Conversion from laparoscopic to open cholecystectomy

Sir,

I read the article by Tayeb et al[1] with interest. Even though the authors have identified the majority of risk factors for conversion to open cholecystectomy, I don’t know why the sex of the patient was not considered. Male gender has been a significant risk factor for a high conversion rate in majority of the studies.[2,3] Another factor is the time of surgery from the onset of symptoms. Patients undergoing intervention within 48 hours of the onset of symptoms experience a lower conversion rate to an open procedure.[4] A study from Belgium highlighted that preoperative C reactive protein serum level less than 10 mg% represent the best candidates for laparoscopic surgery.[5]

In another study, the American Society of Anaesthesiologist (ASA) class of more than 2 also predicted conversions in patients undergoing non-elective cholecystectomies.[6] Finally, no matter how much preoperative risk grading or a diagnostic score is done to predict difficult laparoscopic cholecystectomy, the experience of the surgeon is the foremost factor. There should always be a low threshold of conversion whenever he faces any difficulty irrespective of the preoperative predictability.

Bhattacharya K
Department of Surgery, Subham Hospital and Diagnostic Centre, Cooch Behar - 736101, West Bengal, India

Correspondence:
Kaushik Bhattacharya, E-mail: kaushik_srmc@rediffmail.com

References

Authors' Reply

Sir,

We appreciate the comments by Dr. Bhattacharya on our paper to identify the risk factors for conversion to open cholecystectomy (OC). Male gender though pointed out as a major risk factor for conversion; was not identified in our study[1] as a potential risk factor [Table 2]. Hence, we did not include it in the final multivariate model. The goal of our analysis using logistic regression was to present the best fitting yet the most parsimonious model describing the independent relationship of factors with conversion to OC. The criteria for selection of possible factors was based on a p value of less than 0.25 on univariate analysis,[2] and therefore ‘gender’ was not a good variable to be selected (P value = 0.39) for the final model. Use of p value of 0.25 as a screening criterion for selection of candidate factor is based on work by Mickey and Greenland on logistic regression. These authors have shown that the use of a more traditional P value of 0.05 on univariate analysis often fails to identify the factors that are known to be important.[3,4]

Dr. Bhattacharya has in fact extended and clarified other potential risk factors for OC very well. We believe that well-designed trials are needed that will present these risk factors in a more lucid way.

Tayeb M, Raza SA, Khan MR, Azami R
Department of Surgery, Faculty of Health Sciences, The Aga Khan University, Stadium Road, P.O. Box 3500, Karachi, Pakistan

Correspondence:
Syed Ahsan Raza, E-mail: ahsan.raza@aku.edu

Spontaneous macular haemorrhage in a patient on aspirin

Sir,

Aspirin is the most commonly used anti-platelet medication in conditions of myocardial and cerebral ischemia.[5] It is known, however, to have a dose-dependent effect on gastrointestinal haemorrhage[6] and can rarely cause fatal cerebral haemorrhages.[9]

We report a 68-year-old male who presented with a two-day history of loss of central vision in his left eye. His visual acuity was 6/5 and 6/36 in the right and left eye, respectively. Anterior segment examination was unremarkable. Fundus examination showed an area of sub-foveal haemorrhage of approxi-
mately one-and-half disc diameters (DD) in the left eye and drusens of age-related maculopathy (ARM) in the right eye.

There was no past ocular history of relevance. He was on treatment for hypertension and angina, and had undergone a coronary bypass operation in 1991. His medications included Isosorbide di-nitrite 20 mg, Atenolol 50 mg, Amlodipine 5 mg, Simvastatin 10 mg and Aspirin 75 mg daily. On further questioning, he complained of prolonged bleeding from minor wounds such that he had to have his chin cauterised after scratching while shaving few months before.

A week later, his vision dropped to finger-counting and the bleeding area enlarged to almost 3 DD, while the right eye remained unchanged [Figure 1]. His prothrombin time was marginally prolonged (14.4 sec; normal=11-13.8 sec). Full blood count, activated partial thromboplastin time and coagulation screen were normal. Three months later, the haemorrhage absorbed and left eye visual acuity improved to 6/18. He had an area of retinal pigment epithelium (RPE) atrophy at the macula and a central scotoma on Amsler chart.

Fluorescein angiography showed no signs of choroidal neo-vascular membrane (CNVM) in either eye but window defect hyperfluorescence corresponding to the areas of drusens and atrophic RPE [Figures 2 and 3] was seen. Upon a discussion with the patient’s physician, he was prescribed clopidogrel as it was felt to be a safer option.

In this report, an unprovoked sub-macular haemorrhage occurred in a non-myopic patient on 75 mg-a-day of aspirin, the dose commonly prescribed for thrombo-prophylaxis. Spontaneous choroidal haemorrhage attributable to aspirin has been reported before in patients with neo-vascular ARM[4] and recently in a patient with high myopia.[5] Old age and hypertension were additional risk factors for bleeding in those patients as well as in ours. Fluorescein angiography, however, did not disclose signs of active CNVM in our patient. Although the presence of an occult neo-vascular membrane at the time when the haemorrhage happened could not be entirely excluded, the bleeding tendency and increased prothrombin time strongly suggest that aspirin was the main predisposing factor for macular haemorrhage. Using Naranjo’s Algorithm, a 10-item questionnaire that assigns a numerical score to grade the overall probability of a drug related adverse event as either definite, probable, possible, or unlikely,[6] we graded macular haemorrhage relationship to aspirin as ‘possible’ (score=4).

Aspirin permanently inactivates prostaglandin synthase enzyme activity and hence inhibits the synthesis of thromboxane \( \text{A}_2 \) necessary for platelet aggregation and thrombus formation.[7] This mechanism is particularly important in the absence of an exogenous thrombogenic stimulus such as after spontaneous vessel rupture[8] or minor trauma,[9] where aspirin leads to increased bleeding. In major surgery, local thrombin production and collagen exposure lead to normal platelet stimulation thus preventing aspirin from causing excessive bleeding.[10]

The implications of this case are relevant to the large propor-
tion of elderly ophthalmic patients who are on aspirin and have signs of ARM and clinical manifestations of bleeding tendency. The benefits of keeping these patients on aspirin will need careful consideration against the potential risks that include the possibility of losing the central vision. It is yet to be determined if any of the other anti-platelets will be a safer alternative.

Elgohary MA, Gormley PD
Ophthalmology Department,
Essex County Hospital, Colchester, UK

Correspondence:
Mostafa A. Elgohary, E-mail: m.elgohary@doctors.org.uk

References


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Pituitary apoplexy following bilateral total knee arthroplasty

Sir,
A 65-year-old female patient with American Society of Anaesthesiologists physical status 2 was admitted for bilateral total knee arthroplasty. She reported a ten year history of Type II diabetes mellitus adequately controlled with oral glipizide 5 mg/day and hypertension controlled by oral enalapril 2.5 mg/day, metoprolol 25 mg x 2/day. ECG showed incomplete right bundle branch block (RBBB) with T wave inversion in all chest leads. A multigated acquisition (MUGA) showed a normal left ventricular function. All routine laboratory investigations were within normal limits.

After a failed attempt at combined spinal epidural anaesthesia, general anaesthesia was induced with fentanyl 100 μg IV, sodium thiopentone 250 mg IV and vecuronium bromide 4 mg IV. Anaesthesia was maintained with isoflurane in nitrous oxide, oxygen (66%, 33%), inavenous morphine. The blood sugar was 80 mg% preoperatively, 104 mg%, 182 mg% at 2 and 4 hours intraoperatively. Tourniquets were used on each limb in sequence resulting in minimal blood loss. Intraoperatively, except for a rise in blood pressure to 180/110 mmHg lasting for 5 minutes, which responded to inanavenous morphine, hemodynamics were stable. No hypotension occurred with bone cement insertion or deflation of the tourniquets. Surgery lasted for 4 hours. Neuromuscular blockade was reversed with IV neostigmine and glycopyrrolate. The trachea was extubated when the patient was breathing spontaneously and responding to verbal commands. In the post-anesthesia care unit (PACU) she remained stable and received IV boluses of fentanyl 20 μg each, (60 μg total) and intramuscular dicyclofenac sodium (75 mg) for analgesia. Twelve hours postoperatively subcutaneous low molecular weight heparin (LMWH, enoxaparin 20 mg) was administered for thromboprophylaxis.

The next day the patient complained of double vision, right ptosis and episodic emesis. Her blood pressure was 90/50 mmHg and central venous pressure was 15 cm H(2)O. Dopamine (5 mg/kg/min) infusion was started. On examination III, IV, and VI cranial nerve palsies and congestion of right fundus were present. Brain computerised tomographic scan the brain was suggestive of cavernous sinus thrombosis. Magnetic resonance imaging of revealed a large pituitary mass with haemorrhage diagnosed as pituitary apoplexy with paraspinal extension with pontine haemorrhage. Intravenous mannitol and dexamethasone were administered. Her blood pressure stabilized and the dopamine was stopped.

On the 2nd postoperative day, the patient’s vision deteriorated to perception of light, subsequently she became disoriented and unconscious. The right pupil was fixed, dilated and the left was constricted. Laboratory investigation showed normal thyroid functions and follicular stimulating hormone levels. However, serum luteinising hormone was 2.61 mIU/ml (normal range 8-33 mIU/ml), indicating hypopituitarism in a post-menopausal woman. Transsphenoidal hypophysectomy was performed and the patient mechanically ventilated for a day. The histopathological analysis revealed a pituitary adenoma with haemorrhage and necrosis consistent with pituitary apoplexy. The patient was discharged 4 weeks post surgery on steroid replacement therapy. The right-sided ptosis persisted, the right pupil was larger (3 mm) than the left (2 mm), but both the pupils had normal reaction to light. Her vision improved to finger counting at a meter with spectacles and she had bitemporal field losses.

Pituitary apoplexy (PA) is an acute life-threatening haemorrhage or infarction in the pituitary gland, leading to damage to the gland, and surrounding sellar structures depending on the increase in the size of the tumour. The commonest abnormality noted is III and IV cranial nerve involvement with ophthalmoplegia, diplopia, ptosis and mydriasis due to compression of the cavernous sinus.[1] Our patient presented with similar symptoms progressing to loss of consciousness.