

Beware of the anticoagulated elderly with minor head injury

提防服用抗凝血劑的輕度頭部受傷長者

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A 69-year-old man first presented to the emergency department after a fall. He had no history of loss of consciousness or vomiting. He sustained a 3 cm long laceration over the right occipital region of the head. There was no fracture in the X-rays of the skull. He was on warfarin because of cardiac problem. He was discharged after suturing. He re-attended the next morning because of left sided weakness. Non-contrast brain computed tomogram showed acute subdural haematoma. Burr holes were performed subsequently. Special precautions should be undertaken in managing the elderly with minor head injury, with a lower threshold for computed tomography and coagulation profile studies. (*Hong Kong j.emerg.med.* 2005;12: 108-111)

一名 69 歲男性病者初時因跌倒到急症室求診。他的頭右枕部有一長 3 厘米的裂傷，無人事不省或嘔吐病歷，頭部 X 光顯示無骨折現象；但他因心臟問題需要長期服用華法令抗凝血劑，他接受傷口縫合後出院。翌日早上他因為左側軟弱無力而再次求診，無造影劑的腦部電腦掃描顯示為急性硬膜下血腫，其後需要進行顱骨鑽孔術。故此在處理頭部輕度受傷的年長者時應特別小心，需要調低這類病者做電腦掃描及凝血測試的界限。

Keywords: Aged, anticoagulants, brain computed tomography, closed head injuries, subdural haematoma

關鍵詞：年長者、抗凝血劑、腦部電腦掃描、閉合性頭部受傷、硬膜下血腫

Introduction

Head injury of varying clinical severity is a common presentation at emergency departments. External physical injury has poor correlation with the seriousness of the underlying brain injury. The ultimate morbidity and mortality of the head injury patient essentially depend on the extent of the primary and secondary brain injury sustained.¹ Emergency physicians should not be misled into a false sense of

security by trivial scalp wounds, especially in high-risk patients.

Case report

A 69-year-old man first presented to the emergency department in March 2003 after a fall down three steps of stairs. He had no history of loss of consciousness or vomiting. He had an acute myocardial infarction in August 2000. Coronary artery bypass grafts were performed in October 2000. Because of a left ventricular apico-septal aneurysm with clot, he was put on life-long warfarin sodium since November 2000. He had an embolic stroke with right hemiparesis in December 2001 and a minor recurrence in January 2003, but he could still walk unaided afterwards. He

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had also diabetes mellitus and hypertension. He neither drank nor smoked. The blood pressure was 149/94 mm Hg, pulse rate 93 beats/min and respiratory rate 18 breaths/min. On physical examination, there was a 3 cm long laceration over the right occipital region of the head. He was fully conscious, with Glasgow coma scale score 15/15. His pupils were equal and reactive. The right elbow was contracted with 90 degrees fixed flexion. There was a 4 cm diameter haematoma around the right elbow. There was no fracture in the X-rays of the skull, chest and right wrist, but a fracture was seen in the head of the right radius. Suturing to the scalp laceration and splintage to the right elbow were done. He was discharged with paracetamol, head injury advice and a follow-up appointment in one week's time. He re-attended the next morning because of left sided weakness. The blood pressure was 133/80 mm Hg, pulse rate 80 beats/min, respiratory rate 80 breaths/min, temperature 37°C and SpO₂ 98% on room air. The spot blood sugar was 10.5 mmol/L and the Glasgow coma scale score was 15/15. His speech was slurred. His pupils were equal and reactive. Non-contrast brain computed tomogram (CT) showed acute subdural haematoma mainly along the right side of the falx cerebri, with a maximum thickness of 15 mm and little mass effect (Figure 1). There was



Figure 1. Acute subdural haematoma along falx cerebri.

also some bleeding along the right fronto-temporal convexity. The blood results showed prothrombin time of 24.7 seconds (normal 10.4-12.6), activated partial thromboplastin time 36.3 seconds (normal 24.5-37.6) and international normalised ratio 2.2. He was admitted and put under observation. Warfarin was withheld. Burr holes were performed after two weeks, when a repeat CT scan showed increased size of the subdural haematoma. Unfortunately, he died of chest infection six weeks later.

Discussion

Traditionally, subdural haematoma is arbitrarily classified into three stages after the acute injury – acute, subacute and chronic.¹⁻³ The subacute phase begins 3-7 days after injury and the chronic phase begins 2-3 weeks after injury. It can also be classified as simple or complicated, with roughly the same incidences. Simple subdural haematoma has no accompanying brain injury, with a mortality rate of around 20%. Complicated subdural haematoma has accompanying brain injury such as contusion or laceration, with a mortality rate of about 50%.²

Elderly patients commonly develop subdural haematoma after apparently trivial blunt head injury.²⁻⁴ Often, the antecedent event is not recognised or remembered.^{2,3} These patients generally lose consciousness, but this is not absolute.² Because of brain atrophy in elderly patients, the bridging veins traverse greater distances resulting in higher risks of tearing after head injury.^{1-3,5} The effect of increased intracranial pressure may be delayed as venous bleeding is slow and the atrophied brain is shrunken away from the inner table of the skull.^{5,6}

Patients with coagulopathy (e.g. haemophilia) or on anticoagulants (e.g. warfarin, heparin, aspirin) may develop subdural haematoma with a seemingly trivial head injury and warrant a lowered threshold for CT scans.^{2,7-13} Alcoholics are also prone to thrombocytopenia, prolonged bleeding times and blunt head injury, in addition to brain atrophy.² Spontaneous subdural haematoma, although rare and limited to sporadic case reports only, has been associated with

coagulopathy.³ Consequently, the occurrence of subdural haematoma is much higher in patients receiving anticoagulant therapy.^{9,14} It has been claimed that there is a 10-fold increase in the likelihood of developing an intracranial haematoma¹⁵⁻¹⁷ and a 4-fold higher risk of death¹⁸ in patients on warfarin after apparently minor head injury. The triad of anticoagulation with warfarin, age greater than 65 years, and traumatic head injury frequently produces intracranial haemorrhage with a very high mortality (up to 80%).¹⁸⁻²⁰

Stein et al showed that delayed brain injury and presence of coagulopathy were strongly associated.^{11,12,21} They also found that delayed brain injury was significantly associated with higher mortality, slower recovery and poorer outcome.^{12,21} They recommended that all patients with head injury should have basic coagulation panel tested (prothrombin time, activated partial thromboplastin time and platelet count), and coagulopathy corrected if necessary.^{3,11} If coagulopathy is discovered in the patient with head injury, early CT scanning is advocated to discover intracranial lesions that are likely to occur.¹¹

However, the results of some studies evaluating head injury and anticoagulation were contradictory or inconclusive.¹⁸ A few retrospective studies claimed that the incidence of clinically significant intracranial injury was extremely low in the anticoagulated patient suffering from minor blunt head trauma without loss of consciousness, amnesia or acute neurologic abnormality, and CT scanning might not be necessary,²² and morbidity and mortality might not be increased.²³ Yuen and others concluded that evidence supporting routine brain CT in warfarinised minor head trauma patient was lacking, after searching the medical literature.²⁴

The efficacy of reversing the anticoagulant effect at the time of hospital admission remains unanswered.^{18,25} Moreover, the original medical problem necessitating anticoagulation will become a concern if anticoagulation is stopped. Reversing the effects of warfarin with fresh frozen plasma and vitamin K may cause thrombotic complications in approximately 10% of these trauma

victims.²⁶ If the patient is to be discharged, it is logical that he should be advised to stop the anticoagulant temporarily for some days, as the elimination half-time of warfarin is 40 hours.²⁷ Again, research evidence support is lacking. At a minimum, it is wise to arrange an early follow-up within one or two days to re-assess the coagulation profile and neurological status - and not one week as in this case. Once again, this is not evidence-based.

Delayed diagnosis of subdural haematoma may result in poor outcome and medico-legal litigation.^{2,3,12,21} Precautions should be undertaken in managing the elderly with minor head injuries, especially those on anticoagulants. Basing on the limited evidence available at present, it is recommended that blood coagulation profile studies and neuro-imaging should be undertaken liberally, and they should be mandatory in those with coagulopathy. At a minimum, implementing neuro-observation in the emergency ward may clear some of the dilemma facing the emergency physician.

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