Letters to the Editor

Death during prostatectomy

In a recent report from this hospital, three cases, one fatal, were described, of cardiovascular collapse due to absorption of irrigation fluid, occurring during transurethral resection of the prostate. However, the implications for pathologists conducting necropsies in such cases were not emphasised. Since then a further possible fatal case has occurred, as well as several “near misses” that were successfully treated. We now report the necropsy findings in these two fatal cases and stress the role of the pathologist in identifying the cause of death in such cases.

The first case, as described in the original paper, was a man aged 76 who collapsed and died 30 minutes after the start of a routine transurethral resection of prostate (TUR). The anaesthetist, who had diagnosed and successfully treated other cases, suspected the diagnosis and took a blood sample for estimation of the serum Na+ during attempted resuscitation. This revealed a concentration of 86 mmol/l. At necropsy, there was evidence of pulmonary oedema and congestion and small effusions in all the body cavities. There was no evidence of significant cardiac disease, and the remainder of the necropsy was essentially negative. Further confirmation of absorption of irrigating fluid was obtained by estimation of the glycine concentration in the same blood sample as was used for the Na+ estimation. A level of 80 mmol/l (600 mg/100 ml) was reported. The concentration of glycine in the irrigating fluid is 1·5 g/100 ml of water. Glycine, a low molecular weight amino acid, is presumably distributed throughout the extracellular fluid, given time. However since death can occur rapidly, the degree of equilibration at the time of collapse or death is difficult to assess. Despite these limitations, one can calculate that at least three litres and probably more, was absorbed.

The second patient was a man aged 77 who was admitted to a medical ward for investigation of drop attacks. Two days after admission he developed acute retention. Cardiological studies revealed evidence of ischaemic heart disease, and a systolic murmur was also apparent. A prostatectomy three years earlier had been uneventful. Two weeks after admission, a TUR was performed. However, 10 minutes after the start of the procedure bradycardia developed followed by asystole, and resuscitation was unsuccessful. At necropsy, there was evidence of severe pulmonary oedema (right lung 850 g; left lung 770 g). The heart (430 g) showed evidence of left ventricular hypertrophy, patchy myocardial fibrosis and severe calcific atheroma involving particularly the right coronary and left circumflex branches. In addition, there was evidence of aortic stenosis due to degenerative calcification of a congenitally bicuspid valve. These findings were thought to be sufficient to explain death. However a sample of blood taken at necropsy showed a serum Na+ of 123 mmol/l despite an infusion of 200 ml of 8·4% sodium bicarbonate administered by the anaesthetist, who suspected hyponatraemia.

The interpretation of the results in this second case is much more difficult due to two factors. The necropsy was performed 65 hours after death, and there is evidence that Na+ concentrations fall after death, although there is considerable individual variation in the rate of fall. In addition, the patient was given 200 ml of 8·4% bicarbonate during resuscitation. In retrospect, we should have estimated the Na+ level in vitreous humour, since the concentrations at this site are much more stable post mortem, and estimated the blood glycine. Only then would we have been able to assess the degree of absorption. The amount of fluid absorbed and the degree of hyponatraemia was undoubtedly much less than in the first case, but with significant cardiac disease, it seems reasonable that lesser degrees may be fatal.

In conclusion, the message seems clear. When conducting a necropsy on a man who has collapsed and died during or soon after a prostatectomy, the possibility of absorption of the irrigation fluid should always be considered, and steps taken to confirm or refute the diagnosis. If the anaesthetist has taken a blood sample during the collapse and attempted resuscitation, one should estimate serum Na+ and glycine concentrations from this specimen. If the only material available is that obtained at the time of necropsy, a sample of vitreous should be taken for Na+ estimation and blood sent for glycine estimation. In our opinion, it appears likely that this condition is under-diagnosed, largely due to pathologists being unaware of it. Certainly since it has been recognised by the urologists and particularly the anaesthetists, more cases are being diagnosed, although fortunately the majority are not fatal.

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References


Resistance of urine pathogens to trimethoprim in Finland

Since 1973, trimethoprim has been recommended to be used as the sole antimicrobial drug for the prophylactic treatment of chronic and recurring urinary tract infection (UTI) in Finland. Unfortunately, there is no baseline against which to compare the present findings of trimethoprim resistance and no sufficiently extensive surveys have so far been published. The epidemiological information would also be extremely useful in other countries which start using trimethoprim alone for prophylactic treatment of UTI. We therefore analysed consecutive urine isolates of E coli, Klebsiella sp and Proteus sp at Roanvemi Public Health Laboratory, the Clinical Laboratory of Savonlinna Central Hospital and the Department of Medical Microbiology, University of Oulu. Samples analysed at the first two laboratories were obtained from the Central Hospital and its sur-
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