Letters

THE PATIENT IMPACT OF A&E CLOSURES

Editor,

Accident and emergency (A&E) department closures in Northern Ireland have stimulated controversy in public, media and political opinion. We raise the question, what is the impact of A&E closures on patients?

We summarise below the results from our recent survey, following the closure of Midulster A&E in May 2010. The Midulster residents now have access to acute healthcare services only via alternative hospitals.

METHODS:

We surveyed patients attending Moneymore Medical Centre and Coagh Medical Practice from 7-17 November 2011 (N=100).

RESULTS:

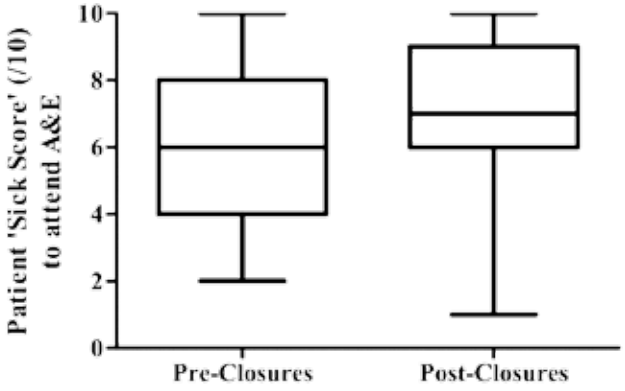
Demographics: Following the closure of Midulster A&E, the closest A&E to 80% of the respondents was Antrim Area Hospital. The mean reported driving time to their current nearest A&E service was 43 minutes. Furthermore, 74% of respondents reported having used at least one of the following hospital services (outside of the Midulster Hospital) within the previous 12 months: A&E, inpatient stay, outpatient clinic or specialist scan.

Responses 1: 82% of respondents rated access to health services as "worse" following Midulster Closures; 17% rated "same"; and 0% rated "better" (1% did not answer this question). A majority of respondents reported "further distance" (86%) and/or "long waiting times" (80%) as the main difficulties introduced by hospital closures. Only 12% reported improved services after the centralisation of acute services.

Responses 2: We asked patients to rate on a ten-point scale how unwell they would need to feel in order to attend A&E (with 1 as least severe and 10 as life-threatening). In the same questionnaire, we asked for two ratings – one for prior to the closure of Midulster A&E, and one post-closure. We refer to each rating as the Patient Sick Score. We found a significant difference between the pre- and post- closure ratings ($p<0.0001$) (Figure 1).

CONCLUSION & DISCUSSION

Our survey shows that the patients perceived a negative impact on ease of access to acute healthcare services following Midulster closures. Few patients perceived benefits to the centralisation of services. The Patient Sick Score measurements (Figure 1) further indicate that patients are only willing to attend A&E when they perceive a more severe illness, following the closure of Midulster A&E. This could strain local primary healthcare services since patients are more reluctant to access acute hospital services.



Mann-Whitney U Value = 3087 ($p<0.0001$)

Fig 1. Boxplot showing Patient Sick Scores to attend A&E pre- and post-hospital closures. Post-closure, patients reported that they would need to feel more unwell in order to attend A&E.

While we acknowledge the limitations of the survey (e.g., retrospective nature and small sample size), the responses suggest that the Midulster closures have negatively impacted this patient group.

We therefore recommend a re-evaluation of the measures that were instituted to ensure continued adequate access to acute healthcare services following the Midulster closures. We also highlight this as a topical issue, since there is ongoing debate regarding further closures of acute hospital services in Northern Ireland.

The authors have no conflicts of interest

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HOARSING AROUND: IS IT TIME TO GROW UP?

Editor,

Dysphonia is common in children with incidence rates ranging from 6% to 23%[1]. Despite this practice varies considerably regarding what, if any, investigations are performed and how the condition is managed. Although childhood dysphonia is mostly due to simple causes such as voice misuse, serious underlying pathology such as papillomatosis or malignancy needs to be excluded.

Paediatric voice disorders typically have been blamed on vocally "abusive" behaviours or pubertal changes, and many practitioners tend not to provide intervention because they believe that children will "grow out of it." However, changes in pitch, loudness, and overall vocal quality tend to interfere with a child's ability to effectively communicate. Research focused on paediatric voice disorders has shown that there is an association with perceived negative attention, limited participation in activities and an overall decrease in life quality in children suffering from them[2]. Voice disorders are common in the paediatric population and have recently been gaining more attention however there is still a lack of information available to clinicians regarding their evaluation and treatment.

Paediatric head and neck malignancy is rare with paediatric laryngeal cancers being especially rare. Conventional risk factors, such as active and passive smoking, exposure to chemicals or radiation therapy are frequently not present in the clinical history and little may be found on clinical examination. This can therefore be falsely reassuring to the clinician leading to significant delays in investigation and treatment which would not happen in an adult patient. Head and neck cancer in paediatric patients can be particularly devastating with significant long term effects even following successful treatment. In addition to surgical morbidity and mortality, late effects of treatment in children receiving radiotherapy for head and neck cancer are frequent, including neuroendocrine, dental, thyroid, and cognitive toxicity.

Voice clinics and voice therapy services are now well established in most adult health services in the developed world. Equivalent services for children are less common at least in the United Kingdom. NICE currently recommends that any adult with dysphonia for greater than three weeks is investigated and referred onto ENT services[3]. There are currently no national guidelines for referring paediatric patients with voice disorders to the ENT service. In the United States, Johns Hopkins Paediatric ENT department recommends referral following four weeks of persistent hoarseness while Miami's Children's Hospital recommends referral after only one week[4,5]. With increasing incidence of head and neck cancer in adolescents and the associated negative effects of voice disorders on children we need to ask should we be reassessing the need to refer children and adolescents with dysphonia in line with adult referral patterns?

It is the authors view that the paediatric patient with persistent dysphonia for greater than three weeks should be investigated with the same degree of clinical suspicion that their adult counterparts would. Although few will ever be found to have significant pathology, options are available to improve the quality of life of paediatric patients with chronic voice problems.

The authors have no conflicts of interest.

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A DIFFERENT TYPE OF FIGHT BITE- AN UNUSUAL CAUSE FOR RUPTURE OF THE PECTORALIS MAJOR TENDON

Editor

A 40-year-old right hand dominant doorman presented approximately one year following an injury to his left pectoralis major tendon. He reported that whilst restraining an individual in a headlock he was bitten in his left anterior axillary fold and that immediately following the incident he had pain, extensive bruising and swelling in relation to his left anterior chest wall. He did not seek medical attention at that time. Clinical examination revealed bunching of the left pectoralis major muscle towards its sternal origin (Figure 1) and weakness of shoulder adduction. MRI scanning

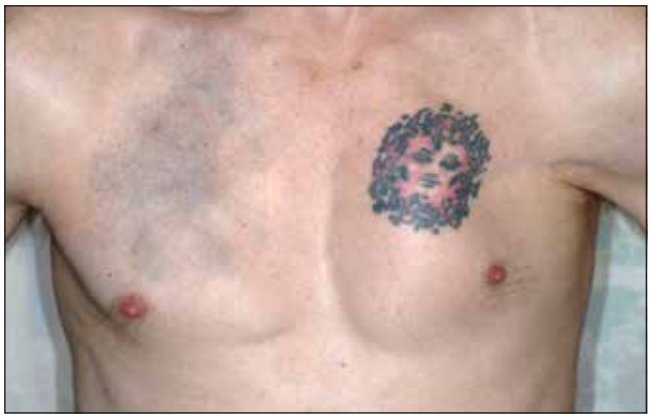

Fig 1. Clinical picture demonstrating loss of contour of the left anterior axillary fold with medial bunching of the pectoralis muscle (Note – distinguishing mark airbrushed from right anterior chest wall)

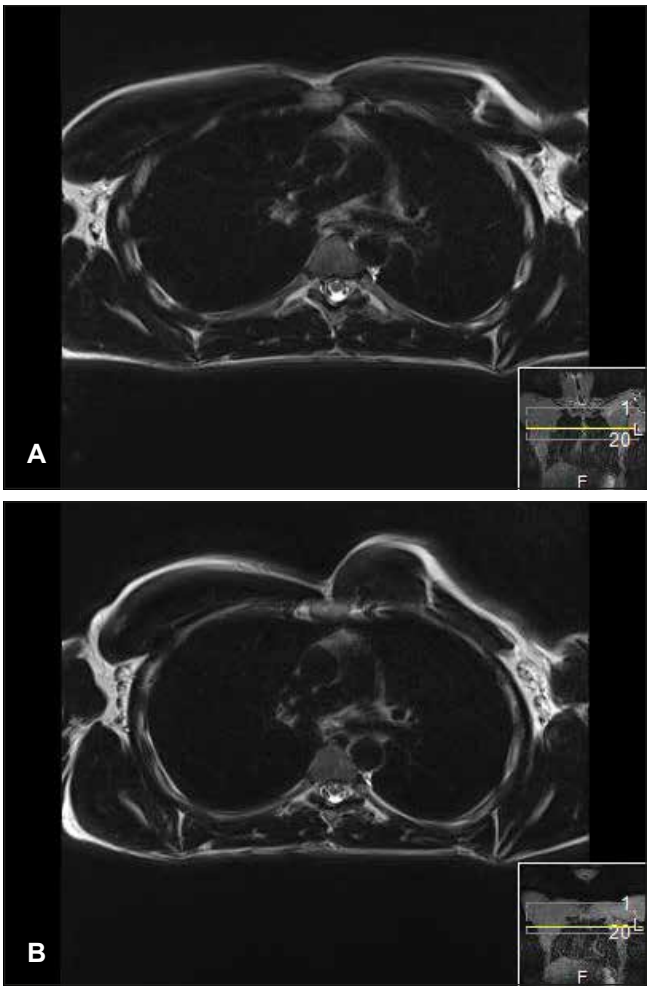

Fig 2 (a) and (b). (a) Axial T2 BLADE studies demonstrating linear high T2 signal traversing the sternal head of the pectoralis major musculotendinous junction in keeping with a full thickness tear in relaxation with medial displacement of the muscle belly during active contraction (b).

demonstrated a full-thickness, complete-width defect of the sternal head of the left pectoralis major tendon (Figures 2a and 2b). The patient declined surgical reconstruction.

Pectoralis major tendon rupture is an uncommon injury. Following a comprehensive review of the literature, Elmaraghy and Devereaux[1] identified 365 reported cases of pectoralis major injury between 1822 and 2010 with only 24% reported between 1822 and 1990. Bak et al reported a meta-analysis of 112 cases [2].

Tears of the pectoralis major tendon predominantly occur in active men aged between 20 and 40 years[1,2]. The majority of ruptures occur at or between the musculotendinous junction medially and the humeral insertion of the pectoralis major tendon laterally (67%)[1]. Delayed presentation is not uncommon with these injuries. MRI has been helpful in identifying complete-width, full-thickness ruptures but is less reliable in identifying partial-thickness ruptures because MRI cannot distinguish between the bilaminar tendon layers[2, 3]. Often it is only at the time of surgery that the true morphology of the rupture can be definitively characterised[4].

Indirect trauma is the most common mechanism of injury (83% cases) with most injuries occurring when a maximal force is applied with the pectoralis muscle eccentrically contracting during an abducted and externally rotated shoulder movement e.g. during the deep part of the lift during a bench press manoeuvre[2].

Studies have shown that the average bite force of an adult male ranges from 382N to 909N with maximal force being applied by the molar teeth[5] which is more than suffice to cause damage to the pectoralis major tendon, which is on average 4mm thick and 4cm wide[1]. We postulate that with the shoulder abducted, flexed and internally rotated, when restraining an individual in a headlock, the pectoralis major tendon is accentuated and thus susceptible to injury from a bite.

Numerous repair techniques have been reported to reconstruct pectoralis major tendon injuries[6]. The decision to proceed with operative treatment depends on the degree of injury and the functional demands of the patient. Better outcomes have been reported with operative treatment particularly in high performance athletes[7, 8]. Due to the chronicity of our case, we suspect that direct repair would not have been possible and that a tendon graft or mesh repair would have been required. To the best of our knowledge rupture of the pectoralis major tendon due to a human bite has not been previously reported.

The authors have no conflicts of interest.

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PYODERMA GANGRENOSUM: THE ROLE OF SURGICAL TREATMENT.

Editor,

In a recent case, we anecdotally found a positive benefit to surgical management of Pyoderma Gangrenosum (PG). The disease process behind PG remains poorly understood, however through epidemiological and etiological studies an understanding of the risk factors involved has developed. There is increasing recognition of post trauma or post surgical PG, with cases reported from a number of specialities[1]. Key to the principles of current treatment is an abnormal over-active response to trauma, or pathergy.

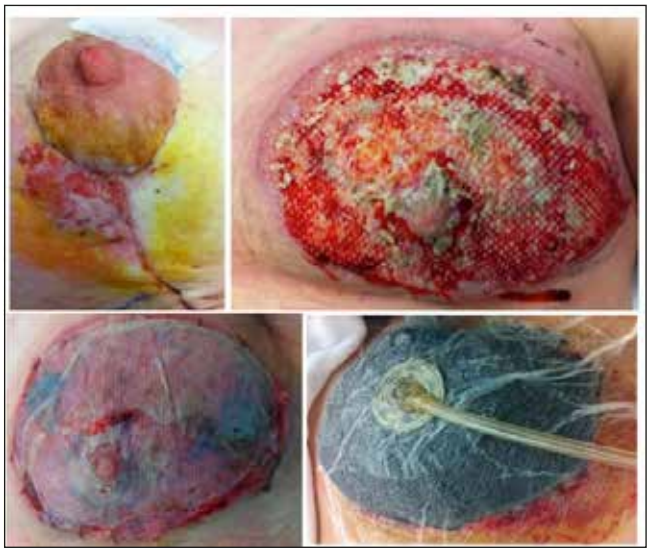

Fig 1. Showing, clockwise from top left, the patients wound; at presentation, after five days of medical treatment, with vacuum dressing in situ, and finally at graft check day 5 post op.

An otherwise fit and well 47 year old female, whom had underwent wide local excision and chemo-radiotherapy four years previously, attended for right breast reduction and left mastopexy. The surgery was carried out without complication and she was discharged home the following day. She presented one week later with erythema, tenderness and an enlarging area of ulceration arising from the mastopexy wound on the left breast. Despite medical management for presumed surgical site infection, the condition progressed. Dermatology reviewed the patient and commenced intravenous steroids for a presumptive diagnosis of PG. Histopathological examination of biopsies showed no vasculitic or malignant process. However it identified a spongiotic, inflamed epidermis, which was infiltrated with neutrophils. The dermis demonstrated extensive necrosis, and mixed neutrophil and lympocytic infiltration. These are the histopathological findings of PG.

Due to the extensive skin loss, after a full discussion of the risk of exacerbation, the patient elected to proceed onto surgical debridement. A split thickness skin graft provided coverage for the left breast, and a vacuum dressing applied. The surrounding skin was covered with a border of hydrocolloid dressing, to prevent maceration of the tissues. Secondly the sponge was deliberately cut slightly small to prevent pressure on the skin edge. She had an uncomplicated recovery, and she continued on a reducing dose of oral steroid and was reviewed in the outpatient clinic at four weeks where the graft was found to have healed fully. At six month review, she has suffered no wound breakdown, and is coping well with the psychological impact of altered aesthesis of the breast.

PG carries a significant risk of mis- or indeed delayed diagnosis. Our case demonstrates the dilemma, and the safe practice of treating the more common diagnosis of surgical site infection in the first instance. It is important to consider PG in the post surgical patient with an apparent infection, which grows no micro-organisms. Early referral to Dermatology and, the commencement of corticosteroids to control the inflammatory response is vital to arresting disease progression. In light of the skin loss, and the psychological impact upon these three women, we also requested the involvement of our clinical psychologist.

We recommend the careful consideration of surgical treatment as an adjunct to appropriate systemic management, as it can achieve an aesthetically good result in the instance of significant skin loss. Meticulous surgical technique to minimise trauma including careful dressing providing the key to success[2].

The authors have no conflicts of interest.

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'TOO MUCH, TOO SOON,' OR 'A STEP TOO FAR?' EXERCISE INDUCED RHABDOMYOLYSIS

Editor,

Introduction: Rhabdomyolysis is clinical condition characterised by damage to striated muscle membranes with resulting biochemical and clinical sequelae. Relatively uncommon and underdiagnosed, it is a potentially severe and debilitating condition with an increasingly multi-factorial aetiology. Exertional rhabdomyolysis refers to skeletal muscle damage induced by exercise, most commonly strenuous and eccentric exercise with clinical features developing after 48-72 hours[1,2].

Case Presentations: Two cases of Exercise Induced Rhabdomyolysis were admitted to a Belfast Teaching Hospital over a 2-week period. Each patient presented de-novo with debilitating myalgia, dark urine and significant CK elevation (CK levels 68,589 and 21,713 respectively). Previously healthy but exercise-naïve, both patients presented 72 hours following structured exercise programmes under 'qualified' supervision (spin class, fitness assessment). Aggressive fluid resuscitation with meticulous fluid balance facilitated renal preservation and complete recovery.

In the absence of muscle biopsies, one could argue that underlying genetic metabolic dysfunction could be responsible. Such conditions are uncommon; therefore the probability of serial independent non-consanguineous presentations is unlikely. Each case presented de-novo in adulthood; no history of previous rhabdomyolysis or exercise induced myalgia was elicited. In addition, both patients had previously participated in sporting activities to a level akin with their peers. Furthermore, the initial patient had persistent elevation of CK levels despite normal renal function, initiating interrogation of biochemical pathways; glucose-6-phosphate dehydrogenase, plasma acylcarnitine, free carnitine and white cell enzymes all within normal range. Undoubtedly the most significant aetiological factor pertinent to these cases was over-exertion by unaccustomed athletes.

Discussion: It is recognised that people who exercise regularly are less likely to develop rhabdomyolysis than their more sedentary counterparts[3]. It would therefore stand to reason that incremental physical training initiates adaptive cellular mechanisms which facilitate increased metabolic demand. Physically conditioned athletes can undergo severe exercise with only modest rises in CK[3]. For any given muscle mass as physical conditioning improves, severe exertion is less likely to produce significant CK elevation. Metabolic adaptation is therefore a dynamic mechanism.

Hamburg et al 2007 demonstrated metabolic changes consistent with physical inactivity. These included the development of insulin resistance, dyslipidaemia, increased blood pressure and impaired microvascular function in otherwise healthy volunteers[4]. Sedentary individuals also developed a down-regulation in reactive hyperaemic responses and a decrease in resting blood flow. These processes result in an imbalance between supply and demand of intracellular adenosine triphosphate (ATP) obligating the myocyte to utilise alternative oxidative pathways. The resulting cascades of pathophysiological reactions stimulate proteolytic enzymes that degrade cellular membranes. Compounds including CK, LDH, ALT, calcium, myoglobin, potassium and phosphate are released systemically. With limited ability to maintain homeostasis, direct tissue toxicity and systemically mediated pro-inflammatory reactions potentiate complications including; compartment syndrome, hyperkalaemia, disseminated intravascular coagulation, coagulopathies and acute renal failure.

Conclusion: Poor physical conditioning and intensive prolonged exercise coupled with inadequate hydration precipitated rhabdomyolysis in these patients. These cases illustrate the perils of over-exertion; 'too much too soon' and 'a step too far.'

Consent: Consent was obtained from both patients for the publication of these case reports.

The authors have no conflicts of interest.

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