CE

University of Florence, Florence e-mail: pa.modesti@unifi.it

G. Parati

Division of Cardiology, Department of Clinical Medicine,
Prevention and Applied Biotechnologies
Hospital San Luca Istituto Auxologico Italiano
University of Milano-Bicocca, Milan

Published online: 31 March 2007

We agree with Drs. La Regina and del Prato that problems in medicine can be approached from a variety of perspectives, although some basic directions should be shared by physicians working in different fields. There is no doubt that arterial hypertension is today a diagnosis that rarely leads to hospitalisation. There are however occasions characterised by a high risk for the patient, when this will be necessary, as in cases of pulmonary oedema or transient ischaemic cerebral attack. Concerning the patients without life-threatening hypertensive crises, the current Guidelines for the management of high blood pressure provide clear indications, suggesting the need for repeated visits to confirm the presence and the severity of an elevation of blood pressure, and to assess the global risk profile of the patient and the presence of target organ damage. In most cases there is no need to hasten the start of antihypertensive treatment until the diagnostic process has been completed. The time interval before starting treatment is obviously a function of the patient's risk level. An appropriate diagnostic work-up should also lead to identification of cases with secondary hypertension. Primary Care Physicians (PCP) have the duty to start management of patients with hypertension, in cooperation with specialised centres that provide all the necessary support for definition of the patient's risk profile and organ damage. This approach, accompanied by a good relationship between the patient and the physician, will also reduce patients' utilisation of the emergency service for false hypertensive crisis, often due to anxiety and sympathetic hyper-reactivity.

The diagnostic work-up of the patient should not be carried out in the Emergency Department, but should be part of the routine interaction between PCP and specialists.

Intern Emerg Med (2007) 2:63–65 DOI 10.1007/s11739-007-0017-y

Bilateral posterior shoulder dislocations following seizure

M.E. Betz · S.J. Traub

M.E. Betz (☒) • S.J. Traub One Deaconess Road WCC-2 Department of Emergency Medicine Beth Israel Deaconess Medical Center Boston, MA 02215, USA e-mail: mbetz@bidmc.harvard.edu M.E. Betz • S.J. Traub Harvard Medical School Boston, MA, USA

Received: 9 October 2006 / Accepted in original form: 8 November 2006 / Published online: 31 March 2007

Anterior shoulder dislocations are a common complaint in emergency medicine. Most emergency physicians can recognise an anterior shoulder dislocation based on physical examination alone, and the diagnosis is quickly confirmed with appropriate radiographs. Reduction can be accomplished with any number of techniques; a standard textbook of emergency medicine procedures lists nine ways, each with variations, to reduce anterior shoulder dislocations [1].

Posterior shoulder dislocations are rare, and account for less than 5% of all shoulder dislocations [1]. Although classical physical examination findings are described, they are frequently not appreciated by the initial examiner. The anteroposterior (AP) radiograph of the shoulder may appear normal, which contributes to a high misdiagnosis rate.

We report a case of bilateral posterior shoulder dislocations after seizure in a young man with no orthopaedic history.

A 25-year-old man with type I diabetes was at a local sporting event when he became hypoglycaemic and suffered a tonic-clonic seizure. Nearby fans caught him before he fell and helped him to the ground. He was taken to the stadium first aid station, where a fingerstick blood glucose level was too low to register on the glucometer. He received IV glucose and was then transported to our Emergency Department (ED) for further evaluation.

On arrival at the ED, he was post-ictal and unable to offer either a complaint or a history of present illness. On physical examination, his vital signs were: pulse, 77 beats/min; blood pressure, 144/87 mmHg; respirations, 21/min; temperature, 35.9°C. General assessment revealed a well developed, muscular young man who was confused but otherwise in no significant distress. Initial examination of the heart, lungs and abdomen were unremarkable. A fingerstick glucose obtained on arrival in the ED was 66 mg/dl (4 mmol/l).

The patient's mental status improved as his post-ictal state resolved. He reported a history of type I diabetes for approximately 1 year with a prior history of hypoglycaemia from poor compliance with his insulin regimen. He denied any other medical or surgical history. He complained that his shoulders hurt and that he could not move them.

More detailed examination of the extremities revealed shoulders that were symmetric but deformed, with empty glenoid fossae bilaterally and loss of the normal superior contour on each side. The humeral heads were palpable posteriorly on each side. The shoulders were adducted bilaterally. The patient was unable to lift or externally rotate either arm. Neurovascular status of the upper extremities was intact bilaterally.

AP radiographs of each shoulder (Fig. 1) appeared normal. Axillary radiographs of the shoulders (Fig. 2) revealed bilateral posterior dislocations without fractures. Procedural



Fig. 1 AP radiograph of left shoulder appears normal despite posterior dislocation

sedation was performed with fentanyl (50 mcg) and propofol (60 mg). When the patient was sufficiently sedated, each shoulder was reduced with a two-physician technique. The first physician flexed the elbow to 90° and applied gentle axial traction, while the second physician applied gentle anterior pressure to the posterior aspect of the dislocated humeral head. Reduction was successful on each side on the first attempt. The neurovascular examination was normal on each side after the reduction.

Repeat radiographs showed successful reduction of both shoulders and no associated fractures. The patient was observed in the ED overnight for monitoring of his hypoglycaemia and had no further episodes. He was discharged the next morning to follow-up with orthopaedic surgery, but the patient did not attend this appointment and was subsequently lost to follow-up.

Posterior shoulder dislocations account for only 4% of all shoulder dislocations; anterior shoulder dislocations (95%) are far more common. Inferior shoulder dislocations (luxatio erecta), occurring in only 0.5% of cases, are extremely uncommon [2]. Posterior shoulder dislocations are frequently associated with seizure, trauma or electrocution, and almost all bilateral posterior dislocations are the result of a seizure [2]. Posterior shoulder dislocation or subluxation is also associated with neonatal brachial plexus injury, with incidence estimates of dislocation in 8% of infants and children with a brachial plexus injury [3]. Unilateral posterior dislocations are among the most commonly misdiagnosed joint injuries [4], with delays to diagnosis of over one year [5]. Diagnosis of bilateral posterior dislocations can also be delayed, with reports of erroneous initial work-ups for aortic dissection or myocardial infarction because of pain in the chest or shoulder area [4].

The mechanism of posterior shoulder dislocation is believed to be unbalanced muscle contraction. During seizure activity, the internal rotator muscles of the shoulder contract with greater force than the external rotators, which causes the humeral head to move superiorly and posteriorly [6]. Associated humeral head fracture can occur due to continuing pressure against the glenoid rim.

On physical exam, the arm is usually adducted and internally rotated. Anteriorly, the shoulder may appear flat and "squared off", while posteriorly the humeral head may be palpable [2]. It is important to note that in cases of bilateral posterior dislocation the shoulders may be symmetric but still abnormal. Unlike in anterior shoulder dislocations, in which there may be damage to the axillary, musculocutaneous or radial nerves, neurologic or vascular injury is rare in posterior shoulder dislocations [2].

Proper joint imaging in cases of suspected posterior shoulder dislocation is crucial. AP radiographs can appear normal, as occurred in this patient (Fig. 1). In one series of 40 patients, only 50% of posterior dislocations were apparent when only AP and lateral radiographs were used [5]. Abnormalities commonly noted on the AP view include a humeral head that resembles a "light bulb" or "ice cream cone" rather than the usual "walking stick" [2]. Addition of axillary views raises the diagnosis rate to 100% [5].

If axillary views cannot be obtained because of patient pain (a common occurrence), computed tomography (CT) can show both the dislocation and any associated fractures [4]. For infants and children, ultrasound may be preferable to conventional imaging, as it does not expose the child to radiation or necessitate sedation, and it also allows for real-time assessment of reduction attempts [3].

In adults, reduction should be attempted with procedural sedation or general anaesthesia. The most commonly described technique is a two-operator method in which the first operator applies constant, gentle longitudinal traction on the adducted arm while a second operator uses both thumbs to press on the humeral head from the back to push it forward and rotate it internally. Special care should be taken in cases



Fig. 2 Axillary view of same shoulder reveals posterior dislocation

CE

with concomitant fracture; although some authors recommend one attempt at closed reduction, it is likely that such patients will require open reduction with fixation of the fracture, so early consultation with an orthopaedic surgeon is advisable [7, 8]. If closed reduction appears successful, the arm should be immobilised and post-reduction radiographs should be obtained to verify placement and identify any new fractures.

Patients with posterior shoulder dislocations should be seen by an orthopaedic surgeon, either in the ED or within 5–7 days after discharge, and the patient should remain in a shoulder immobiliser until this evaluation. Some patients may require early surgical intervention, while others may be treated with immobilisation [9]. Rotator cuff exercises or physical therapy can be useful in preventing recurrence of dislocation, especially in those with seizure disorders who are at risk of future dislocations during seizures [10].

We report a case of bilateral posterior shoulder dislocations that were identified and successfully reduced in the ED. Posterior shoulder dislocations occur rarely but are often missed on initial presentation, resulting in ongoing patient discomfort, long-term morbidity and elevated health care costs. Posterior shoulder dislocations should be considered in post-ictal patients with shoulder pain or an abnormally appearing shoulder. ED physicians may attempt to reduce the dislocation if there is no concomitant fracture, but early consultation with orthopaedic surgery is often advisable.

References

- Ufberg J, McNamara R (2003) Management of common dislocations. In: Roberts JR, Hedges JR (eds) Clinical procedures in emergency medicine, 4th edn. W.B. Saunders Company, St. Louis, pp 948–960
- Price DD, Wilson SR (2006) Dislocations, shoulder. Available at: http://www.emedicine.com/emerg/topic148.htm. Accessed 14 April 2006
- Moukoko D, Ezaki M, Wilkes D, Carter P (2004) Posterior shoulder dislocation in infants with neonatal brachial plexus palsy. J Bone Joint Surg Am 86:787–793
- 4. Elberger ST, Brody G (1995) Bilateral posterior shoulder dislocations. Am J Emerg Med 13:331–332
- Hawkins RJ, Neer CS 2nd, Pianta RM, Mendoza FX (1987) Locked posterior dislocation of the shoulder. J Bone Joint Surg Am 69:9–18
- Shaw JL (1971) Bilateral posterior fracture-dislocation of the shoulder and other trauma caused by convulsive seizures. J Bone Joint Surg Am 53:1437–1440
- Ogawa K, Yoshida A, Inokuchi W (1999) Posterior shoulder dislocation associated with fracture of the humeral anatomic neck: Treatment guidelines and long-term outcome. J Trauma 46:318–323
- Tellisi NK, Abusitta GR, Fernandes RJ (2004) Bilateral posterior fracture dislocation of the shoulders following seizure. Saudi Med J 25:1727–1729
- Quillen DM, Wuchner M, Hatch RL (2004) Acute shoulder injuries. Am Fam Physician 70:1947–1954
- 10. Sankar B, Aby NG, Rameto AS et al (2004) Spontaneous

reduction of posterior shoulder dislocation following repeated epileptic seizures. Indian J Med Sci 58:131–132

Intern Emerg Med (2007) 2:65-67

Amino acid sequence homologies between HCV polyprotein and thyroid antigens

A. Martocchia · P. Falaschi

A. Martocchia • P. Falaschi
Department of Medicine
II Faculty of Medicine
University of Rome "La Sapienza"
Rome, Italy
A. Martocchia (☒)
c/o Prof. Paolo Falaschi
S. Andrea Hospital
Via di Grottarossa 1035, I-00189 Rome, Italy
e-mail: a_martocchia@virgilio.it

Received: 25 May 2006 / Accepted in revised form: 31 August 2006 / Published online: 31 March 2007

Recent evidence in the literature suggests that molecular mimicry between viral and self antigens may be involved in the pathogenesis of autoimmune thyroid diseases in patients with chronic hepatitis C virus (HCV) infections [1–3].

Chronic HCV infection has been reported to be associated with thyroid autoimmunity and thyroid function disorders with a mean incidence of 10% and 3%, respectively [4, 5]. Alfa-IFN therapy may exacerbate or induce underlying latent thyroid disorders, increasing the incidence of thyroid autoimmunity and thyroid function disorders to 20% and 11%, respectively [4, 5].

In keeping with the tenets of the clonal selection theory of acquired immunity, an infectious agent may circumvent the deletion of anti-self lymphocytes activating clones with receptors sufficiently degenerated to respond to mimicking epitopes and host antigens [6].

A minimum of five to six amino acids are necessary to induce an immune response, and the probability of 20 amino acids occurring in six identical residues between two proteins is 20^6 (for each peptide, irrespective of the sequence) or 1 in 128 000 000 [7].

We performed the comparison between the amino acid sequence of the HCV polyprotein and five tissue-specific antigens of human thyroid, available in the database on www.ncbi.nlm.nih.gov/pubmed.

In particular, we examined the following HCV genotypes (with the respective NCBI sequence identification number): HCV1a (GI:130455), HCV1b (GI:130469), HCV1c (GI:385131), HCV2a (GI:130466), HCV2b (GI:130468),