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		M	Spinal anaesthesia	Few details recorded	Died
Welch*	1959	69	M	Retropubic prostatectomy for retention (L2/3 spinal anaesthesia, 22 gauge needle)	Chronic bronchiectasis; intraoperative coughing	Died
		42	F	Cholecystectomy under spinal anaesthesia	Prolonged intraoperative hiccups	Recovered
Arseni*	1970	27	M	Spinal anaesthesia for epigastric hernia (1), hydrocele (1), inguinal hernia (2), haemorrhoids (2)	Postoperative hypotension, dehydration, vomiting, and prediposing vascular factors	All recovered
		30	M			
		46	M			
		49	M			
		58	M			
		62	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 documented[4] 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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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-

toxigenic strains of *Staph aureus* that produced enterotoxin F were isolated from high vaginal swabs from each patient (see table). Treatment with intravenous fluids and appropriate anti-staphylococcal antibiotics was followed by complete clinical resolution.

Cases of toxic-shock syndrome: clinical features and properties of Staphylococcus aureus *strains isolated*

	Case 1	Case 2	Case 3	Case 4
Date of presentation	Dec 1980	Oct 1981	Nov 1981	March 1982
Age (years)	18	18	18	35
Temperature ≥38·9°C	+	+	+	+
Scarlatiniform rash	+	+	+	+
Desquamation during convalescence	+	+	+	+
Syncope during previous 24 hours	+	−	+	+
Blood pressure (mm Hg) on admission	100/55	80/50	60/30	80/60
Profuse watery diarrhoea	+	+	+	+
Myalgia	+	+	+	+
Conjunctival hyperaemia	+	+	+	+
Vaginitis on admission	+	+	−	−
Symptoms associated with menstruation	+	+	+	−
Tampon present	Lillet	Lillet Super	Lillet Super Plus	−
Staph aureus strain isolated	+	+	+	+
Site of isolation	High vaginal swab*	High vaginal swab†	High vaginal swab	Breast abscess
Phage type	29/52/80	83A	29/52	29/25/80
Enterotoxins detected	A + F	B + D + F	A + F	A + F

*A phage group II strain which produced epidermolytic toxin A was also isolated from vaginal and nose swabs.
†A non-typable strain that produced enterotoxin F alone was isolated from the nose of this patient.

Case 4—A 35-year-old Filipino woman presented with a two-day history of pain in the right breast, fever, vomiting, and profuse watery diarrhoea three weeks after forceps delivery of a healthy infant. While in the maternity hospital the baby was reported to have developed a sticky eye, but no bacteriological report was available. On examination the patient had a fever of 39°C, her blood pressure was 80/50 mm Hg, and her right breast was diffusely tender. She had severe muscle tenderness but results of vaginal examination were normal for three weeks post partum. Investigations showed a neutrophil leucocytosis with a platelet count of 75×10^9/l, abnormal electrolyte values (sodium 128 mmol(mEq)/l), urea concentration 29·5 mmol/l (177 mg/100 ml), and creatinine concentration 583 mmol/l (5·8 mg/100 ml). Serum creatinine phosphokinase activity was raised at 332 IU/l, calcium concentration low at 1·6 mmol/l (6·5 mg/100 ml), and alkaline phosphatase activity 234 IU/l. Blood cultures, high vaginal swabs, faeces, and throat swabs collected on admission yielded no *Staph aureus* or other pathogens. Septicaemic illness or toxic-shock syndrome was diagnosed and she was given intravenous antibiotics and fluids. On day 1 she passed only 300 ml urine and developed a macular rash on face and trunk. On day 3 her right breast became more swollen and an abscess was drained: the pus yielded a pure heavy growth of *Staph aureus* (phage type 29/52). She improved rapidly and on day 9 showed the characteristic skin desquamation on palms, soles, and face. On discharge her renal function was normal and abscess wound dry. The strain was subsequently shown to produce enterotoxins A and F.

Comment

That three girls presented with tampon-associated toxic-shock syndrome in this area within 11 months indicates that the syndrome may not be as rare as generally believed. There is, however, a much higher proportion of young women than average in the St Stephen's Hospital catchment area. There has been a suggestion that the cellulase activity of certain bacteria on carboxymethylcellulose may occur in vivo and that this may contribute to the pathogenesis of toxic-shock syndrome.[3] Interestingly carboxymethylcellulose was not contained in the tampons used by our patients. Enterotoxin F is considered to be important in the aetiology of toxic-shock syndrome.[4] Examination of the *Staph aureus* isolates from our patients showed that they belonged to different enterotoxin-F-producing strains.

There have been reports from the United States of toxic-shock syndrome postpartum.[5] These cases were often associated with the use of tampons. Our case 4 was not associated with tampons and is the first reported case during the puerperium in Britain.

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Failure of rifampicin and co-trimoxazole in Q fever endocarditis

Information on the response of Q fever endocarditis to the newer antibiotics is slow to accumulate because of the rarity of the disease. Recently, the primacy of tetracycline in its treatment[1] has been challenged, at least in vitro, and by one clinical success with rifampicin,[2] and by co-trimoxazole as a potential first choice.[3] We report the complete failure of these and other antibiotics even to suppress *Coxiella burnetii* infection in a patient in whom tetracycline could not be used.

Case report

A male tannery worker aged 38 years was referred to this unit because of the recurrence of fever with embolic phenomena four weeks after a culture-negative endocarditis had apparently been successfully treated with penicillin and gentamicin. The underlying lesion was a discrete subvalvar aortic stenosis. Blood culture results again were negative but echocardiography showed massive highly mobile echoes in the aortic root; the complement-fixation test result for Q fever was positive at a titre of 1/320 (phase 1 and 2).

Although the initial response to tetracycline was favourable the drug had to be withdrawn after a week because of a severe pancreatitis and hepatic and renal failure. Treatment was continued with co-trimoxazole alone (960 mg three times daily). The immediate clinical response seemed very satisfactory, but after 10 months of continuous treatment there was relapse with fever, Osler's nodes, and a rise in Q fever titres to 1/640 (phase 1) and 1/5120 (phase 2). Lincomycin 2 g daily was added to his regimen with a dramatic initial response followed by relapse after one month of treatment. Rifampicin 600 mg daily was substituted for the lincomycin, the co-trimoxazole being continued throughout. The immediate clinical response was good, but since this seemed likely to be as delusory as the response to previous antibiotics a surgical exploration of the valve was undertaken after three weeks of rifampicin. The aortic valve was heavily infected, with a perforation of one cusp; vegetations were attached to the cusps and extended below to the subvalvar diaphragm. Both valve and diaphragm were excised and a disc-valve prosthesis inserted. The vegetations showed colonies of *C burnetii* on microscopy. Rifampicin and co-trimoxazole were continued during and after the operative period. Two months after operation he felt well but the Q fever titres remained high and a diastolic murmur not heard in the immediate postoperative period was present, suggesting a paraprosthetic leak.

Four months later he was readmitted with florid endocarditis and gross aortic reflux. There was no response to the addition of either lincomycin or erythromycin and he died three weeks later, after 16 months of continuous treatment with co-trimoxazole and five months with rifampicin and co-trimoxazole in combination. At necropsy there was extensive dehiscence of the prosthetic valve ring and many fresh vegetations composed microscopically of clumps of *C burnetii*.