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Case Report:

Iatrogenic acute angle closure glaucoma masked by general anaesthesia and intensive care

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Acute angle closure glaucoma is a medical emergency which can result in blindness. As it is very painful patients are usually referred rapidly to an ophthalmologist. If it occurs following general anaesthesia however, the diagnosis may not be considered and symptoms such as pain and vomiting wrongly attributed. Delayed diagnosis puts the patient at risk both from the ocular complications of acute angle closure glaucoma, and also from inappropriate investigation and intervention. We report an illustrative case where bilateral acute angle closure glaucoma followed a general anaesthetic. The correct diagnosis was delayed for 11 days.

CASE REPORT. A 66 year old lady underwent abdominal hysterectomy. During her general anaesthetic she was preoxygenated. Her intubation with an endotracheal tube was moderately difficult. The intravenous anaesthetic agents administered (in order of use) were; atropine, thiopentone, suxamethonium, vecuronium, cyclizine, neostigmine, doxapram and glycopyrrolate. There were no intra-operative complications but extubation was difficult. Immediately post- operatively she became hypoxic, as assessed by her oxygen saturation, and was reintubated and transferred to the Intensive Care Unit. The clinical impression at that time was that she had aspirated during surgery and developed left ventricular failure. A chest radiograph confirmed pulmonary oedema. She was ventilated for two days and during this time developed a pneumothorax following the insertion of a central venous line. This was successfully treated with the insertion of a chest drain. In the Intensive Care Unit she received intravenous frusemide, digoxin, amoxycillin and subcutaneous heparin. Clinically at this time she appeared to have developed a chest infection with the development of coarse crepitations at the lung bases, and to aid respiration nebulised solutions of ipratropium bromide and salbutamol were commenced. A subsequent chest radiograph showed lung fields to be clear and her pneumothorax to have resolved.

She returned to the ward on the fourth postoperative day with nausea and vomiting. Her right eye was noted to be injected but she made no complaint of specific ocular discomfort. Her abdominal wound dehisced and was resutured under general anaesthetic. She made an uneventful recovery from this procedure. Over the following two days she received repeated doses of intramuscular pethidine and cyclizine for pain and nausea. She also continued to receive nebulised solutions of ipratropium bromide and salbutamol.

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On day six following her first operation she became increasingly confused and aggressive and appeared to have visual hallucinations. She was also noted to have markedly reduced vision with bilateral injected eyes. She complained of eye discomfort at that time. Her eye condition was treated with topical chloramphenicol eye drops and further intramuscular pethidine injections for pain relief. She was treated with oral thioridazine for confusion, but this worsened on day seven and intramuscular chlorpromazine hydrochloride was administered. A provisional diagnosis of cerebral infarction was made. However computerised tomography of brain was normal.

Subsequently her confusion lessened and her eyes although remaining injected became less painful. On day eleven following her first operation she was seen by an ophthalmologist. He noted visual acuity as hand movements in each eye, bilateral corneal oedema, shallow anterior chambers, non-reactive mid-dilated pupils and raised intraocular pressures. He also noted that the patient was hypermetropic (by spectacle measurement) and had bilateral lens opacities. A diagnosis of spontaneously resolving bilateral acute angle closure glaucoma was made and the patient treated with oral acetazolamide and pilocarpine, dexamethasone and betaxolol eye drops. The intra-ocular pressures after this treatment were 44 mmHg and 15 mmHg on right and left side respectively.

Her corneal oedema slowly resolved over the following month. No specific glaucomflecken were noted but sectorial iris atrophy was present in her right eye consistent with the previous acute rise in intra-ocular pressure. Gonioscopy revealed 270 degree permanent angle closure in her right eye and a 360 degree grade 1 angle in her left eye. She underwent bilateral laser iridotomies two months later. Persistent elevated intra-ocular pressures of 40 mmHg od and 17 mmHg os and cataract development necessitated subsequent combined right trabeculectomy, cataract extraction and intra-ocular lens implantation. Five months after this surgery she still requires pilocarpine eye drops in her left eye and betaxolol eye drops in both eyes to control intra-ocular pressures. Current visual acuity is 6/9 and 6/12 in her right and left eyes respectively and current intra-ocular pressures are 21 mmHg ou. Optic discs show minimal cupping with a 0.2 cup – disc ratio in each eye and intact neuroretinal rims, consistent with her hypermetropia. Visual fields show marginal field loss with a nasal arcuate scotoma in the right eye only

DISCUSSION

Acute angle closure glaucoma occurs in 0.1 % of the British population over the age of 40. It affects females four times more commonly than males and is more common in patients who are hypermetropic or who have an enlarged lens due to age or cataract. The acute rise in the intra-ocular pressure produces pain, vomiting, blurred vision and haloes (due to corneal oedema). Sequelae include iris atrophy, cataract, chronic angle closure glaucoma, chronic corneal oedema, glaucomatous visual field loss and anterior ischaemic optic neuropathy. Drugs which increase pupillary dilatation can precipitate acute angle closure glaucoma in a susceptible individual.[1,2,3] These drugs include anti-muscarinic agents such as ipratropium bromide, chlorpromazine bromide, thioridazine and cyclizine, all of which this patient received whilst suffering from acute angle closure glaucoma. In addition salbutamol, a β_2 adrenoreceptor agonist, can compound the problem as it increases aqueous humour production and thus acts in synergy with ipratropium bromide to increase intraocular pressure.[1]

It is likely that in this case the acute angle closure glaucoma was precipitated by the anti-muscarinic agents (intravenous cyclizine and nebulised ipratropium bromide) and nebulised salbutamol, which the patient received during and shortly after her first anaesthetic. Intravenous atropine administered in standard premedication doses has no effect on the

intra-ocular pressure in healthy[4] or glaucomatous eyes.[5] The postoperative vomiting which contributed to her wound dehiscence was probably caused by glaucoma as were the pain, nausea and visual hallucinations for which she received further inappropriate medication. The oral thioridazine and intramuscular chlorpromazine hydrochloride given for her confusion may have perpetuated and compounded the attack of glaucoma and also prevented the patient communicating her symptoms.

Cases of iatrogenic acute closed angle glaucoma have been described before.[1-3] What this case illustrates in particular is the need for a high index of suspicion in an intensive care situation where the classic signs of acute angle closure glaucoma can be masked by a patient's more obvious surgical or anaesthetic needs, and the diagnosis of underlying acute closed angle glaucoma can be delayed for several days. Failure to consider it in a patient with a red eye may result in drug therapy which can both further obscure the correct diagnosis and prolong the glaucomatous episode. Acute angle closure glaucoma is an important differential diagnosis in a patient with a painful red eye. If suspected, ophthalmic advice should be obtained urgently, so that further complications can be avoided. Early detection and treatment of acute angle closure glaucoma lead to a better visual outcome.[6]

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