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Pulmonary embolism following tonsillectomy

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Abstract
Acute post-operative pulmonary embolism is a serious potentially life-threatening complication which is not anticipated in young patients undergoing non-major surgery. We report a case in which a 32-year-old previously healthy woman developed a major pulmonary embolism following tonsillectomy. Subsequent investigations revealed the presence of an occult malignancy. This case highlights the role of paraneoplastic hypercoagulable states in the aetiology of venous thromboembolism and the importance of thromboprophylaxis in the presence of confirmed or suspected malignancy. To our knowlege no case of major pulmonary embolism occurring after tonsillectomy has been previously reported.

Key words: Tonsillectomy; Pulmonary Embolism

Introduction

Venous thromboembolism (VTE) remains a major cause of morbidity and mortality in hospitalized patients. Assessment of individual patient risk factors and use of specific prophylaxis in moderate to high-risk patient groups is recommended for prevention.[1] However, some patient risk factors may not be apparent at the time of surgery, and choice of prophylaxis in surgical patients is complicated by risk and likely consequences of peri-operative haemorrhage. We report an unanticipated major thromboembolic event occurring in a young patient following tonsillectomy and discuss the probable aetiology and subsequent management.

Case report

A 32-year-old woman presented for interval tonsillectomy. She had a three-year history of recurrent tonsillitis and six weeks previously had presented with a peri-tonsillar abscess which had resolved with antibiotic therapy. She was a non-smoker and her past medical history was unremarkable. There was no personal or family history of venous thromboembolism, varicose veins, or procoagulopathic conditions, and she had no history of recent trauma or immobolization. She had been taking a third generation combined oral contraceptive pill (OCP) containing ethinyloestradiol 30 micrograms and gestodene 75 micrograms for the previous three years but had discontinued it six weeks pre-operatively. Examination revealed a body mass index (BMI) of 24 kg m^{-2} and mild upper cervical lymphadenopathy. The pre-operative full blood count was normal and a pregnancy test was negative. Graduated elastic compression stockings were applied pre-operatively.

During tonsillectomy, using sharp dissection, the right tonsillar tissue was noted to be irregular and friable and was sent for histological examination to rule out the presence of malignancy. Laryngoscopy at the end of the procedure revealed significant supraglottic oedema and therefore a decision was made to leave the endotracheal tube *in situ*. Intravenous hydrocortisone 100 mg six-hourly was commenced and the patient was transferred to the intensive care unit. Within one hour the patient was awake and co-operative with her own nursing care. Fourteen hours post-operatively an adequate leak was present with endotracheal tube cuff deflation, flexible nasendoscopy revealed marked reduction of supraglottic oedema and extubation proceeded uneventfully. On transfer to the high dependency unit (HDU) for continued airway monitoring, the patient was ambulant and self-caring.

Thirty-two hours post-operatively the patient developed acute dyspnoea, cough, sweating and cyanosis with a decrease in oxygen saturation to 85 per cent. Her heart rate increased to 130 beats min^{-1} but the arterial blood pressure remained stable. Chest examination and chest X-ray were unremarkable, 12-lead ECG showed a sinus tachycardia and initial arterial blood gases on an FiO2 = 0.6 were PaO_2 = 6.8 kPa, PCO_2 = 3.8 kPa, pH = 7.49. A presumptive diagnosis of acute pulmonary embolism was made. Supplemental oxygen with an $FiO_2 = 0.8$ and continuous positive airway pressure via facemask were given. Systemic anticoagulation with intravenous heparin was commenced.

Subsequent (^{133}Xe) ventilation/(^{99m}Tc) perfusion lung scan confirmed the diagnosis of pulmonary embolism. Lower limb venous ultrasonography was negative. Tonsillar histology revealed a high-grade non-Hodgkin's lymphoma (NHL) with staging investigations including computed tomography (CT) scan of abdomen and thorax confirming stage IIA disease.

The patient remained haemodynamically stable and showed a gradual clinical improvement allowing discontinuation of continuous positive airway pressure (CPAP) and reduction in FiO_2 over the next three days. Oral warfarin was substituted for heparin without any bleeding

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complications, and the patient was discharged from the HDU on day 6. At three-month follow-up she continues oral anticoagulation while undergoing chemotherapy and has had no recurrence of thromboembolic disease.

Discussion

Peri-operative pulmonary embolism is an uncommon complication in young, previously healthy patients undergoing non-major surgery and to our knowledge has not been previously reported following tonsillectomy.

Most fatal pulmonary emboli arise from lower limb deep venous thrombosis (DVT),[1] although in the majority of hospitalized patients with fatal pulmonary embolism, preceding VTE is not recognized.[2] Therefore identification of individual patient risk factors for VTE and use of appropriate prophylaxis in moderate to high-risk patient groups is the recommended approach to prevention of fatal and nonfatal pulmonary embolism.[1,3]

Age, type of surgery, and length of immobolization significantly influence risk of VTE and need for prophylaxis in surgical patients. This patient was young, previously healthy, and undergoing non-major surgery with anticipated early post-operative mobilization. Obesity has been identified as a risk factor for VTE, but the specific BMI which is associated with increased risk, and above which prophylaxis should be provided remains unclear. A BMI > 25 was a weak independent risk factor for VTE in a recent World Health Organisation study,[4] while other studies select a BMI $\geqslant$30 as associated with increased risk.[5] The most common complication of tonsillectomy is haemorrhage. Therefore, in this patient with no other association risk factors, and in the setting of early post-operative mobilization, a BMI of 24 was not considered an indication for specific thromboprophylaxis.

Oral contraceptive pills have been linked with a small absolute increase in the incidence of thromboembolic disease in many epidemiological studies,[6,7] with the risk being decreased by reduction in dosage of the oestrogenic component.[8] Recently concerns have been raised regarding a possible increased risk of VTE with the so-called third generation progestogen (gestodene and desogestrel)-containing pills.[4,5] Thus, the question of whether or not to discontinue oral contraceptive therapy prior to elective surgery has been a subject of controversy[9] and practice varies among otolaryngologists.[10] Routine pre-operative OCP cessation is no longer recommended, and an approach based on individual patient risk factor assessment is advised.[1] Specific thromboprophylaxis is not considered necessary for women taking the OCP without additional risk factors, and who are having uncomplicated minor procedures. Our patient had discontinued the OCP six weeks pre-operatively thereby minimizing any associated risk.

This patient had an occult malignancy. Suspicion of malignancy was raised intra-operatively and confirmed by histology. The association between malignancy and thromboembolism was first described in 1872 by Trousseau[11] and the spectrum of thromboembolic disorders occurring in the setting of malignancy is termed Trousseau's syndrome. The incidence of thromboembolism in patients with neoplastic disease ranges from one to 11 per cent, with the reported incidence in NHL being up to 6.6 per cent.[12]

The complex pathogenesis of this hypercoagulable state has been elucidated[13] and includes: cancer cell tissue factor (membrane-bound glycoprotein) activation of factors VII and X, platelet adhesion and activation by tumour cell adhesion receptors, and tumour cell release of procoagulants (e.g. cysteine protease) and antifibrinolytics (e.g. plasminogen-activator inhibitor). In addition, neoplastic cells may directly injure vascular endothelium stimulating thrombogenesis as does vessel obstruction by bulky tumour.

Management of paraneoplastic thromboembolism may be difficult, particularly in the peri-operative period, and requires treatment of the underlying malignancy in conjunction with systemic anticoagulation. To our knowledge there are no previous reports of initiation of systemic anticoagulation therapy in the immediate post-operative period following tonsillectomy. The most common complication of tonsillectomy – tonsillar bed haemorrhage, which may be severe and life-threatening – was a major concern. However, as the risk to the patient of further thrombus formation was considered to outweight the risk of bleeding, systemic anticoagulation with intravenous heparin followed by oral warfarin was given. No bleeding complication occurred.

Resistance to warfarin therapy, although not encountered in this patient, may occur in patients with Trousseau's syndrome and may result in recurrence of thromboembolic disease after discontinuation of heparin. Successful management of such patients with long-term low molecular weight heparin (LMWH) has been reported.[13,14] Further evaluation of the efficacy and safety of LMWH therapy in this setting is required.

In conclusion, this case demonstrates that paraneoplastic hypercoagulable states may exist in young, apparently healthy surgical patients and may be associated with life-threatening peri-operative pulmonary embolism. It reports the successful institution of full systemic anticoagulation therapy in the early post-operative period following tonsillectomy without haemorrhagic complication.

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