

SHORT REPORTS

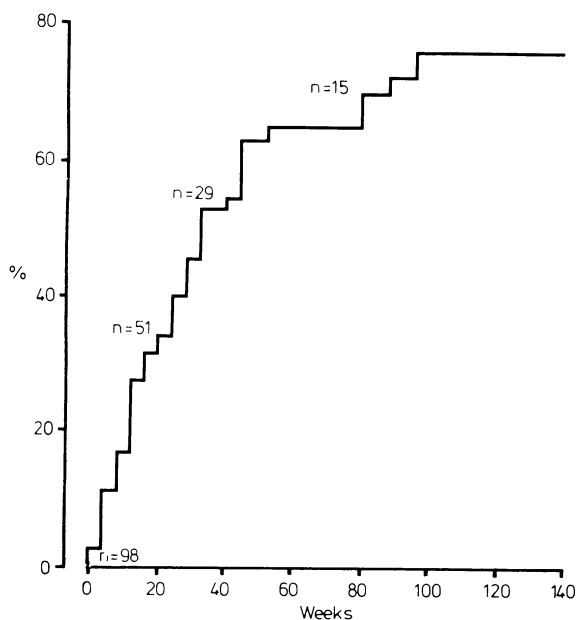
High incidence of amiodarone-induced photosensitivity in North-west England

Amiodarone is a potent cardiac antiarrhythmic agent which has been used in Europe for more than a decade but which has only recently become available in Britain. Facial pigmentation and photosensitivity are well recognised side effects,¹⁻⁴ but their incidence is disputed. We report the incidence of photosensitivity in a large group of patients receiving amiodarone at a regional cardiothoracic centre.

Patients, methods, and results

Ninety-eight of 114 patients who had received amiodarone for up to three years were assessed at a special dermatology clinic. Of these, 83 were still receiving amiodarone; eight had stopped treatment because of photosensitivity, five because it was no longer required, one because it was ineffective, and one because of headaches. Three of the 16 non-attenders had died; none of the remainder was known to have cutaneous disease.

Age, sex, concurrent medication, and skin type⁵ (an indication of inherent susceptibility to sunburn) were recorded. Duration of treatment and total cumulative dose and maintenance dose of amiodarone received were noted; in addition, the corresponding values when symptoms first appeared were



Cumulative percentages of patients becoming susceptible to photosensitivity during treatment with amiodarone.

estimated. After clinical assessment each patient was placed in one of four categories: (a) no photosensitivity, patients showing normally expected reaction to sun exposure; (b) mild photosensitivity, symptoms definitely abnormal for degree of sun exposure but not severe enough to restrict normal activity; (c) moderate photosensitivity, protective measures (for example, hats and long sleeves) or restriction of normal outdoor activities required; and (d) severe photosensitivity, reactions necessitating hospital treatment or major restriction of normal activity or withdrawal of drug.

Photosensitivity occurred in 54 patients (mild 25, moderate 17, severe 12), eight of whom had to stop treatment. No patient showed evidence of primary photosensitivity, but four with mild and one with moderate symptoms were taking other potentially photosensitising drugs (thiazides four, chlorpropamide one). Almost all symptoms reported by patients occurred in Britain during the spring and summer. In most cases symptoms began within two hours of exposure to sun, often within half an hour. Typically, a sensation of burning or tingling of exposed skin would be followed by erythema, which would normally disappear by the following day but which in severe cases lasted up to a week. Two patients had blistering of the skin and one required admission to hospital after one morning's exposure to the sun in Crete. Photosensitivity persisted for up to four months after treatment had stopped. Neither window glass nor sunscreens afforded protection, but most patients were

able to continue treatment by limiting exposure and wearing protective hats and clothing. Several mildly affected patients considered the resultant tanning a positive boon.

Susceptibility to photosensitivity was not related to sex, skin type (97 patients were white), duration of treatment, or cumulative dose or daily maintenance dose (mode 400 mg), but severe reactions tended to occur in younger patients. The cumulative percentage of patients affected within two years of starting treatment was over 70% (see figure). Blue-grey pigmentation of facial skin was seen in three patients, all of whom had received amiodarone for at least two years. No other cutaneous side effects were noted.

Comment

The incidence of amiodarone-induced photosensitivity is commonly quoted as 10% or less.¹⁻⁴ We have found a much higher incidence which, even after exclusion of mildly affected patients, is still nearly 30%. The high proportion of patients affected, the rapid onset of symptoms, and the lack of protection given by window glass and sunscreens suggest a phototoxic reaction to long-wave ultraviolet radiation. Whereas pigmentation, although persistent and cosmetically disfiguring, was infrequent, photosensitivity was very common and was the main reason for stopping treatment. Avoidance of bright sunlight and the wearing of protective hats and clothing may be the price which some patients must pay for a highly effective treatment.

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¹ Marcus FI, Fontaine GH, Frank R, Grosgeat Y. Clinical pharmacology and therapeutic applications of the antiarrhythmic agent, amiodarone. *Am Heart J* 1981;101:480-93.

² Simpson WT. A review of the therapeutic results and unwanted effects of amiodarone. In: Simpson WT, Caldwell ADS, eds. *Amiodarone in cardiac arrhythmias: Royal Society of Medicine International Congress and Symposium Series No 16*. London: Academic Press and Royal Society of Medicine, 1979:45-52.

³ Labouche F, Balouet G, Daoulas R, et al. Les accidents cutanés de l'amiodarone. *Revue générale à propos de 15 observations personnelles Arch Med Ouest* 1973;5:37-49.

⁴ Anonymous. Amiodarone—a new type of antiarrhythmic drug. *Drug Ther Bull* 1981;19:86-8.

⁵ Wolff K, Gschnait F, Hönigsmann H, Konrad K, Parrish JA, Fitzpatrick TB. Phototesting and dosimetry for photochemotherapy. *Br J Dermatol* 1977;96:1-10.

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Subdural haematoma as a complication of spinal anaesthetic

Intracranial bleeding after spinal puncture for diagnostic or anaesthetic purposes has been previously reported as an exceptionally rare event in patients without preexisting intracranial disease. Recently, Eerola et al¹ found 12 such patients quoted in five reports. A critical review of these, however, shows that at least nine of these patients had a possible predisposing cause present either before or at the time of the procedure (see table). Only three of the quoted cases could be considered to have been previously healthy and we report a further two.

Review of cases of intracranial bleeding after spinal puncture for diagnostic or anaesthetic purposes

Author	Year	Cases		Procedure	Possible aetiologic factors	Outcome
		Age (years)	Sex			
Gerlach*	1949	69	M	Spinal anaesthesia	Few details recorded	Died
Welch*	1959	42	F	Retropubic prostatectomy for retention (L2/3 spinal anaesthesia, 22 gauge needle)	Chronic bronchiectasis; intraoperative coughing	Died
Arseni*	1970	42	F	Cholecystectomy under spinal anaesthesia	Prolonged intraoperative hiccups	Recovered
		27	M	Spinal anaesthesia for epigastric hernia (1), hydrocele (1), inguinal hernia (2), haemorrhoids (2)	Postoperative hypotension, dehydration, vomiting, and predisposing vascular factors	All recovered
		30	M			
		46	M			
		49	M			
58	M					
Pavlin*	1979	23	F	Inadvertent spinal tap during attempted epidural anaesthesia for labour (18 gauge needle)	None	Recovered
		37	M	Spinal anaesthesia for rectal abscess (3 failed attempts with 25 gauge needle followed by success with 22 gauge needle)	Head trauma three years before; previous spinal anaesthesia; myelogram (normal) two weeks before (18 gauge needle)	Recovered
Edelman*	1980	22	F	Inadvertent spinal tap during attempted epidural anaesthesia for labour (16 gauge needle)	None	Died
Present cases	1982	29	F	Inadvertent spinal tap during attempted epidural anaesthesia for labour (18 gauge needle)	None	Recovered but persistent visual loss
		67	M	Uneventful spinal anaesthesia for inguinal herniorrhaphy (22 gauge needle)	None	Died

*Quoted by Eerola *et al.*¹

Case reports

Case 1—A previously healthy 29-year-old woman was admitted as an emergency complaining of headache, drowsiness, unsteadiness of gait, and visual disturbance. At the delivery of her third child some four weeks before an attempted epidural anaesthetic was abandoned because of inadvertent spinal puncture with an 18-gauge Tuohy needle. Non-specific generalised headaches had occurred a few days after delivery and had steadily worsened until the week before admission, when frequent vomiting, blurring of vision, and clumsiness of all four limbs became apparent. Examination on admission showed marked drowsiness, bilateral ptosis with external ophthalmoplegia, severe papilloedema, mild neck stiffness, and mild right-sided limb weakness. Bilateral subdural haematomas, larger on the left, were confirmed by carotid angiography, and a total of 100 ml of blood was removed through bilateral parietal burr holes. The patient was left with severe permanent visual defects.

Case 2—A fit 67-year-old man underwent bilateral inguinal herniorrhaphy under spinal anaesthesia performed uneventfully with a 22-gauge needle. Three days postoperatively he developed generalised headaches which persisted and were accompanied by a fluctuating level of consciousness but no other symptoms until his admission 10 days later. Examination showed a drowsy, disorientated man with generalised hyperreflexia and extensor plantar responses but without localising signs or papilloedema. Computed tomography showed a large left-sided subdural haematoma, which was evacuated within four hours of admission. Postoperatively his level of consciousness deepened and he died eight days later. Necropsy confirmed the diagnosis of left-sided subdural haematoma.

Comment

Although rare, subdural haematoma can undoubtedly occur in previously fit, healthy patients after spinal puncture even when it is uncomplicated or performed with a small diameter needle. Various aetiological mechanisms have been proposed.^{2,3} There is strong circumstantial evidence to implicate the dural puncture itself rather than any associated drugs or other procedures. Correlation between needle size and the incidence of headache after spinal puncture is well documented⁴ and persistent leakage of cerebrospinal fluid after puncture has been observed days after the event at necropsy and up to three weeks later with radioisotope myelography.⁵ Any rise in cerebrospinal fluid pressure at the level of the puncture (from coughing or straining) would also increase the loss of fluid. Reduced volume due to persistent leakage of cerebrospinal fluid could lead to brain sagging with traction on delicate blood vessels causing rupture and subsequent formation of an intracranial haematoma.

In the reported cases mortality was high (four out of 14) with half the deaths occurring in those with no predisposing factors. In those reported cases for whom sufficient clinical details are recorded the cardinal feature was severe headache beginning within two or three days of the procedure and persisting or worsening. Focal neurological symptoms or signs were variable and often a late feature.

With the increasing use of spinal and epidural anaesthesia it would seem prudent to use the smallest diameter needle necessary and to be aware of the possibility of subdural haematoma in those patients in whom severe headaches persist for more than a week postoperatively.

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- 1 Eerola L, Kaukinen L, Kaukinen S. Fatal brain lesion following spinal anaesthesia. Report of a case. *Acta Anaesthesiol Scand* 1981;25:115-6.
- 2 Frankson C, Gordh T. Headache after spinal anaesthesia and a technique for lessening its frequency. *Acta Chir Scand* 1964;94:413.
- 3 Lee JA, Atkinson RS. *Sir Robert MacIntosh's lumbar puncture and spinal analgesia: intradural and extradural*. Edinburgh, London, New York: Churchill Livingstone, 1978.
- 4 Phillips OC, Ebner H, Nelson AT, Black MH. Neurological complications following spinal anaesthesia with lidocaine. A prospective review of 10 440 cases. *Anesthesiology* 1969;30:284.
- 5 Leiberman LM, Tourtellotte WW, Newkirk TA. Prolonged post-lumbar puncture cerebrospinal fluid leakage from the subarachnoid space demonstrated by radioisotope myelography. *Neurology* 1971;21:925.

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Toxic-shock syndrome: four cases in a London hospital

Toxic-shock syndrome has become well recognised in the United States during the past two years and about 90% of cases have been associated with tampons. In contrast there have only been a few published British reports.^{1,2} We describe four cases of toxic-shock syndrome, one of which was associated with a postpartum breast abscess. All four patients were admitted as emergencies from the Kensington area during a 15-month period.

Case reports

Cases 1-3—Three 18-year-old women presented with the major criteria for toxic-shock syndrome, including high fever, erythematous rash, desquamation, and either history of syncope during the previous 24 hours or severe hypotension (table). One of them (case 3) had had similar but milder symptoms during her last menstrual period. On admission all were dehydrated with biochemical evidence of renal and hepatic dysfunction. Blood cultures, faeces, and throat swabs on admission yielded no growth of *Staphylococcus aureus* or other pathogens. Penicillin-resistant entero-