ABUSE OF FENTANYL ANALGESIC PATCHES

Editor,

A 34-year-old male with a background history of drug abuse, alcohol dependence and depression was recently admitted to our ICU. The patient had acquired a Fentanyl patch, of unknown brand name. He removed the gel matrix containing the active drug, ignited it and inhaled the fumes generated through a rolled-up tin foil.

He immediately lost consciousness and was found by a friend who called the Ambulance Service, who in turn found him comatose with vomitus visible in the mouth. He was given Naloxone and regained consciousness and was brought into hospital, complaining of dyspnoea. He was admitted under the respiratory team with suspected aspiration pneumonia. A chest x-ray on admission showed mild bibasal infiltrates (Figure 1). Over the next 12 hours he deteriorated, developing diffuse bilateral infiltrates and hypoxic respiratory failure in keeping with Acute Respiratory Distress Syndrome (ARDS) requiring tracheal intubation and mechanical ventilation. He spent 1 month in our ICU, undergoing tracheostomy on day 15. His respiratory function remained poor for around 21 days before slowly improving and weaning from ventilation. His overall diagnosis was chemical pneumonitis due to both aspiration and the direct toxic effects of the inhaled fumes.

This case highlights the dangers of smoking opioid based patches, both in terms of the potential for rapidly absorbing an extremely high dose of the drug systemically and inhaling the fumes of the ignited matrix. We have not identified another case report of chemical pneumonitis resulting from smoking a Fentanyl patch, but there is at least one report of a patient developing alveolar proteinosis after smoking \(^1\). Indeed, there are several media reports of deaths from smoking Fentanyl patches recently. \(^2,3\)

It is worrying that a simple internet search engine query for smoking “Fentanyl Patch” reveals a large number of websites of forums devoted to the subject, with many references to the fact that opioid based patches are often worn by the elderly with chronic pain conditions, making them an easy target for theft.

Diarmaid Dillon and Finbarr O’Neill
Regional Intensive Care Unit, Royal Victoria Hospital, 274 Grosvenor Road, Belfast BT12 6BA Northern Ireland UK
Correspondence to: diarmaiddillon@gmail.com

REFERENCES:
1. Chapman E, Leipsic J, Satkunam N, Churg A. Chest. 2012;141(5):1321-3.
2. Martin R. Addicts die after smoking pain-relief patches. The Local SE, Sweden’s news in English. 2012 Jan 18. Available from: http://www.thelocal.se/20120118/38570. Last accessed May 2016.
3. Ghebreslassie M, Layson G, Doucette T. Fentanyl kills ‘perfect wife’ and other addicts in Ontario City. CBS News. 2012 Jul 20. Available from: http://www.cbc.ca/news/canada/windsor/fentanyl-kills-perfect-wife-and-other-addicts-in-ontario-city-1.1155726. Last accessed May 2016.

HYPERTROPHIC OSTEOARTHROPATHY ASSOCIATED WITH NON-METASTATIC RENAL CELL CARCINOMA; REPORT OF AN UNUSUAL CASE.

Editor,

Hypertrophic Osteoarthropathy (HOA) is an uncommon syndrome comprising periostitis of long bones, digital clubbing and synovial effusions. HOA is divided into primary and secondary forms\(^1\). Secondary HOA is associated with a variety of medical conditions, including malignancy. The vast majority of malignancy-associated HOA is in the setting of...
thoracic malignancy. Association with extrathoracic, non-metastatic malignancy is very rare. We report a case of hypertrophic osteoarthopathy associated with non-metastatic renal cell carcinoma (RCC).

A 60-year old gentleman was referred by his General Practitioner to our department for radiographs of both knees, having reported a short history of right-sided knee pain, with mild tenderness on examination. He was a non-smoker and had no significant medical history. Radiographs of both knees demonstrated periosteal reaction at the medial and lateral aspects of both distal femora (Figure 1A), in keeping with HOA. Radiographs of both ankles demonstrated a similar periosteal reaction in both distal tibiae. The patient did not have any clinical features of pachydermoperiostosis (primary HOA) or a known underlying condition to account for the development of secondary HOA. A chest radiograph was normal. Given that no cause for the patient’s HOA was apparent, a decision was made to perform a Computed Tomography (CT) scan of the thorax, abdomen and pelvis to evaluate for an underlying malignancy. This demonstrated a 3.5 cm right renal mass, suspicious for a renal cell carcinoma (Figure 1B). There were no metastases or other abnormality identified. The patient was referred to the local Urology Service and underwent an open partial nephrectomy. Histological analysis confirmed a grade II renal cell carcinoma. The patient recovered well and remains disease free at six months following surgery.

It was Virchow in 1895 who first described HOA in the setting of malignancy, in a patient with pulmonary metastases. A strong association of HOA with malignancy has since been established. Given the preponderant association of HOA with thoracic malignancy, it has been commonly referred to as “hypertrophic pulmonary osteoarthopathy”. The small number of reported cases associated with an extra-thoracic malignancy have predominantly been in the young adult and paediatric populations.

On review of the English medical literature we identified seven other cases of HOA associated with renal cell carcinoma. In five of the reported cases, the development of HOA was seen in patients with pulmonary metastases. The other two cases reported HOA in patients with renal cell carcinoma without pulmonary metastases. Recent evidence has implicated vascular endothelial growth factor (VEGF) as a key factor in the pathogenesis of HOA. Over expression of VEGF has been well documented in renal cell carcinoma and we hypothesise that this had a role in the development of HOA in this case.

In conclusion, this is the third case in the English medical literature of HOA associated with renal cell carcinoma without pulmonary metastases. It emphasises the importance of further investigation when a patient presents with HOA without an apparent cause. In rare cases an extra-thoracic malignancy may be present.

The authors have no conflict of interest.

Redmond CE, Healy GM, Redmond PL.
Department of Radiology, Midland Regional Hospital Tullamore, Co. Offaly.

REFERENCES:
1. Pineda C, Martinez-Lavin M. Hypertrophic osteoarthopathy: what a rheumatologist should know about this uncommon condition. Rheum Dis Clin North Am. 2013;39(2):383–400.
2. Virchow R. (1895) Veränderungen des Skelets durch Akromegalie. Berliner Klinische Wochenschrift, 1895;32: 1102.
3. Chen YC, Tiu CM, Bai LY, Liu JH. Hypertrophic pulmonary osteoarthropathy associated with disease progression in renal cell carcinoma. J Chin Med Assoc. 2003;66(1):57–62.
4. Pandita KK, Afaq S, Singh D, Mushfaq D, Masood I. Finger clubbing in a patient of myelofibrosis with renal cell carcinoma. J Assoc Physicians India. 2012;60:124–6.
5. Silveira LH, Martinez-Lavin M, Pineda C, Fonseca MC, Navarro C, Nava A. Vascular endothelial growth factor and hypertrophic osteoarthopathy. Clin Exp Rheumatol. 2000;18(1):57–62.

HEPATOCELLULAR CARCINOMA EXTENDING INTO THE RIGHT ATRIUM.

Editor,
Cardiac tumours are rare and often secondary, rather than primary in origin. The commonest malignant primary tumours are sarcomas (95% of malignant primary cardiac tumours) and the commonest benign primary tumours are myxomas (mostly left sided in origin).1 Tumours which locally invade the heart are frequently lung and breast cancers, although hepatocellular carcinoma (HCC) can extend via the inferior vena cava into the right atrium and this is seen in approximately 1-4% of HCC.2 This phenomenon of extension is more commonly seen with renal cell carcinomas.

A 62-year-old man presented to Antrim Area Hospital with a short history of rapidly progressive dyspnoea. Four weeks previously, he had been able to walk 2 miles with no difficulty but this had reduced to 10-20 metres by the time of presentation. He had noticed increasing abdominal girth and ankle swelling. Of note, in his social history, was heavy smoking and a short history of right-sided knee pain.

Fig 1B: Axial CT image demonstrates a 3.5 cm right renal mass suspicious for a renal cell carcinoma (arrow).
alcohol intake over many years, although not in the last 10 years. D-Dimer was elevated at 573ng/ml so he proceeded to CT pulmonary angiogram to rule out a pulmonary embolus (PE). This demonstrated a small sub-segmental PE, but more significantly, a right atrial mass. This was further investigated with an echocardiogram which showed the mass extending through the tricuspid valve into the right ventricle. A large liver mass in segments IVa and VIII and extending into the middle hepatic vein, IVC and right atrium was then found on subsequent CT scanning. The atrial mass was felt to be an extension of an HCC (Figure 1).

The patient became increasingly dyspnoeic at rest and was transferred to the Royal Victoria Hospital, Belfast, where he underwent surgery to remove the atrial component of the tumour (Figure 2). Histology confirmed HCC. Following liaison with the hepatobiliary team, trans-catheter arterial chemoembolization (TACE) was felt to be the best treatment option. The patient was greatly improved following the surgery with symptoms of dyspnoea completely resolved and he was restored to normal mobility at the time of discharge (10 days post-surgery).

TACE involves delivery of chemotherapeutic agents and concurrent embolization which intends to deliver a highly concentrated dose of chemotherapy to tumour cells, prolong the contact time between the two and minimise systemic toxic effects. The concentration of chemotherapy achieved at the site of the tumour is thought to be around 100 times higher than systemic chemotherapy. The process is based upon the observation that agents delivered by the hepatic artery are preferentially taken up by hepatic tumours in comparison to normal liver parenchyma which is preferentially supplied from the portal venous system. Due to the dual blood supply of the liver, the treatment does not compromise healthy liver tissue. Survival rates vary after TACE but two randomised control trials showed 1 year survival between 57%-82%, and 2 year survival rates between 31%-63%, in patients with unresectable HCC undergoing TACE.

McNeice A1, Graham ANJ2, Trouton TG1
1 Department of Cardiology, Antrim Area Hospital
2 Department of Cardiothoracic Surgery, Royal Victoria Hospital, Belfast

REFERENCES
1. Shapiro LM. Cardiac tumours: diagnosis and management. Heart. 2001;85(2):218-22.
2. Vallakati A, Chandra PA, Frankel R, Shani J. Intra-atrial tumor thrombi secondary to hepatocellular carcinoma responding to chemotherapy. N Am J Med Sci. 2011;3(9):435-7.
3. Konno T. Targeting cancer chemotherapeutic agents by use of lipiodol contrast medium. Cancer. 1990;66(9):1897-903.
4. Ramsey DE, Geschwind JFH. Chemoembolization of unresectable hepatocellular carcinoma: a review. Appl Radiol. [Internet]. 2004;33(3). Available from: http://appliedradiology.com/articles/chemoembolization-of-unresectable-hepatocellular-carcinoma-a-review. Last accessed May 2016.

GENERAL SURGICAL TRAINING AND SURGICAL TRAINEE WORKING PATTERNS.

Editor,

Surgery, as a craft-based speciality, relies on hours of practical apprenticeship to acquire technical skills. To ensure these skills are developed, the Joint Committee on Surgical Training (JCST) has provided guidelines of indicative numbers for procedures required for Certificate of Completion of Training (CCT).

The New Deal and European Working Time Directive (EWTD) have reduced the amount of time doctors spend in work, increased staffing requirements for service provision and thereby diluted training. A new junior doctor contract to replace the New Deal has recently been the subject of much controversy across the United Kingdom. Additional emphasis has been placed on service provision and limited attention paid to the need for effective training. It has been suggested that to ensure optimal training with adequate time for exposure and to provide high quality patient care with increased continuity, it is necessary to return to a working...
week of approximately 65 hours.

The aim of this study was to examine the impact of changes in rota pattern and working hours on operative experience for specialty registrars in General Surgery.

The Ulster Medical Society grants to all users on the basis of a Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International Licence the right to alter or build upon the work non-commercially, as long as the author is credited and the new creation is licensed under identical terms.

Methods

Surgical registrar working patterns and operative experience were assessed in a district general hospital providing elective and emergency General Surgical services to a population of approximately 400,000. Over a period of 18 months, the working pattern changed progressively each six months, from a full shift rota (48 hours per week) to a 24-hour partial shift rota (56 hours per week), and ultimately to an on-call rota (65 hours per week). Trainee operative experience was compared for each rota pattern.

Results

When working on a 48 hour per week full shift rota, the Specialty Registrars in our unit had a reduced operative experience. Operative experience did not increase significantly when working a partial shift pattern, but was significantly increased by working a 65 hour per week on-call rota (Figure 1). Operative experience did not vary with grade of registrar.

Differing operative experience was evident for the total number of operations excluding endoscopy (p=0.005) and for individual procedures with regards to the number of cholecystectomies (p=0.020). There was no significant difference in the number of emergency cases (p=0.262), endoscopy (p=0.958), inguinal hernia repair (p=0.058), emergency laparotomy (p=0.266) or appendicectomy (p=0.055) between rota patterns although the number of doctors involved were small.

Results for each six-month period were extrapolated over a six-year period to allow for assessment of the training delivered by each rota pattern in the context of the JCST indicative numbers for CCT (Table 1). On a full shift rota, the total operative numbers required for CCT would not be achieved.

Conclusions

Concerns raised by the Royal Colleges of Surgeons and the Association of Surgeons in Training with regards to the impact of the EWTD on surgical training appear to have been well founded. Unless a national or specialty specific opt-out is made available, surgical trainees are likely to be disadvantaged by the EWTD when attempting to gain the experience necessary to achieve excellence. Contract negotiations should prioritise training within this context. We would encourage all possible attempts to allow doctors within craft specialties such as surgery to work outside the constraints of the EWTD in order to produce highly qualified consultants with the requisite experience to deliver high quality patient care.

Ethical approval for this study was not required.

R. Scott McCain, R. Stephen McCain, David A Mark, Kevin McCallion, Eamon J Mackle.

1 Department of Surgery, Craigavon Area Hospital, 64 Lurgan Road, Portadown
2 Department of Surgery, The Ulster Hospital, Upper Newtownards Road, Belfast, BT16 1RH.
3 Centre for Public Health, Queen’s University Belfast.

Corresponding author.

R. Scott McCain

e-mail: smccain01@qub.ac.uk

REFERENCES

1. Purcell Jackson G, Tarpley J. How long does it take to train a surgeon? BMJ. 2009; 339 (b4260).

2. Maxwell AJ, Crocker M, Jones TL, Bhagawati B; Papadopoulos MC, Bell BA. Implementation of the European Working Time Directive in neurosurgery reduces continuity of care and training opportunities. Acta Neurochir (Wien). 2010; 152(7): 1207-10.
3. Creswell B, Marron C, Hawkins W, Harrison E, Fitzgerald E, von Roon A. Optimising working hours to provide quality in training and patient safety: a position statement. London: The Association of Surgeons in Training; 2009.

4. Royal College of Surgeons of England. Surgeons and trainees. Surgical standards. Working practices. Workforce. Working Time Directive. Summary of personal responses to the European Working Time Regulations (EWTR) taskforce consultation. London: Royal College of Surgeons of England; 2010.

DUAL POSTERIOR LIP AUGMENTATION DEVICES FOR RECURRENT INSTABILITY OF A CHARNLEY TOTAL HIP REPLACEMENT

Editor,

An 85-year-old female presented with a posterior dislocation of a right Charnley total hip replacement (THR) despite previous posterior lip augmentation. Past medical history included myocardial infarction, left ventricular dysfunction, dual antiplatelet therapy, atrial fibrillation and Alzheimer’s disease.

Her original arthroplasty was performed in 1983 via a posterior approach. The acetabular component was revised in 1998 due to dislocations associated with acetabular loosening, but the femoral component remained well fixed. Due to continuing instability, a posterior lip augmentation device (PLAD) was inserted in 2004 following a third dislocation. The hip remained stable until 2011 when a further dislocation occurred. Radiographs revealed a fracture of the PLAD through the middle screw hole. This was revised with a new 22mm PLAD to the postero-superior rim of the acetabular component, which was noted to have significant wear.

The patient had a further posterior dislocation following a fall in March 2015 (Figure 1).

An open reduction revealed significant wear of the acetabular component and inferior aspect of the PLAD with inferior instability on flexion and internal rotation. The femoral head had multiple abrasions from rubbing on the exposed metal of the PLAD, with significant metal debris in the surrounding soft tissues. The femoral and acetabular components were well fixed. Due to the significant co-morbidities, revision arthroplasty was not felt to be appropriate by the multidisciplinary team. A second 22mm PLAD was placed on the postero-inferior aspect of the acetabular component, overlapping the two inferior holes of the existing PLAD. This appeared to address the issue of inferior instability (Figure 2).

The approach was by partial re-incision of the previous scar with an operative time of 49 minutes. There were no peri-operative complications.

The PLAD was developed as an alternative to revision surgery for the management of recurrent posterior instability following Charnley THRs in the setting of well fixed, correctly positioned components1. It consists of a contoured ultra-high molecular weight polyethylene bearing and stainless steel backing. This is attached to the posterior aspect of the existing acetabular component via five 4.5mm cortical screws. Previous studies have reported dislocation rates following PLAD insertion ranging from 0-10% and revision rates of between 1.3-3.7%1-5. Charlwood et al2 reported that operative time, blood loss, and hospital stay were all significantly reduced in patients treated with PLAD when compared to revision surgery with no significant difference in Oxford Hip Scores in short and long term follow-up. Campbell et al3 reported two cases or recurrent dislocations past a PLAD which were subsequently treated with excision arthroplasty. Yeung et al5 have advocated the use of a postero-lateral and antero-medial augmentation to manage recurrent dislocations instead of a constrained liner. Interestingly, the first PLAD in this case had fractured through a screw hole which has only been documented once before3.

We propose that in the setting of recurrent dislocation...
past a PLAD the use of a second PLAD to supplement the augmentation over a wider circumference can be successful in patients who are of low functional demand.

Mr Kevin J Donnelly, Mr Graham Finlayson, Mr Chris Andrews
Department of Trauma and Orthopaedic Surgery, Royal Victoria Hospital, 274 Grosvenor Road, Belfast, Northern Ireland BT12 6BA
Correspondence addressed to:
Mr Kevin Donnelly
Email: kevdonnelly@hotmail.com

REFERENCES
1. Charlwood AP, Thompson NW, Thompson NS, Beverland DE, Nixon JR. Recurrent hip arthroplasty dislocation: good outcome after cup augmentation in 20 patients followed for 2 years. Acta Orthop Scand. 2002 Oct;73(5):502-5
2. Charlwood AP, Thompson NW, Brown JG, Nixon JR. The Belfast Posterior Lip Augmentation Device (PLAD) in the management of recurrent posterior dislocation following primary total hip arthroplasty. J Bone Joint Surg [Br] Orthop Proc. [Online]. 2002; 84-B(Suppl II): 154. Available from: http://www.bjjprocs.boneandjoint.org.uk/content/84-B/SUPP_II/154.2. Last accessed May 2016.
3. Campbell D, Muthusamy K, Sturdee S, Finlayson D, Stone M. The Posterior Lip Augmentation Device for Recurrent Dislocation. J Bone Joint Surg [Br] Orthop Proc. [Online]. 2001; 84(Suppl II): 154. Available from: http://www.bjjprocs.boneandjoint.org.uk/content/84-B/SUPP_II/154.3
4. McConway J, O’Brien S, Doran E, Archbold P, Beverland D. The use of a posterior lip augmentation device for a revision of recurrent dislocation after primary cemented Charnley/Charnley Elite total hip replacement: results at a mean follow-up of six years and nine months. J Bone Joint Surg Br. 2007;89(12):1581-5.
5. Yeung D, Rourke K, Pradhan N. Use of two posterior lip augmentation devices for recurrent total hip arthroplasty dislocation in select patients. Ann R Coll Surg Engl. 2013 Mar; 95(2): 156

MUTYH ASSOCIATED POLYPOSIS [MAP] - A ‘LESSER KNOWN’ POLYPOSIS SYNDROME!
Editor,

Familial Adenomatous Polyposis (FAP) and Hereditary Non-Polyposis Colorectal Cancer (HNPCC) are well recognised syndromes that increase the risk of colorectal carcinoma (CRC). First described in 2002, MUTYH associated polyposis (MAP) results in colorectal polyposis and increased risk of malignancy, with a lifetime risk of CRC of >90%1,2. Like FAP and HNPCC, extracolonic manifestations can occur in MAP including duodenal and gastric polyps. Similarity to other polyposis syndromes may lead to diagnostic confusion and underrecognition of MAP in clinical practice.

To illustrate this we report the case of a 67 year old female, who was first diagnosed with colorectal cancer and synchronous tubulovillous adenoma aged 40. Consequently she underwent a subtotal colectomy with ileorectal anastomosis and lifelong rectal surveillance in 1988. Following this, she was ascribed the diagnosis of HNPCC leading to annual surveillance colonoscopy and identification of further rectal polyps. In 2008, an oesophagogastroduodenoscopy (OGD) for dyspepsia identified extensive periampullary polyposis, since managed by endoscopic excision and annual surveillance OGDS. In October 2014, following investigations by the clinical geneticists, a diagnosis of MAP was made. Despite the impressive periampullary carpeting (Figure 1) surveillance biopsies have shown stable low grade dysplasia. Endoscopic ultrasonic surveillance has been enlisted to ensure confinement of disease to the mucosa.

Fig 1. Extensive Periampullary Polyposis in a patient with MUTYH associated polyposis (Blue arrow indicates location of polyposis, around the ampulla of Vater. Image taken in second part of the patient’s duodenum)

MAP is an autosomal recessive disorder characterised by biallelic germline mutations in the MUTYH gene (chromosome 1p34.1). It has been estimated that 2% of the British population are heterozygous for a MUTYH mutation; however no accurate figures exist for the prevalence of MAP. MAP is usually diagnosed at an older age than classical FAP (mean 45 to 56 years), similar to attenuated FAP3.

Concomitant neoplasms are common in colonic polyposis syndromes and like FAP, duodenal polyposis is a feature of MAP. The prevalence of duodenal polyposis in MAP is 17-25%, as compared to 90% in FAP, with a lifetime risk of duodenal cancer of 4%. Gastric adenomas and fundic gland polyps are also described in MAP. Like HNPCC, cutaneous sebaceous tumours can also occur4.

The propensity for polyposis of the periampullary region in FAP and MAP is noteworthy. Bile acids can induce production of DNA-damaging reactive oxygen species. This is of particular relevance to MAP as the MUTYH gene is responsible for repair of oxidative DNA damage1. Furthermore, bile removed from FAP patients has enhanced mutagenic capacity as compared to bile from non-FAP patients. This has not yet been demonstrated in patients with MAP5.

This case highlights the need for awareness of this rarer polyposis disorder that increases the risk of gastro-intestinal
Letters

209

and non-gastrointestinal tumours. The phenotypic spectrum of MAP may lead to diagnostic confusion with other syndromes such as FAP, HNPCC and serrated polyposis syndrome or failure to recognise this as an inheritable condition.

A diagnosis of MAP should be considered when: there is a family history of CRC suggestive of a condition inherited in an autosomal recessive manner; CRC with a c.34G>T mutation in codon 12 of the KRAS gene; multiple synchronous colorectal polyps, including hyperplastic/serrated polyps, (typically >10, or 1-10 prior to age 40) in the absence of a mutation in the APC gene. For those diagnosed with MAP, the British Society of Gastroenterology recommend biannual surveillance colonoscopy from the age of 25, and 3–5 yearly OGDs.

Cathal Hannan¹, Paul Kelly¹, Barry Clements¹
¹ Upper GI Surgery Unit, BCH
² Department of Pathology, RVH.

Contact email – cathalhannan@icloud.com

REFERENCES

1. Al-Tassan, Chmiel, Maynard, Fleming, Livingston,Williams et al.Inherited variants of MYH associated with somatic G:C to T:A mutations in colorectal tumors. Nat Genet. 2002; 30: 227 – 232.
2. Cairns SR, Scholefield JH, Steele RJ, Dunlop MG, Thomas HJ, Evans GD et al; British Society of Gastroenterology; Association of Coloproctology for Great Britain and Ireland. Guidelines for colorectal cancer screening and surveillance in moderate and high risk groups (update from 2002). Gut. 2010; 59(5):666-89.
3. Lindor NM. Hereditary colorectal cancer: MYH-associated polyposis and other newly identified disorders. Best Pract Res Clin Gastroenterol. 2009; 23(1):75-87.
4. Vogt S, Jones N, Christian D, Engel C, Nielsen M, Kaufmann A et al. Expanded extracellular tumor spectrum in MUTYH-associated polyposis. Gastroenterology. 2009; 137(6):1976-85.
5. Slupska MM, Baikalov C, Luther WM, Chiang JH, Wei YF, Miller JH. Cloning and sequencing a human homolog (hMYH) of the Escherichia coli mutY gene whose function is required for the repair of oxidative DNA damage. J Bacteriol. 1996; 178(13):3885-92.
6. Scates DK, Spigelman AD, Nugent KP, Phillips RK, Venitt S. DNA adducts, detected by 32P-postlabelling, in DNA treated in vitro with bile from patients with familial adenomatous polyposis and from unaffected controls. Carcinogenesis. 1993; 14:1107–1110

THE COST OF HISTORY REPEATING ITSELF: A REVIEW OF LABORATORY INVESTIGATIONS ON THE MANAGEMENT OF TONSILLITIS AND QUINSY IN THE SECONDARY CARE SETTING.

Editor,

Tonsillitis and Quinsy are common acute admissions into hospitals throughout Northern Ireland. Patients are frequently referred from the community due to worsening symptoms or failure to respond to initial treatments. While there are guidelines¹³ for management in the community, there are no specific guidelines for those admitted into secondary care facilities and as such, management and investigations are often guided by anecdotal and historical practice rather than being evidence based. To explore this, we assessed the current investigative practice for patients admitted to Antrim Hospital with tonsillitis and quinsy during 2014.

Data was collected for patients admitted during 2014 with a diagnosis of sore throat, Tonsillitis, Glandular Fever, Infectious Mononucleosis, Quinsy or peri-tonsillar abscess including the length of stay, diagnosis, type of investigations performed, and the type of treatment received by the patients.

Sixty-nine patients accounting for 72 admission episodes in 2014 were assessed. The average length of stay of patients was 1.34 days. Fifty-seven percent of admissions had investigations such as throat culture or monospot testing with 8% having both. A total of 18 throat cultures were performed, but only 83.3% came back with a positive result requiring an average of 3.61 days for colonisation. Thirty-five monospot tests were performed with only 8.8% being positive. Antibiotics were prescribed in 68 of the cases admitted to hospital, with 10.8% receiving corticosteroids, and all patients presenting with a quinsy undergoing surgical drainage.

Our data suggests that additional laboratory investigations have a limited impact on the acute management of patients presenting with tonsillitis or quinsy given the short period of time that the patients spend in hospital (p: 0.597). Our figures estimate that it would cost £911.76 to detect a positive monospot test and £450 to grow a positive culture for every 100 patients admitted with these ailments.

The potential of complications and subsequent litigation has driven clinicians to perform multiple investigations without considering the effect on overall patient management and the cost to the healthcare system. Throat culture has a wide sensitivity range between 40% - 95%¹⁴,¹⁵ and is restricted by the temporal factor of time, and as such will have limited effect on immediate management. While monospot testing can help guide patient management (such as antibiotic avoidance and restrictions on subsequent contact sports), a blanket policy for its routine use is ill advised as only 8.8% showed a positive result in this series. Often, routine laboratory findings and clinical factors will point to a diagnosis of Infectious Mononucleosis and we would suggest the monospot test be used to confirm this suspicion rather than to make a diagnosis.

The authors suggest that tonsillitis and quinsy are primarily clinical diagnoses and that additional laboratory testing is of limited use. Given that the vast majority of these patients are successfully treated with a 24-hour course of intravenous antibiotics, allocation of funding to short stay facilities may be a better use of resources than the excessive investigation that we currently carry out.

The authors have no conflict of interest.

Mr Mohd Afiq Mohd Slim, Mr Philip Robert Bell, Mr Marcel Valko
Department of Otorhinolaryngology, Antrim Area Hospital, 45, Bush Road, Antrim, BT41 2RL, Northern Ireland.

Correspondence to: bellpr@hotmail.co.uk
REFERENCES:

1. SIGN National Clinical Guideline; 117. Management of sore throat and indications for tonsillectomy. Edinburgh: Scottish Intercollegiate Guidelines Network; 2010. Available from: http://www.sign.ac.uk/pdf/sign117.pdf.

2. ESCMID Sore Throat Guideline Group, Pelucchi C, Grigoryan L, Galeone C, Esposito S, Huovinen P, Little P, et al. Guideline for the management of acute sore throat. Clin Microbiol Infect. 2012;18(Suppl 1):1-28.

3. Shulman ST, Bisno AL, Clegg HW, Gerber MA, Kaplan EL, Lee G, et al. Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. Clin Infect Dis. 2012;55(10):1279-82.

4. Wakode PT, Gawarle SH, Joshi SV, Bajoriya R. Throat swab culture & sensitivity reports - an overview. Indian J Otolaryngol Head Neck Surg. 2003;55(2):76-80.

5. Choby BA. Diagnosis and treatment of streptococcal pharyngitis. Am Fam Physician. 2009;79(5):383-90.