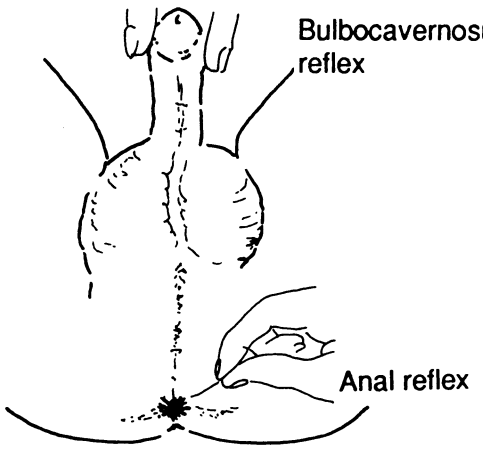

Sacral reflexes.

Neurological examination is mandatory in all unconscious patients, and baseline observations are extremely important in patients with spinal injury, not least for medicolegal reasons. Use of a Glasgow or similar coma chart will allow limb movements and strength to be charted. Some head injury charts do not include this facility. Neurological examination in unconscious patients is usually limited to completing the coma chart, funduscopy, and assessing tone and reflexes, but in patients with suspected cord injury the abdominal, anal, and bulbocavernosus reflexes should be recorded. The sensory response to pain can also be assessed in patients with depressed consciousness. Beware of flaccidity and areflexia in an arm as this may result from brachial plexus injury or spinal cord trauma, or both (particularly in motorcyclists).

The illustrations of the lateral position, transfer to a trauma trolley, and log rolling were prepared by the department of education and medical illustration services, St Bartholomew's Hospital.

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The ABC of Major Trauma has been edited by Mr David Skinner, FRCS; Mr Peter Driscoll, FRCS; and Mr Richard Earlam, FRCS.

Lesson of the Week

Aplastic anaemia associated with a non-steroidal anti-inflammatory drug: relapse after exposure to another such drug

Richard Andrews, Nigel Russell

Patients who have had aplastic anaemia while taking a non-steroidal anti-inflammatory drug should avoid subsequent exposure to all such drugs

Aplastic anaemia is one of the most serious of all drug complications and has a high mortality despite advances in treatment. Roughly 30% of cases are related to environmental factors or drugs.[1] We report a case of fatal aplastic anaemia related to use of the non-steroidal anti-inflammatory drugs sulindac and fenbufen.

Case report

A 65 year old woman presented in February 1984 with a four week history of spontaneous bruising and a rash. A full blood count showed a haemoglobin concentration of 75 g/l, a white cell count of $1{\cdot}4\times10^9$/l (neutrophils $0{\cdot}64\times10^9$/l), and a platelet count of 10×10^9/l. A trephine biopsy of bone showed a severely hypocellular marrow. An acidified serum test (Ham's test) yielded negative results. For the past three years she had been taking propranolol 80 mg twice daily for hypertension and ibuprofen 400 mg thrice daily for osteoarthritis of her knees and spine. Five weeks previously she had been prescribed sulindac 200 mg daily for further analgesia.

Aplastic anaemia was diagnosed and sulindac thought to be the likely causative agent. All analgesics were stopped, a single blood transfusion was given, and oxymetholone 200 mg daily was started. She responded well, and four months later a blood count showed a haemoglobin concentration of 103 g/l, a white cell count of $5{\cdot}45\times10^9$/l (neutrophils $2{\cdot}8\times10^9$/l), and a platelet count of 105×10^9/l. The oxymetholone was stopped because she developed jaundice, but she remained well for three years, not taking any treatment and with blood counts similar to those noted above.

In April 1987 worsening knee pains caused her to consult her general practitioner, who prescribed fenbufen 300 mg during the day and 600 mg at night. Two weeks later she presented with epistaxis, widespread bruising, and a purpuric rash. A full blood count showed recurrent pancytopenia with a haemoglobin concentration of 64 g/l, a white cell count of $2{\cdot}6\times10^9$/l (neutrophils $1{\cdot}6\times10^9$/l), and a platelet count of 49×10^9/l. A relapse of her aplastic anaemia was diagnosed on the basis of results of a bone marrow biopsy. Fenbufen was implicated and stopped. She was given a blood transfusion and remained dependent on transfusions for the next six months. Despite further treatment with oxymetholone and anti-thymocyte globulin her pancytopenia progressively worsened. Her clinical condition deteriorated, and she died of respiratory failure in October 1987. Necropsy showed haemorrhagic pneumonia and aplastic anaemia.

Discussion

Both sulindac[2-5] and fenbufen (Committee on Safety of Medicines, personal communication) have been implicated as causative agents in aplastic anaemia. This, however, is the first case to be reported in which a non-steroidal anti-inflammatory drug was implicated in the relapse of aplastic anaemia that had originally been associated with another such drug. We recommend that extreme caution is exercised when any non-steroidal anti-inflammatory drug is given to patients with a history of aplastic anaemia related to such drugs.

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