

## Letters

### Cholethorax following Percutaneous Transhepatic Biliary Drainage

Editor,

We report the case of a 51 year old man who developed the unusual complication of a bilious pleural effusion, or 'Cholethorax' following percutaneous transhepatic biliary drainage.

**Case Report:** A 51 year old man with locally advanced gastric adenocarcinoma presented with painless jaundice one year following the completion of palliative chemotherapy. Laboratory investigations revealed a bilirubin level of 299  $\mu\text{mol/L}$  with AST 117 U/L, ALT 134 U/L, GGT 2447 U/L, ALP 2159 U/L and an ultrasound of abdomen confirmed the presence of biliary obstruction. Percutaneous Transhepatic Cholangiography (PTC) was arranged as the presence of a gastric tumour precluded an approach using Endoscopic Retrograde Cholangiopancreatography (ERCP). The right hepatic duct was cannulated and contrast injected, demonstrating a complicated stricture of the common bile duct. An internal-external biliary drain was then inserted across this stricture to decompress the biliary tree and the position of the drain is shown in figure 1. Three days after the PTC our patient complained of severe right sided pleuritic chest pain and shortness of breath. A chest x-ray revealed right basal atelectasis and provisional diagnoses of a lower respiratory tract infection and possible pulmonary embolus were offered.



Fig 1.

Over the next 48 hours the patient became increasingly dyspnoeic, with signs of a right sided pleural effusion on

examination, and so a repeat chest radiograph was carried out (fig 2). The output of bile into the drainage bag had dramatically decreased and the bilirubin level had risen further to 387  $\mu\text{mol/L}$ . A pleural aspiration was performed which yielded dark brown pleural aspirate with a bilirubin level of 766  $\mu\text{mol/L}$  (fig 3).

A diagnosis of a bilious pleural effusion (Cholethorax) as a complication of percutaneous transhepatic biliary drainage was made. The insertion of a 28F chest drain and rapid drainage of the bilious pleural fluid provided immediate relief of the shortness of breath and pleuritic chest pain. A further PTC was carried out urgently and three self-expanding metal stents were inserted across the complicated biliary stricture to provide adequate biliary drainage.



Fig 2.

**Discussion:** PTC and biliary drainage is used for the management of malignant biliary obstruction in cases where ERCP is inappropriate or has been unsuccessfully attempted. It involves the percutaneous cannulation of either hepatic duct followed by placement of a biliary drain to decompress the biliary tree and subsequent insertion of a stent during the initial procedure or a number of days later. During biliary cannulation it may be necessary to traverse the pleural cavity to gain access to either hepatic duct. An internal-external biliary drain is inserted consisting of a pig tail drain with a hole at the tip to allow the bile to exit into the duodenum and a number of side-holes along the distal length. These side-holes should be placed inside the common bile duct (Fig 1) to allow entry of the bile which then drains internally into the duodenum or externally into a drainage bag.

In our patient's case the drainage catheter became dislodged with the tip remaining in the right hepatic duct while the side-holes formed a direct communication with the pleural cavity. This occurred due to the trans-pleural approach taken during the PTC and as a result bile rapidly drained into the pleural cavity causing a 'Cholethorax'. Bile is an intense chemoirritant and so extensive pleural inflammation was established which also allowed the chest drain to be removed relatively quickly as it essentially caused a pleurodesis



Fig 3.

to occur. Bile also provides a good medium for bacterial growth and so infective sequelae often occur in the setting of a cholethorax.

Biliary pleural fistulas and the formation of bilious pleural effusions are known complications of hepatic trauma<sup>1,2</sup>, parasitic liver disease<sup>3</sup> and development of a subphrenic abscess in the setting of biliary obstruction. Iatrogenic causes include biliary stent migration<sup>4</sup>, radio-frequency ablation<sup>5</sup> and following cholecystectomy<sup>6</sup> and liver biopsy<sup>7</sup> However, it is the increasing use of percutaneous biliary drainage which has led to the greatest number of cases.<sup>8-10</sup> For a Cholethorax to arise disruption of the pleural space needs to have occurred and this may not necessarily be obvious during the procedure. Rapid thoracentesis, correction of the cause of the fistula, adequate analgesia and the treatment of infective sequelae are essential in the management of this group of patients.

The authors have no conflict of interest

Richard C Turkington,\* *SpR, Oncology*

Julian J Leggett<sup>+</sup>, *Consultant in Respiratory Medicine*

Jane Hurwitz, *SpR Oncology*

Martin M Eatock, *Consultant in Medical Oncology*

Departments of Oncology and Respiratory Medicine<sup>+</sup>, Belfast City Hospital, 51 Lisburn Road, Belfast, BT9 7AB, United

Kingdom

\* rcturkington@hotmail.com

#### REFERENCES:

1. Franklin DC, Mathai J. Biliary pleural fistula: a complication of hepatic trauma. *J Trauma* 1980;**20**(3):256-62.
2. Bamberger PK, Stojadinovic A, Shaked G, Golocovsky M. Biliary-pleural fistula presenting as a massive pleural effusion after thoracoabdominal penetrating trauma. *J Trauma* 1997;**43**(1):162-3.
3. Amir-Jahed AK, Sadrieh M, Farpour A, Azar H, Namdaran F. Thoracobilia: a surgical complication of hepatic echinococcosis and amebiasis. *Ann Thorac Surg* 1972;**14**(2):198-205.
4. Dasmahapatra HK, Pepper JR. Bronchopleurobiliary fistula. A complication of intrahepatic biliary stent migration. *Chest* 1988;**94**(4):874-5.
5. Liberale G, Delhay M, Ansay J, Houben JJ, Coppens E, Gelin M, et al. Biliary pleural fistula as a complication of radiofrequency ablation for liver metastasis. *Acta Chir Belg* 2004;**104**(4):448-50.
6. Lehur PA, Guiberteau-Canfrere V, Bury A, Cloarec D, Le Borgne J. ["Cholethorax" revealing injury to the common bile duct after celioscopic cholecystectomy]. [French] *Ann Chir* 1992;**46**(5):450-2.
7. Pisani RJ, Zeller FA. Bilious pleural effusion following liver biopsy. *Chest* 1990;**98**(6):1535-7.
8. Armstrong CP, Taylor TV. Intrapleural leakage of bile complicating percutaneous transhepatic drainage of the obstructed biliary tree. *J R Coll Surg Edinb* 1982;**27**(5):308-9.
9. Herschman Z, Amin D, Lehrfield A. Bilious pleural effusion as a complication of attempted percutaneous biliary drainage. *Crit Care Med* 1991;**19**(1):128-9.
10. Strange C, Allen ML, Freedland PN, Cunningham J, Sahn SA. Biliopleural fistula as a complication of percutaneous biliary drainage: experimental evidence for pleural inflammation. *Am Rev Respir Dis* 1988;**137**(4):959-61.

#### Diffuse sclerosing variant of papillary thyroid carcinoma – a rare cause of goitre in a young patient

Editor,

Papillary thyroid carcinoma is the most common thyroid malignancy. We report a case of a rare variant - diffuse sclerosing papillary thyroid carcinoma (DSPC).

**Case History:** An 18 year old girl presented with a smooth symmetrical goitre. She was clinically euthyroid and had no palpable cervical lymph nodes. Thyroid function tests and anti-thyroid peroxidase level were normal. Ultrasound scan of thyroid showed marked nodular enlargement of the entire gland in keeping with a multinodular goitre. A hypoechoic 1cm nodule was identified at the right lobe which was found to be 'cold' on radio-isotope scanning. A fine needle aspiration of this 'cold' nodule was reported as papillary carcinoma.

She was booked for total thyroidectomy. At surgery she had an enlarged thyroid, with a gross appearance in keeping with a thyroiditis or lymphoma. Frozen section confirmed papillary carcinoma. The gland was hard and gritty. Several local lymph nodes were also excised. Post-operative recovery was uneventful.

Sectioning revealed a diffusely firm, white, gritty gland (fig 1). Histopathology showed this to be the rare diffuse sclerosing

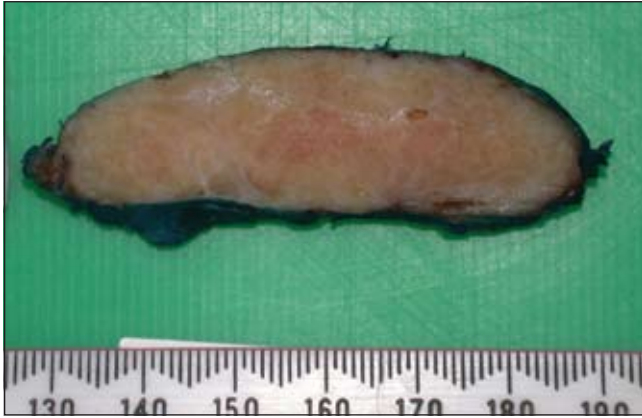


Fig 1. Sclerotic hemisection of thyroid lobe

variant of papillary thyroid carcinoma. Scattered islands of tumour tissue, squamous metaplasia, stromal sclerosis, heavy lymphoplasmic infiltrate and abundant psammoma bodies were found diffusely throughout both lobes and through the capsule (fig 2). All excised lymph nodes contained metastatic carcinoma.

She underwent radioablative therapy followed by replacement levothyroxine. There was no residual uptake on subsequent <sup>123</sup>Iodine isotope scanning.

**Discussion:** First described in 1985, diffuse sclerosing papillary carcinoma of the thyroid (DSPC) is a rare variant malignancy, recently reported to account for 0.8% of papillary thyroid carcinomas.<sup>1,2</sup> Patients present with a diffuse goitre and are mostly clinically euthyroid, but can also be hypothyroid or hyperthyroid. It occurs most frequently in young females and may be mistaken clinically for benign disease particularly thyroiditis.<sup>3-5</sup> Most patients have lymph node metastases at the time of diagnosis and lung metastases are common.<sup>3</sup> Cerebral metastases have also been reported.<sup>6</sup>

The presence of several pathological characteristics is diagnostic: diffuse firm enlargement of the thyroid gland, scattered islands of papillary carcinoma, extensive lymphatic permeation and lymphocytic infiltration, squamous

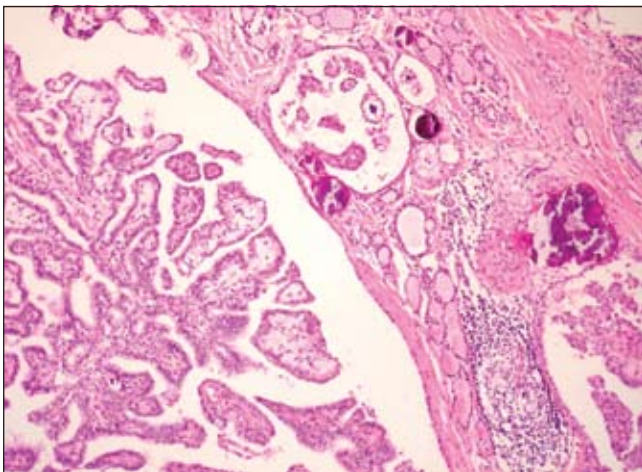


Fig 2. Papillary carcinoma, with psammoma bodies and squamous metaplasia. Sclerosis and chronic inflammation, to right of field. (x 200)

metaplasia, sclerosis and numerous psammoma bodies.<sup>7</sup> Detection of abundant psammoma bodies on ultrasonography may provide pre-operative evidence of DSPC.<sup>4</sup> This could facilitate improved surgical planning for a technically challenging thyroidectomy.

Early studies suggested that DSPC had a poorer prognosis than classical papillary carcinoma due to its aggressive nature with frequent lymph node and distant metastases at the time of presentation. It had also been reported that eradication required a more aggressive therapeutic approach.<sup>3</sup> However, more recent studies suggest that DSPC patients have a similar prognosis and that the treatment should be that for classical papillary thyroid carcinoma i.e. radical surgery, radio-iodine ablation and/or external radiotherapy.<sup>2,8</sup>

There are potential pitfalls which may delay the diagnosis of DSPC. In this case, the clinical presentation, biochemical, serological and initial radiological findings were all indicative of benign pathology. FNA indicated malignancy leading to surgery demonstrating its importance in the diagnosis of DSPC. As metastases are frequently present it is therefore important to consider this rare malignancy when investigating a goitre in a young patient.

The authors have no conflict of interest.

\*Kevin McElvanna, *Senior House Officer*

Grainne McCusker, *Consultant*

Ivan Stirling, *Consultant*

\*Department of General Surgery, Craigavon Area Hospital, 68 Lurgan Road, Portadown, BT63 5QQ.

kevinmcelvanna@doctors.org.uk

#### REFERENCES:

- Vickery AL, Carangiu ML, Johannessen JV, Sobrinho-Simoes M. Papillary carcinoma. *Semin Diag Pathol* 1985;2:90-100.
- Chow SM, Chan JK, Law SC, Tang DL, Ho CM, Cheung WY, et al. Diffuse sclerosing variant of papillary thyroid carcinoma – clinical features and outcome. *Eur J Surg Oncol* 2003;29(5):446-9.
- Carangiu ML, Bianchi S. Diffuse sclerosing variant of papillary thyroid carcinoma. Clinicopathologic study of 15 cases. *Am J Surg Pathol* 1989;13(12):1041-9.
- Fujimoto Y, Obara T, Ito Y, Kodama T, Aiba M, Yamaguchi K. Diffuse sclerosing variant of papillary carcinoma of the thyroid. Clinical importance, surgical treatment and follow-up study. *Cancer* 1990;66(11):2306-12.
- Wu PS, Leslie PJ, McLaren KM, Toft AD. Diffuse sclerosing papillary carcinoma of thyroid: a wolf in sheep's clothing. *Clin Endocrinol (Oxf)* 1989;31(5):535-40.
- Imamura Y, Kasahara Y, Fukuda M. Multiple brain metastases from a diffuse sclerosing variant of papillary carcinoma of the thyroid. *Endocr Pathol* 2000;11(1):97-108.
- Kumarasinghe MP. Cytomorphologic features of diffuse sclerosing variant of papillary carcinoma of the thyroid. A report of two cases in children. *Acta Cytol* 1998;42(4):983-6.
- Albareda M, Puig-Domingo M, Wengrowicz S, Soldevila J, Matias-Guiu X, Caballero A, et al. Clinical forms of presentation and evolution of diffuse sclerosing variant of papillary carcinoma and insular variant of follicular carcinoma of the thyroid. *Thyroid* 1998;8(5):385-91.

### A case of primary lung malignancy presenting as pericardial effusion with associated localised Epstein-Barr virus infection or persistence.

Editor,

Acute pericarditis and pericardial effusion has many causes including infections, malignancy, collagen vascular disease, autoimmune diseases, uraemia, myocardial infarction, trauma, surgery, medications and hypothyroidism. We report a rare case in which pericardial fluid was positive for both malignant adenocarcinoma cells and PCR positive for Epstein-Barr virus.

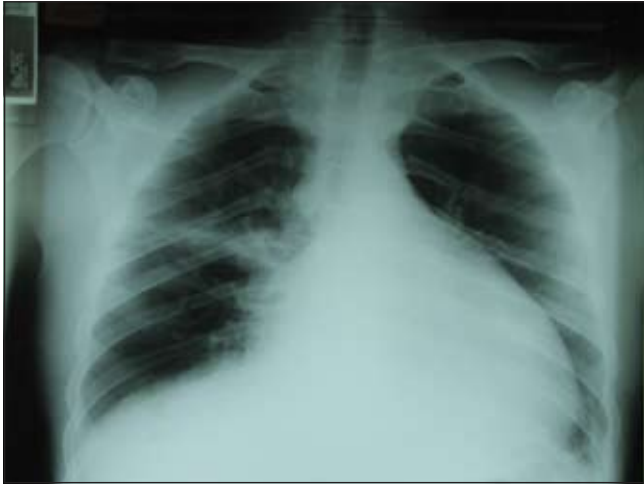


Fig 1.

**Case report:** We report the case of 44 year old male mechanical engineer admitted with two weeks history of lethargy, malaise, vomiting, breathlessness and two episodes of syncope. There was no previous history of cardiorespiratory disease and he was a non smoker. On examination he was tachycardic, hypotensive and had elevated jugular venous pressure. On auscultation heart sounds were muffled with no murmur or pericardial rub heard. Chest X-ray showed cardiomegaly, (Fig 1), whilst ECG showed sinus tachycardia with no significant ST or T wave changes. A transthoracic echocardiogram showed large pericardial effusion with right atrial and ventricular collapse (Fig 2). These features suggest he was in cardiac tamponade. A pigtail catheter was inserted and 1550ml of frank haemorrhagic fluid was drained subxiphoidally.

Pericardial fluid was analysed as per guidelines for diagnosis and management of pericardial diseases of European Society of cardiology. Pericardial fluid was positive for Epstein-Barr virus on polymerase chain reaction while polymerase chain reaction for Epstein-Barr virus from leucocytes in circulation and IgM antibodies for Epstein-Barr virus antigens were negative, consistent with localised pericardial presence of Epstein-Barr virus<sup>1</sup>.

Further study on pericardial fluid revealed malignant epithelial cells with morphology suggestive of adenocarcinoma. Immunohistochemistry was positive for CK-7, CEA, TTF1, EMA, and weakly positive for CK5, CK-6, and Ber EP4 and negative for HMBE1, PSA, HMB 45 and CK-20. In summary TTF-1 and CK-7 being positive was highly specific

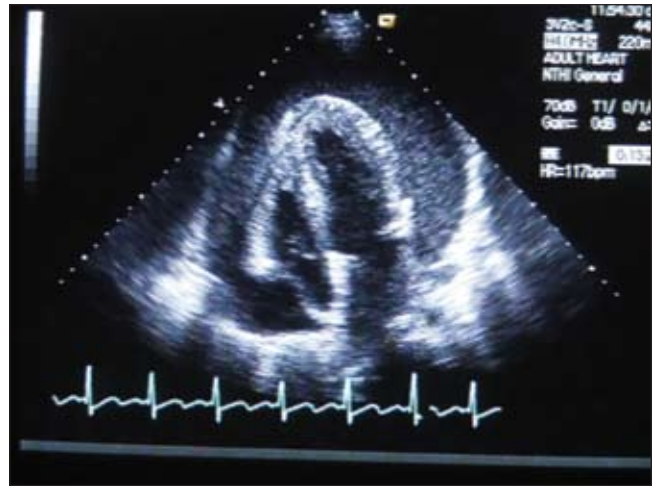


Fig 2.

of lung primary<sup>2</sup>. TTF-1 is a lineage marker for tumour arising from peripheral airway or alveolar epithelium and has no prognostic relevance<sup>3</sup>.

Chest X-ray after therapeutic drainage of pericardial fluid showed two opacities in right middle and lower zones. A computerised tomographic scan of the chest and abdomen revealed marked right hilar lymphadenopathy and a further nodal mass just below the right carina. A 1.7cm speculated mass was seen in the right upper lobe adjacent to the horizontal fissure with a little fluid in the fissure and surrounding consolidation. There were bilateral pleural effusions and further pleural-based lesion in the right lower lobe. Based on clinical and radiological findings the tumour was staged T1 N1 M1. An incidental finding of the presence of pulmonary thromboembolic disease bilaterally extending into 3<sup>rd</sup> pulmonary division was also seen and hence the patient was started on a therapeutic dose of low molecular weight heparin. The patient was referred to oncology for further treatment

**Discussion:** Pericardial inflammation and effusion due to Epstein-Barr virus infection is rarely reported. There are a few case reports of Epstein-Barr virus causing adenocarcinoma lung especially in Asian populations<sup>4</sup>. Malignant pericardial effusion and localised presence of Epstein-Barr virus can be explained either by malignancy secondary to Epstein-Barr virus infection<sup>5,6</sup> or due to co-existing infection, thus making this a very rare case. It has been proposed that viral load estimation from malignant cell and non-malignant cell would have proved the causative role of Epstein-Barr virus<sup>4</sup>. As identification of causative role of Epstein-Barr virus has no implication in treatment or prognosis tissue biopsy and viral load estimation was not attempted.

The authors have no conflict of interest

\*Vivek Kodoth<sup>1</sup> Senior House Officer

Joe Leyon, Foundation Year 1

Vincent Moohan, Consultant Cardiologist,

Whiteabbey Hospital, Doagh Road, Newtownabbey, United Kingdom

<sup>1</sup>Flat 4, Ashley Court, Ashley Avenue, Belfast BT9 7BX

vivekkodoth@btinternet.com

## REFERENCES:

1. Brengel-Pesce K, Morand P, Schmuck A, Bourgeat MJ, Buisson M, Barges G, *et al.* Routine use of real-time quantitative PCR for laboratory diagnosis of Epstein-Barr virus infection. *J Med Virol* 2002;**66**(3):360-9.
2. Reis-Filho JS, Carrilho C, Valenti C, Leitao D, Ribeiro CA, Ribeiro SG, *et al.* Is TTF1 a good immunohistochemical marker to distinguish primary from metastatic lung adenocarcinomas? *Pathol Res Pract* 2000;**196**(12):835-40.
3. Stenhouse G, Fyfe N, King G, Chapman A, Kerr KM. Thyroid transcription factor 1 in pulmonary adenocarcinoma. *J Clin Pathol* 2004;**57**:383-7.
4. Zhang L, Liu H, Wang Z. The role of Epstein-Barr virus infection and and infective copy number in pulmonary carcinogenesis. [Chinese]. *Zhonghua Bing Li Xue Za Zhi* 1995;**24**(3):132-5.
5. Huber M, Pavlova B, Muhlberger H, Hollaus P, Lintner F. Detection of the Epstein-Barr virus in primary adenocarcinoma of the lung with Signet-ring cells. *Virchows Arch* 2002;**441**(1):25-30.
6. Grinstein S, Preciado MV, Gattuso P, Chabay PA, Warren WH, DeMatteo E, *et al.* Demonstration of Epstein-Barr virus in carcinomas of various sites. *Cancer Res* 2002;**62**(17):4876-8.

### Merkel cell carcinoma of the cheek: Diagnosis in an elderly woman

Editor,

Merkel cell carcinoma (MCC) is an uncommon primary neuroendocrine skin tumour. It is commonly seen in the elderly, on the sun exposed areas which can mimic benign or less malignant skin tumours. We report a case of Merkel cell carcinoma of cheek in an elderly woman which was initially treated as a boil. This highlights the importance of considering this tumour in the differential diagnosis of head and neck skin lesions as it is fatal if not diagnosed and treated early.

**Case History:** A 93 year old woman was referred, with a left cheek swelling, progressively increasing in size over a period of two to three months. An initial clinical diagnosis of boil necessitated the administration of two courses of antibiotics by her GP, before attending the general surgical clinic. On examination a single, firm, non-tender, purple pink swelling with superficial central ulceration was noted on the left cheek, measuring about 4x4cm size with well-defined margins and normal surrounding skin (Figure). The histology of a biopsy from an ulcer edge was consistent with Merkel cell carcinoma. Immunohistochemistry confirmed the diagnosis. Unfortunately this patient died two months after the diagnosis.

**Discussion:** High index of suspicion is needed to diagnose some of the rare skin lesions. Merkel cell carcinoma of the skin is one of those uncommon, aggressive, neuroendocrine, cutaneous tumour most commonly found in head and neck region. It is a rare neuroendocrine tumour of the skin accounting for less than 1% of cutaneous malignancies, usually presents as red, purple or violaceous firm painless nodule or plaque. It is often mistaken for more common skin tumours because of its rarity.

Diagnosis can be made with histology alone and electron microscopy is encouraged as histologically it can resemble many other neoplastic processes. Immunohistochemistry is required for the definitive diagnosis of Merkel cell carcinoma. In our case malignant cells stained positive for CAM 5.2, CK-20 and chromogranin and negative for S100 and LCA

(leukocyte common antigen).

Surgical treatment is the corner stone of the treatment. Wide local excision with a clearance margin of 3-5 cm is commonly recommended<sup>1-4</sup>. It is widely accepted that patients with regional node metastasis should undergo lymph node dissection. Adjuvant radiation therapy is generally recommended for primary site and lymph node basin in stage I and II disease. It may also be used as the only treatment for patients who are not fit for any surgical resection. Chemotherapy is generally reserved for the stage III disease. And no chemotherapeutic protocol has been able to achieve a significant increase in survival rate<sup>3</sup>

**Conclusion:** Some distinctive features of presentation are



Fig 1.

red, violaceous, intradermal nodule in sun exposed areas. High index of suspicion is needed at first presentation as it frequently proves lethal despite various multimodal therapies if not diagnosed early. It is clear from the available data that early diagnosis and wide local excision will prolong the survival.

The authors have no conflict of interest.

Department of General Surgery, Causeway Hospital  
Coleraine, BT52, 1WP, United Kingdom

Jagannath Sherigar\* *SpR General Surgery*

Susim Kumar *SHO General Surgery*

Jawed Wali *Locum Consultant General Surgeon*

\*9 Knockbracken Park, Coleraine, BT52 1WP

jsmala@yahoo.com

## REFERENCES:

1. Koljonen VS. Merkel cell carcinoma. *World J Surg Oncol* 2006;**4**(1):7[Epub ahead of print].
2. Koljonen V, Bohling T, Grantho G, Tukiainen E. Merkel cell carcinoma: a clinicopathological study of 34 patients. *Eur J Surg Oncol* 2003;**29**(7):607-10.
3. Allen PJ, Bowne WB, Jaques DP, Brennan MF, Busam K, Coit DG. Merkel cell carcinoma: prognosis and treatment of patients from a single institution. *J Clin Oncol* 2005;**23**(10):2300-9.
4. Akhtar S, Oza KK, Wright J. Merkel cell carcinoma: report of 10 cases and review of the literature. *J Am Acad Dermatol* 2000;**43**(5):755-67.

### An Unusual Cause Of Pyrexia Of Unknown Origin In An 81 Year Old Lady

Editor,

We report a rare cause of pyrexia of unknown origin. An 81-year old woman presented to hospital with left hip pain following a fall. On admission, there was reduced range of movement of the left hip but no other abnormal findings. Initial investigations were normal and she was managed with analgesia and low dose enoxaparin. She was a retired missionary nurse who had worked in Africa for most of her life. Her past medical history included episodes of malaria and schistosomiasis.

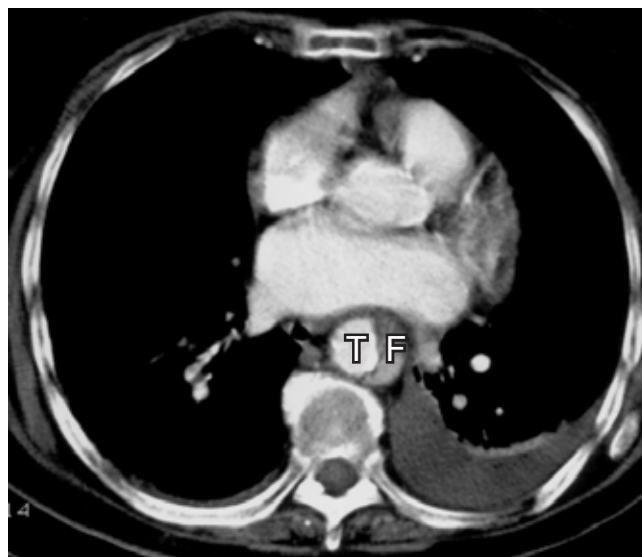
Five days after admission she developed a temperature of 38°C. Clinical examination was normal except for bruising over her left hip. Haemoglobin fell from 12g/dl on admission to 7g/dl, although there was no overt blood loss and she remained haemodynamically stable. It was suspected that she had bled at the site of injury, however an isotope bone scan excluded a hip fracture. Her C-reactive protein (CRP) was elevated at 320 mg/l (normal <10 mg/l) and, as dipstick urinalysis showed blood and protein, she was treated for a possible urinary tract infection.

Over the ensuing weeks unremitting low-grade pyrexia continued. Other than lethargy and anorexia there were no specific symptoms. Repeated physical examinations were normal. There was no growth on repeated blood and urine cultures. A search for acid-fast bacilli was negative. Serological tests for atypical bacteria, viruses and autoimmune causes of her pyrexia were negative. Multiple blood films for malarial parasites were negative. Chest radiographs, an abdominal ultrasound scan and an abdominal computed tomography (CT) scan were unremarkable. No vegetations were identified on a transthoracic echocardiogram. A transoesophageal echocardiogram was not performed at this stage as it was thought unlikely that the patient would tolerate the procedure due to agitation.

An empirical course of intravenous antibiotics followed by a trial of anti-tuberculosis therapy were administered without a significant response.

After 40 days of pyrexia we remained concerned that infective endocarditis had not definitively been excluded. A transoesophageal echocardiogram was performed. This demonstrated a previously unsuspected thoracic aortic dissection that was delineated further on a CT scan of chest (Figure). It arose at the level of an aberrant right subclavian artery and terminated at the level of the coeliac axis. It was not suitable for surgical intervention and the patient was managed with careful blood pressure control (systolic blood pressure <120 mmHg). She spontaneously defervesced after 52 days of pyrexia. Her CRP normalised and following a period of rehabilitation she returned home.

**Discussion:** Aortic dissection usually presents as an emergency, most often associated with tearing chest pain<sup>1</sup>. However approximately 15% can remain almost clinically silent<sup>2</sup>. Although one third of patients with aortic dissection may experience a transient increase in temperature<sup>1</sup>, prolonged pyrexia as the dominant presenting clinical sign is extremely rare<sup>3</sup>. The pyrexia is thought to result from tissue destruction in the aortic wall and the release of endogenous pyrogens in



Computerised tomography of chest demonstrating thoracic aortic aneurysm (T=true lumen, F=false lumen)

the aortic haematoma<sup>4</sup>. The febrile episode may last anywhere between five and eleven weeks and is often accompanied with a rise in inflammatory markers and a normochromic, normocytic anaemia<sup>5</sup>.

In this case the unusual presentation of the dissection along with the confounding factor of previous residence in Africa delayed the diagnosis. This reminds physicians investigating PUO to remain open-minded as not all conditions present themselves as classically as the textbooks would have us believe.

The authors have no conflict of interest.

\*Kim Sinnamon<sup>1</sup>, Registrar

M Ivan Wiggam<sup>2</sup>, Consultant

<sup>1</sup>Department of Health Care for the Elderly, Royal Victoria Hospital, Grosvenor Road Belfast BT12 6BA, United Kingdom, and <sup>2</sup>Stroke Unit, Level 7, Tower Block, Belfast City Hospital, Lisburn Road, Belfast, BT8 8JR United Kingdom.

\* Nephrology Department, Level 11, Belfast City Hospital, Belfast BT9 7AB

kimsinnamon@msn.com

1. Hirst AE, Johns VJ, Kime SW. Dissecting aneurysms of the aorta: a review of 505 cases. *Medicine (Baltimore)* 1958;**37**(3):217-9.
2. Spittell PC, Spittell JA Jr, Joyce JW, Tajik AJ, Edwards WD, Schaff HV *et al*. Clinical features and differential diagnosis of aortic dissection: experience with 236 cases (1980 through 1990). *Mayo Clin Proc* 1993;**68**(7):642-51.
3. McKeown PP, Campbell NP. Pyrexia of unknown origin and aortic dissection. *Int J Cardiol* 1989;**25**(1):124-6.
4. Mackowiak PA, Lipscomb KM, Mills LJ, Smith JW. Dissecting aortic aneurysm manifested as fever of unknown origin. *JAMA* 1976;**236**(15):1725-27.
5. Giladi M, Pines A, Averbuch M, Hershkoviz R, Sherez J, Levo Y. Aortic dissection manifested as fever of unknown origin. *Cardiology* 1991;**78**(1):78-80.