shock, requiring inotropic support. He developed progressive ulceration following surgical drainage of an abscess in his left axilla. This was managed as Necrotizing Fasciitis. A dermatological opinion was sought due to continued deterioration despite four further debridements. Clinically the ulcer was typical for Pyoderma Gangrenosum.

Clinical management was challenging as the patient required immunosuppressive treatment in the setting of his complex comorbidities of pneumonia and septicaemia. A multi-disciplinary approach involving dermatology, tissue viability team, intensive care physicians, surgical team and haematology was required.

Subsequent investigations have revealed this patient likely has a myeloproliferative disorder associated with his JAK2 positivity.

There have only ever been two reported cases of JAK2 positivity in Pyoderma Gangrenosum.[1]

REFERENCES

1. Pyoderma Gangrenosum in association with Janus kinase 2 (JAK2V617F) mutations. J. A. Palanivel, A. E. Macbeth and N. J. Levell. 21 May 2012. Volume 38, Issue 1, Pages 44-46. Clinical and Experimental Dermatology

410PM ORAL

AND THE BAND PLAYED ON! HYPERSENSITIVITY PNEUMONITIS (HP) FROM WIND INSTRUMENTS

Gareth Gamble, Emma Toner, Naomi Chapman & Rory

Convery

Respiratory Medicine, Craigavon Hospital. SHSCT.

A male ex-smoker aged 70 presented with failure to recover following an episode of Streptococcal pneumonia. He started playing saxophone again after several years to 'strengthen' his lungs. Radiograph and serial CT chest showed evidence of mosaic infiltrate in keeping with HP. Empiric steroids failed to resolve his symptoms. A history review suggested reed fungal colonisation as potential trigger. Swabs confirmed Fusarium Dimerum as well as Staphylococcus Aureus. Advice re reed replacement/avoidance was given with good effect.

A 31-year-old female was investigated for right sided chest pain and found to have a nodular infiltrate in the right lower lobe. Open Lung biopsy confirmed an interstitial pneumonia, with MDT discussion suggesting HP. In depth history revealed exposure to stagnant secretions from a borrowed Euphonium several weeks before presentation. Recovery is ongoing.

Initially in susceptible individuals an acute type III and type IV reaction occurs in this complex dynamic clinical syndrome. Wind and brass instruments are rare causes of HP by virtue of biofilms or wooden reed fungal contaminants. Sterilizing instruments is advised but the best treatment is avoidance. Steroids can have a role in speeding up resolution.

425PM ORAL

GRANULOMATOSIS WITH POLYANGIITIS – CEREBRAL INVOLVEMENT

McKnight J, Burns J, McCann S.

Rheumatology department. Northern HSC Trust, Antrim Area Hospital, UK.

A 49-year-old male with a history of granulomatosis with polyangiitis was admitted acutely with a three-week history of headache, vomiting, nystagmus on lateral gaze and ataxia.

Granulomatosis with polyangiitis had been diagnosed 18 months prior; he initially presented with an ulcerating buccal mucosal lesion, epistaxis, nasal crusting, recurrent sinusitis and cavitating lung lesions identified on CT scanning. Previous biopsies of the buccal mucosal lesion confirmed the presence of a mixed inflammatory infiltrate with high concentrations of eosinophils. Initial serology confirmed elevated antibodies to PR3(>8 AI) and cANCA (40). Despite initially achieving disease remission with mycophenolate and high dose oral corticosteroid, the patient's compliance with maintenance immunosuppressive medication remained poor. He also had a history of chronic obstructive pulmonary disease (COPD) and continued to smoke between 40 and 80 cigarettes daily.

During his acute admission, his erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were noted to be markedly elevated. An MRI brain scan with contrast confirmed the presence of multiple enhancing nodular lesions which appear to be in continuity with the choroid plexus within the temporal horns and within the fourth ventricle. The lesions measured up to 2.7cm in diameter with significant adjacent oedema which also involved the brainstem.

Despite induction therapy with intravenous cyclophosphamide 750mg fortnightly and high dose corticosteroid, this patient's vasculitis remains active. He has now been commenced on a course of intravenous rituximab therapy in an attempt to obtain remission.[1,2]

REFERENCES

1. Rinella ME. Nonalcoholic fatty liver disease: a systematic review. Jama. 2015;313(22):2263-73.
2. Federico A, Zulli C, de Sio I, Del Prete A, Dallio M, Masarone M, et al. Focus on emerging drugs for the treatment of patients with non-alcoholic fatty liver disease. World J Gastroenterol. 2014;20(45):16841-57.

POSTER 1

DYSPLASTIC BRAIN

D Cousins, E Kerr. Stroke Team, RVH, Belfast Trust.

A 26-year-old man with a history of migraine presented to the Emergency Department (ED) with headache and an episode of transient right arm heaviness. Neurological examination was normal. Blood pressure was 220/130mmHg. CT brain, urinalysis, ultrasound renal tracts and bloods were normal. The patient was commenced on Amlodipine and discharged with a diagnosis of migraine and probable essential hypertension.

8 weeks later the patient presented to ED with headache and a moderate, persistent right hemiparesis, dyspraxia of his right arm, mild dysarthria and a blood pressure of 156/90mmHg. MRI brain showed bilateral infarction in a 'watershed' distribution. CT angiography showed severe, irregular narrowing of both distal internal carotid arteries, and irregular narrowing with aneurysm formation of the right renal artery. A diagnosis of probable fibromuscular dysplasia (FMD) was made.

He with intravenous fluids, bed rest, discontinuation of antihypertensives and subsequent inotropic support to achieve BP >180/100. He deteriorated over the following days with recurrent stroke. He developed massive bilateral stroke with malignant MCA syndrome. He was not suitable for extracranial-intracranial bypass surgery or decompressive hemicraniectomy and died.

Family history revealed a florid history of 'migraine with aura' and transient ischaemic attack. MR angiography

revealed irregular stenoses of internal carotid arteries bilaterally in the patient's mother and sister. The family were referred to genetics.

DISCUSSION:

This patient had a diagnosis of fibromuscular dysplasia. This results in stenosis, aneurysms and dissections in any artery. The most commonly affected are the renal, carotid and vertebral arteries. 75% of people with FMD have renal artery stenosis and hypertension.

It is likely that this patient's cerebral perfusion had been maintained by hypertension, and introducing amlodipine precipitated reduced perfusion.

FMD has a strong genetic component and although this is a rare case it highlights the importance of an accurate family history in patients with atypical or hemiplegic migraine.

POSTER 2

COMPLICATIONS AFTER BABY NUMBER THREE!

L Kayes, D Comer, R Sharkey, MG Kelly, M McCloskey.

Respiratory Department, Altnagelvin Area Hospital, WHSCT, Londonderry.

This is the case of 28-year-old girl, with no past medical history, who at 34 weeks pregnant with her third child, was admitted on 12-09-2015 with abdominal pain and elevated inflammatory markers. No source of sepsis was identified and she had an exploratory laparotomy, appendectomy and caesarean section. She delivered a healthy baby.

Five days after surgery she developed chest pain and hypoxia. Chest x-ray showed left basal consolidation. CT pulmonary angiogram was normal. She became progressively more hypoxic and developed left lower lobe collapse. Over the next week she developed a complete collapse of her left lung due to mucoid impaction. She had a bronchoscopy on 01-10-2015 and there were copious tenacious secretions left lung. After this she improved. Four days later on 04-10-2015 she developed further left sided chest pain and chest x-ray showed a pneumothorax left lung. Chest drain was inserted and she improved and stabilised. Less than 24 hours later she became unwell again with worsening hypoxia and further chest pain. CT pulmonary angiogram now showed extensive thrombus right lung, left lung was still deflated. Her family were informed that she was gravely ill with significant right heart strain, oxygen requirements of 100% and that there was a high probability that she would not survive. She remained critically ill for seven days in Intensive Care.

Over the next few weeks she slowly improved and was discharged on 26-10-2016. At review she is very well. She does not want any further children but has yet to be sterilised.

This case is presented to discuss the numerous medical issues. Seven different medical specialties were involved in her care at consultant level and numerous investigations undertaken. No identifiable risk factors have been identified.

POSTER 3

A CASE OF VORICONAZOLE INDUCED ADRENAL SUPPRESSION IN A PATIENT WITH POLYARTERITIS NODOSA.

S McDonald, N McKee, G Wright,

Musgrave Park Hospital, Belfast, N. Ireland

ABSTRACT

A 78-year-old man with a long history of polyarteritis nodosa, ischaemic heart disease, chronic kidney disease and a cavitating lung lesion with mycobacterium malmoense was admitted with generalised muscular pain, fatigue, anorexia, decreased mobility and dizziness over the last month. His sputum had recently cultured aspergillus fumigatus and he had commenced on voriconazole six weeks prior. His maintenance therapy for polyarteritis was long term prednisolone and mycophenolate mofetil. His examination demonstrated that he was normotensive with generalised muscular tenderness, ankle oedema, an aortic murmur, bronchial breathing of the left upper lobe but was otherwise unremarkable with a normal postural blood pressure. Bloods demonstrated normal inflammatory markers, a new transaminitis, deteriorating renal function with urea of 37 and hyperkalaemia at 6.2, which required treatment. ANCA tests were negative. A synacthen test was inadequate with cortisol rising from 136 to 268 nmol/L at 30 minutes. Hypoadrenalism due to long-term steroid use was a working diagnosis. His prednisolone was increased to 20mg and he received intramuscular depomedrone 120mg. His voriconazole, mycophenolate and anti-tuberculous drugs were stopped 4 days post admission. He was discussed with renal who felt uraemia wasn't driving his symptoms and agreed there was no evidence of vasculitis relapse. A random morning cortisol checked 12 days post admission was 463nmol/L. He could now mobilise independently and his renal/liver function returned to baseline. It was felt that the voriconazole was the culprit agent and he didn't restart this on discharge, after consultation with respiratory.