Thrombolysis and low back pain

Drs D FISHWICK, A PRASAN, and P ADAMS (Department of Cardiology and General Medicine, Royal Victoria Infirmary Trust, Newcastle upon Tyne) write: We report the case of a 66 year old man who developed acute severe backache after having been given streptokinase but who was subsequently able to tolerate recombinant tissue plasminogen activator with no adverse effects.

The patient was admitted with a six hour history of typical ischaemic cardiac pain. His medical history included femoropopliteal bypass grafting for peripheral vascular disease and quadruple coronary bypass grafting for severe angina seven years previously. On admission he was taking the following drugs: nifedipine 20 mg twice daily, atenolol 50 mg once daily, aspirin 150 mg once daily, isosorbide mononitrate 40 mg twice daily, and indomethacin 25 mg three times daily for gout.

On examination he was in sinus rhythm and his blood pressure was 140/90 mm Hg, with no added heart sounds, murmurs, or signs of cardiac failure. An electrocardiogram on admission showed an acute posterior myocardial infarction, and it was decided to give thrombolysis. Eight minutes into an infusion of 1·5 million units of streptokinase (roughly 250 000 U delivered), the patient developed acute severe backache in the lumbar region. He scored this as 10 out of a maximum score of 10 on a scale of pain severity. The streptokinase was stopped, but the electrocardiographic changes and chest pain continued. Recombinant tissue plasminogen activator was immediately given (15 mg bolus intravenously, 50 mg over 30 minutes, and 35 mg over 60 minutes) because there were no abnormal neurological or vascular findings on examination. The back pain did not recur. Ninety minutes later the electrocardiographic changes had largely resolved.

The effectiveness of thrombolysis in acute myocardial infarction is now well established.[1] Low backache is fairly well documented in patients receiving streptokinase during thrombolysis for myocardial infarction.[2-5] These recent accounts describe a total of 13 cases in which backache occurred. Only in four of these cases was the streptokinase continued after appropriate analgesia; in one it was restarted and subsequently discontinued, and the remaining patients were denied the further benefit of thrombolysis. One patient received recombinant tissue plasminogen activator with no further problems.[2]

Our case supports the rationale of immediate reintroduction of an alternative thrombolytic agent if acute severe backache occurs with streptokinase and no other cause is immediately suspected.

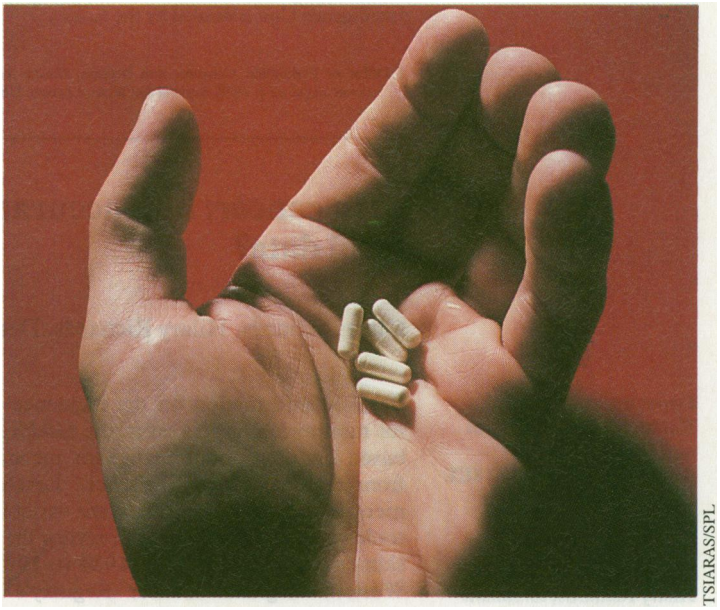
1 Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986;i: 397-402.
2 Lear J, Rajapakse R, Pohl J. Low back pain associated with streptokinase. *Lancet* 1992; 340:851.
3 Bourke J. Streptokinase and low back pain. *NZ Med J* 1992;105: 482.
4 Correspondence. Low back pain associated with streptokinase. *BMJ* 1991;302:111-2.
5 Shah M, Taylor RT. Low back pain associated with streptokinase. *BMJ* 1990;301:1219.

Urinary retention with misuse of "ecstasy"

Dr A A BRYDEN, Mr P J N ROTHWELL, and Mr P H O'REILLY, (Stepping Hill Hospital, Stockport, Cheshire 5K2 7JE) write: A healthy 19 year old man was referred to us with a 24 hour history of acute retention of urine. Other than a painful palpable bladder, general examination and analysis of urine showed nothing abnormal. We inserted a urethral catheter and removed it after 36 hours on free drainage. No further retention of urine occurred. He gave a history of drug misuse which included taking amphetamine and smoking marijuana. For 12 months before admission, however, he had used only 3, 4-methylenedioxymethamphetamine (ecstasy), taking one or two tablets once or twice a week, usually at parties. He later admitted to having taken 15 tablets during the 36 hours before admission.

3, 4-Methylenedioxymethamphetamine is a synthetic amphetamine analogue, structurally similar to methamphetamine. It is a "designer drug," widely and illegally used for its psychotropic effects. Reported complications include trismus, loss of appetite, chest pain, aching muscles, sweating, and high fever.[1,2] To our knowledge, the only urological complication previously noted was urgency of micturition.[3]

The bladder is innervated by both α and β adrenergic neurones; β adrenergic neurones supply the detrusor muscle, α adrenergic fibres supply the trigone and the bladder neck. Blocking α adrenergic fibres relaxes the bladder neck, thus relieving symptoms of urinary retention. Conversely, stimulation of α adrenergic fibres can produce bladder neck dysfunction or closure. 3, 4-Methylenedioxymethamphetamine is a potent α adrenergic agonist, and its misuse in this case almost certainly led to acute urinary retention. The effect of amphetamine on the bladder has been reported previously.[4] In view of this case, and the widespread use of 3, 4-methylenedioxymethamphetamine we recommend that synthetic amphetamine misuse be considered in cases of unexplained urinary retention among young people.



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Large doses of "ecstasy" can cause retention of urine

1 Henry J. Ecstasy and the dance of death. *BMJ* 1992;305:5-6.
2 Sternback GL, Varon J, "Designer drugs"—recognising and managing their side effects. *Postgrad Med* 1992;91:169-76.
3 Greer J, Tolbert R. Subjective reports of the effects of MDMA in a clinical setting. *J Psychoactive Drugs* 1986;18:319-27.
4 Worsley J, Goble NM, Stott M, Smith PJB. Bladder outflow obstruction secondary to intravenous amphetamine abuse. *Br J Urol* 1989;64:320.

Leucopenia associated with lamotrigine

Ms R J NICHOLSON, Dr K P KELLY, and Dr I S GRANT (Western General Hospital, Edinburgh EH4 2XU) write: We report a case of septic shock secondary to leucopenia in a patient receiving lamotrigine for epilepsy.

A 35 year old woman who had been diagnosed as epileptic in December 1992 and was taking sodium valproate (400 mg three times daily) and propranolol (40 mg three times daily) for migraine was given lamotrigine in an attempt to control the complex partial seizures that stopped her driving. Ten days after treatment was started (25 mg daily for seven days, then 50 mg daily), she presented to her general practitioner with an erythematous rash, nausea, vomiting, dizziness, and a sore throat. Although lamotrigine was stopped, her condition deteriorated over the next 48 hours and she was admitted to hospital. On admission she was hypoxic (arterial oxygen pressure 6 kPa), hypotensive (70/40 mm Hg), and feverish (40°C), with a total white cell count of 0·6 × 10^9/l (neutrophils 0·3 × 10^9/l). She was transferred to this intensive therapy unit, where she was treated with high flow oxygen, fluid, and inotropes. Blood cultures grew *Staphylococcus aureus* and *Escherichia coli*, for which she received vancomycin, ciprofloxacin, and gentamicin.

Her condition stabilised over the next 72 hours, her rash resolving, fever abating, and white count rising to 1·9 × 10^9/l with normalised clotting. Her further recovery was complicated by two episodes of ventricular fibrillation associated with extreme electrolyte disturbances (hypokalaemia, hypomagnesaemia, and hypocalcaemia) of unknown cause. By 13 days after her initial admission to the intensive therapy unit her white cell count had risen to 5·1 × 10^9/l (neutrophils 2·3 × 10^9/l). She made a full recovery.

Lamotrigine has been heralded as a promising new drug for patients with epilepsy, with a "low level of clinically significant side effects".[1,2] The Committee on Safety of Medicines has received four other reports of leucopenia and six of neutropenia associated with lamotrigine (personal communication). These cases highlight a serious, and in our case life threatening, complication of this drug. We suggest close haematological monitoring of patients during the first few weeks of treatment with lamotrigine.

1 Brodie MJ. Lamotrigine. *Lancet* 1992;339: 1397-400.
2 Lamotrigine—an add-on antiepileptic. *Drug Ther Bull* 1992;30:75-6.