

Pituitary apoplexy after laparoscopic surgery: a case report

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Abstract. – The occurrence of unprobable adverse events during laparoscopic surgery has increased over the years. Among them, pituitary apoplexy has been reported only twice. The increase in the abdominal pressure might play a role in the pituitary apoplexy, as well as hemodynamic instability, anticoagulant drugs and air-embolism due to insufflation of CO₂ during pneumoperitoneum. We report a case of pituitary apoplexy during laparoscopic resection of sigmoid colon.

Key Words:

Pituitary apoplexy, Pituitary adenoma, Laparoscopic surgery, Colon cancer, Gas embolism.

Introduction

With the increased employment of the laparoscopic surgical approach, a rise in rare incidents has become evident. One such rare incident is pituitary apoplexy, reported only twice since the year 2000^{1,2}. A recent study reports 55 such cases referring to a large UK regional neurosurgical center over a 25-year period (1985-2000). In all cases, apoplexy occurred on a preexisting adenoma, primarily a non-functioning one (84%)³. Head trauma, coagulopathies (or drugs affecting hemostasis) and the early postpartum period are well-known risk factors^{2,3} for this type of condition. Below we report on the third such case as described in literature of pituitary apoplexy during laparoscopic surgery and argue the possible risk factors as in relation to pre-existing literature.

Case Report

An 85-year-old patient admitted to our hospital for treatment of sigmoid colon cancer was scheduled for laparoscopic colorectal resection.

His anamnesis was positive for hypertensive heart disease which had caused him paroxysmal atrial fibrillation pharmacologically treated with dabigatran 110 mg twice a day as an anticoagulant. Due to intestinal bleeding it was withdrawn and substituted with low molecular weight heparin (calciparin 2000 IU twice a day) seven days before surgery. As elective surgery was initiated, with the patient in steep Trendelenburg position during insufflation of pneumoperitoneum (CO₂ to 16 mmHg) sudden hemodynamic instability occurred with marked hypotension (mean arterial pressure 30-40 mmHg) and bradycardia (20 beats per minute) unresponsive to atropine, condition resolved after administration of epinephrine 0.5 mg and deflation of pneumoperitoneum. The surgical technique was immediately switched to a laparotomic approach and the patient was moved to intensive care unit (ICU). Left palpebral ptosis, anisocoria (left > right), divergent strabismus and left eye mydriasis in the absence of photomotor reflex was detected during immediate clinical evaluation in ICU (Figure 1). Subcutaneous emphysema of the abdominal wall was also observed. Neurological examination was immediately requested and performed as well as head Computerized Tomography (CT) scan. The CT scan showed an increase of pituitary gland anterior-posterior diameter (about 17 mm) with a concomitant uneven enlargement of the sella turcica with thinned remodeled margins (Figure 1). These images were suggestive of a pituitary adenoma. In first post-operative day the patient's conditions were considered stable and ET tube was removed. A brain Magnetic Resonance Imaging was performed and the increased size of the pituitary gland (28 × 19 mm) was confirmed (Figure 2). Within the gland were detected hemorrhagic lesions. The gland exerted a modest compressive effect on the pituitary gland stalk, optic chiasm, left oculomotor (III cranial)



Figure 1. Postoperative head TC scan showing increase of pituitary gland anterior-posterior diameter and uneven enlargement of the sella turcica with thinned remodeled margins.

nerve and invasion of the left cavernous sinus. Complete evaluation of the endocrine system and neurosurgery consult were performed. Diagnosis of pituitary apoplexy with hypogonadotropic hypogonadism in nonsecreting pituitary macroadenoma was made. Dexamethasone 4 mg twice daily was prescribed and after 5 days of monitoring in the ICU, the patient was transferred to the ward. Neurosurgical consultant decided to wait for eventual resolution of the condition before attempting to remove the adenoma, and to date the patient has not been submitted to neurosurgery.

Discussion

Pituitary apoplexy is a rare but a potentially life-threatening condition if not properly diagnosed and treated. It is usually caused by a sudden ischemic or haemorrhagic infarction of a pre-existing pituitary adenoma. Apoplexy represents the first clinical manifestation in 80% of adenoma cases⁴.

Clinical signs derive from an acute compression of the perisellar structures that alter the gland's endocrine function with subsequent irritation of the meninges caused by the presence of blood in the subarachnoidal space. Signs and

symptoms develop acutely and include headache, nausea and vomiting, visual acuity and visual field defection (bilateral temporal hemianopsia starting from the superior quadrants), mydriasis, ptosis, ocular paresis and sudden-onset hypopituitarism⁵.

Eye-movement anomaly is due to the compression of the III, IV or V cranial nerve caused by the pressure applied by the mass to the surrounding structures (cavernous sinus and neurovascular bundle) and this is the first sign showed in $\frac{3}{4}$ of apoplexy cases⁶. Chronic clinical signs of hypopituitarism are amenorrhea in females, impotence in males, tiredness, anorexia, decreased libido, headache, depression, postural hypotension and hyponatraemia⁷. Surgical patients affected by pituitary adenoma are at risk for pituitary apoplexy. Major surgery (especially cardiac surgery), prolonged mechanical ventilation, embolization, coughing, unstable blood pressure, anticoagulation, crystalloid haemodilution and previous treatment with bromocryptine or radiotherapy are all considered risk factors⁶. Hypothalamic function might be compromised by the compression of the pituitary mass with consequent disregulation of blood pressure, temperature, cardiac rhythm and altered level of consciousness. These changes could be falsely attributed to the surgical iatrogenic effects and to general anaesthesia⁵. Moreover, hypopituitarism can

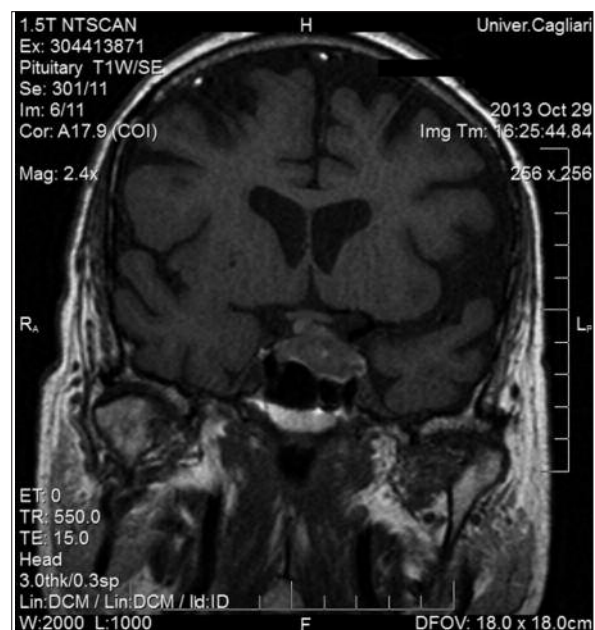


Figure 2. Postoperative T1 weighted MRI coronal section showing pituitary adenoma apoplexy.

determine different post-operative complications such as severe hypoglycemia, due to the reduced glucocorticoid effects of low cortisol response, water overload due to ACTH deficit and respiratory failure due to the loss of respiratory centre responsiveness to hypoxia and hypercapnia caused by hypothyroidism⁷. Keystones for a correct diagnosis are: the appropriate diagnostic suspicion, an early neuroimaging study (MRI scan is superior to CT scan in identifying hemorrhagic nature of the tumor), and urgent pituitary function tests. Treatment consists of acute administration of steroids and urgent surgical intervention for prompt decompression (transsphenoidal decompression), based on individualized cost/benefit ratio. Sole hormonal replacement therapy can be considered as an alternative, if vision is not affected and visual deficit is stable or improving^{8,9}. Pituitary apoplexy has been described after different surgical procedures: abdominal surgery, thyroidectomy, laparoscopic lumbar spinal fusion and bilateral knee arthroplasty⁹. Numerous cases of perioperative pituitary apoplexy have been reported during cardiac surgery as well^{8,10}. The pituitary vascular system is not controlled by an auto-regulated cerebral mechanism. Thus, the pituitary tissue is more susceptible to hypoperfusion, ischemia or intraoperative embolism, particularly during on-pump surgery. The cerebral edema induced by the cardiopulmonary bypass may cause a critical enlargement of a silent pituitary adenoma. Furthermore, the administration of an anticoagulant is another risk factor since it increases the risk of bleeding in the suffering pituitary tissue well^{8,10}. The exact pathophysiology of pituitary apoplexy is unclear but ischemic necrosis and/or haemorrhage are usually present in the histopathological samples¹¹. Numerous factors are involved in the pathogenesis of pituitary apoplexy during surgery: variations in blood pressure, blood dilution after crystalloid infusion, hypotension, anticoagulant therapy, excessive steroid secretion, and transient increases in intracranial pressure (ICP).

In 2001, Liu et al² reported the first case of pituitary apoplexy during laparoscopic anterior lumbar inter-body fusion. They assumed that the abdominal CO₂ insufflation during pneumoperitoneum reducing cerebral spinal fluid absorption determined an increase in ICP. In fact, the increased abdominal pressure during laparoscopic surgery determines an increase in inferior vena cava pressure with the reduction of the drainage of the lumbar venous plexus. Moreover, the

Trendelenburg position may contribute to exacerbate this increase of ICP. In cases of severe hypotension the hemorrhage may be related to the re-canalization of vessels and a reperfusion injury¹².

The second case of pituitary apoplexy following laparoscopic cholecystectomy was described in 2004 by Acar et al¹.

Conclusions

We've herein described the third case of pituitary apoplexy after laparoscopic surgery. Like the other two cases previously reported, we believe that the increase in the abdominal pressure might be the cause of the pituitary apoplexy. However, there are at least two other factors that we must consider: the systemic hypotension and anticoagulant drugs that the patient was assuming perioperatively and the risk of air-embolism due to insufflation of CO₂ during pneumoperitoneum (postoperatively the patient showed signs of mild subcutaneous emphysema). Embolism is considered one of the potential factors involved in the pathogenesis of pituitary apoplexy.

Aside from pituitary apoplexy's being one of the least-described cases in literature, as of today, we must focus our attention on the clinical evaluation of the patient after a laparoscopic procedure. An occasional anisocoria finding, ptosis or other ocular symptoms might hide – if not properly recognized – a series of life-threatening events for the patient.

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Conflict of Interest

The Authors declare that there are no conflicts of interest.

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