Following an uneventful intra-cavernosal injection of six micrograms of alprostadil, he developed satisfactory penile erection. But 20 min after the injection, he developed chest pain. He was taken to the nearest accident & emergency hospital where he was initially diagnosed as having unstable angina. Serial creatine kinase levels were 145 U/L, 794 U/L, and 243 U/L (the laboratory referrence range was 24-190 U/L). Creatine Kinase MB Isoenzyme level was 51 U/L (reference range: 0–16); percentage of total Creatine Kinase was 6.4% (reference range: 0.4–4.0). Serial ECG was suggestive of lateral wall myocardial infarction. There were no signs of cardiac failure. There was an uneventful recovery and he was discharged home in a stable condition and has been attending our follow-up clinic and has been doing well. He was advised not to use alprostadil to achieve penile erection; he and his wife agreed to this suggestion.

Suspected adverse cardiovascular reactions have been reported in five other patients who were using intra-cavernosal alprostadil for impotence (Table 1) (Committee on Safety of Medicines-personal communication). The British National Formularly Number 31 (March 1996) mention the following cardiovascular side effects for intra-cavernosal alprostadil: hypotension or hypertension, vasodilatation, supra-ventricular extrasystole, peripheral vascular disorder, dizziness. In a multicentre alprostadil study on the efficacy antd safety of intra-cavernosal alprostadil in men with erectile dysfunction. Linet and associates[1] reported side effects potentially related to hypotension, such as irregular pulse, lightheadedness, dizziness, diaphoresis, vasodilation, and vasovagal reaction in 1% of cases.

Understandably, a direct casual relationship between alprostadil and myocardial infarction is not established in the case reported herein, especially so, because of the short half-life of alprostadil (which is less than 1 min), and this patient had risk factors for cardiovascular disease. However, we believe that physicians caring for patients with spinal cord injury should discuss with the potential users of alprostadil, (and even before the administration of a test dose of alprostadil, as patient number 5 in Table 1 developed acute myocardial infarction 1.5 h after the test dose of alprostadil), a possible, albeit remote, association between intra-cavernosal administration of alprostadil and cardiovascular side-effects inclusive of myocardial infarction. The risk of serious cardiovascular side-effects of alprostadil appear to be high in the following categories of patients: (1) Age: above fifty years; (2) Pre-existing cardiac disease such as angina; (3) Co-existing systemic hypertension although hypertension might have been adequately controlled with drug(s) as in the case described above.

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Spinal canal obstruction detected by cerebrospinal fluid imaging

Gamma camera imaging of cerebrospinal fluid (CSF) consists of intrathecal administration of a miscible, non-toxic, diffusable substance which remains within the CSF compartment until being absorbed via normal pathways. The most widely employed agent is indium (^{111}In-DPTA) which has a physical half-life of 2.8 days.[1] After injection of the agent (500–800 microcuries) into the lumbar subarachnoid space, activity ascends in the spinal canal reaching the basal cisterns in approximately 4 h in adults.[1] Subsequent images over the next 24 h demonstrate ascent through the intracranial subarachnoid spaces.[1]

When diagnostic (spinal anaesthesia) or therapeutic (baclofen, morphine) intrathecal interventions are planned in patients with spinal cord injury (SCI), CSF imaging may be used to verify spinal canal patency. It is more sensitive than either spinal MR imaging or CT-myelography in detecting compartmentalization of CSF pathways.[2] During CSF imaging, spinal canal obstruction is detected when rostral spread of activity is either delayed or arrested.[1]

We recently reviewed 29 SCI patients who underwent CSF imaging over a 3 year period, prior to such interventions (23 – intrathecal baclofen, spasticity; 6 – diagnostic spinal anaesthesia, pain). Seven patients (24.1%) demonstrated spinal canal obstruction (6 – complete, 1 – partial) in the anatomical area of the neurological injury. CSF samples were collected immediately prior to lumbar subarachnoid injection of indium. Six patients had an increase of CSF protein (ie 97–237, normal range: 10–60 mg/dl). However, 3 patients without evidence of spinal canal obstruction also demonstrated elevated CSF protein levels (81–159 mg/dl).

The presence of spinal canal obstruction may reduce the diagnostic value of spinal anaesthesia during chronic pain assessments, since an injected local anesthetic will not reach sources of nociception cephalad to the area of obstruction, necessitating additional or separate procedures.[3] Use of intrathecal baclofen and morphine will also be affected by spinal canal obstruction since the pharmacodynamics of these agents are influenced by rostral 'washout'.[4] If an intrathecal agent is continuously infused below the area of spinal canal obstruction, local CSF concentrations may progressively increase to the extent that neurotoxicity or systemic absorption may occur.

It is not known currently whether spinal canal obstruction may have other important or significant ramifications for patients with SCI. However, the high incidence in our patient series warrants closer examination and study of this finding.

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