Supraventricular tachycardia as a presenting sign of pulmonary embolism in paraplegia. Case report and review

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Pulmonary embolism is a major complication after spinal cord injury and difficult to diagnose in any patient. Supraventricular tachycardia (SVT) is an unusual presentation for pulmonary embolism (PE). This article documents the records of a 60-year-old patient who was undergoing comprehensive rehabilitation after traumatic spinal cord injury and multitrauma. His treatment programme was interrupted by a PE with SVT as the only presenting symptom. This article outlines the clinical approach to the diagnosis of pulmonary embolism. A high index of suspicion of PE should always be kept in mind when SVT occurs in a spinal cord injured patient.

Keywords: paraplegia; spinal cord injury; pulmonary embolism; supraventricular tachycardia

Introduction

Several studies have investigated the incidence of pulmonary embolism (PE) in the spinal cord injured (SCI) population, with most recent studies noting approximately a 5% incidence.¹ Appropriate medical management of PE is often unsatisfactory due to difficulties in diagnosis, which is of paramount importance because in the absence of the appropriate therapy approximately 30% of patients die, a third within 1h.²

This report highlights the need for a high index of PE suspicion when supraventricular tachycardia (SVT) occurs in a paraplegic patient.

Case report

A 60-year-old man sustained multiple injuries resulting from a road traffic accident, and was admitted to the intensive care unit with the following injuries: cerebral contusion with brain oedema, right parietal subdural haematoma, bilateral subarachnoid haematomas, intraventricular haemorrhage of the left and fourth ventricles, thoracic trauma with multiple rib fractures, right-sided haemopneumothorax, right heart and lung contusions, contusions of the stomach and liver. He underwent evacuation of the subdural and subarachnoid haematomas, tracheostomy, sedation and mechanical ventilation. The patient later developed sepsis with multiorgan failure including the acute respiratory distress syndrome, hepatic and renal failure. He received 'kinetic treatment' consisting of supine and prone positioning until his pulmonary function improved. When he regained consciousness flaccid tetraplegia was noticed. Upper limb function improved, and the patient's final neurological status settled as complete paraplegia at the level of T7–8.

A MRI of the spinal cord revealed a compression fracture of T8 vertebra, discopathy and contusion of his

spinal cord between T7 and T12. During rehabilitation liver and kidney function improved, and the tracheostomy was closed. The patient was hospitalized in another department for 3½ months prior to being admitted to the neurological rehabilitation department. On admission there was a large sacral pressure sore; neurological examination showed that higher mental functions were normal, with a degree of retrograde and of anterograde amnesia, normal upper limbs, complete flaccid paralysis with a sensory level right T8 and left T7 with some preservation of light touch sensation.

The pressure sore was treated conservatively, then, 2 months later, he underwent debridement and a rotation flap closure. One week post surgery there were no complications and mobilisation was gradually started. Medication consisted of subcutaneous heparin 5000 U three times a day, oxybutinin 5 mg three times a day, ranitidine 150 mg twice daily, laxatives and lactulose daily.

Ten days after operation whilst reclining in a 60 degree position, the patient complained of weakness, dyspnoea, tremor and diaphoresis, followed by palpitations. He did not have haemoptysis or chest pain, he was afebrile, blood pressure 130/80, and he was tachypneic and cyanotic. There was no clinical evidence of lower extremity deep-vein thrombosis (DVT). The arterial O₂ saturation at room air decreased to 40%. An electrocardiogram (ECG) showed SVT at a rate of 180 per min; no S1Q3T3 pattern nor Twave inversion were noted. After receiving verapamil 5 mg IV, the ECG reverted to a sinus state, the O₂ saturation rose to 90%, and the cyanosis faded. The chest radiograph was normal and PE was suspected. A ventilation perfusion (V/Q) lung scan was performed, and was interpreted as that of an intermediate probability for PE. Thus the patient was anticoagulated with intravenous heparin by a continuous infusion, followed by the oral administration of Coumadin. There were no further episodes of SVT during the remainder of this patients rehabilitation.

Discussion

Pulmonary embolism is a potentially life-threatening condition, which requires immediate diagnosis so that appropriate treatment can be instituted to prevent death. PE is known as a great 'masquerader'³ because of its multivariant clinical presentations and its often non-specific⁴ associated symptoms and signs. Autopsy studies show that PE remains the number one under-diagnosed catastrophic antemortem event.⁵

The high incidence of DVT and pulmonary embolism in the spinal cord injured population is related to stasis,⁶ intimal injury,⁷ and hypercoagulability,⁸ which are all sequelae of acute spinal cord injury (SCI). These effects are referred to as Virchow's triad for the development of thrombosis.⁹ Stasis is the first risk factor to consider following cord injury. Acute paralysis results in loss of the gastrocnemius-soleus pump as well as peripheral venous dilatation. This results in a decreased maximum venous outflow and venous capacitance leading to a reduced venous return from the lower extremities. Several mechanisms have been reported as the aetiology for intimal injury. In SCI patients vasodilation may be the key factor for intimal injury due to blunt trauma sustained with spinal cord injury and the subsequent release of vasoactive amines (histamine, serotonin, bradykinin), and the vasodilatory effect of anaesthesia if surgery was performed for anatomical alignment and stabilisation of the spine.¹⁰ Hypercoagulability in SCI patients is caused by an increase in factors (F) VIII:C, (F) VIII:Ag (von Willebrand factor antigen), (F) VIIIRCoF (Ristocetin cofactor), which are produced by the endothelial cells and their elevation reflects either endothelial cell damage or stasis of blood resulting from the paralysis.

The diagnostic algorithm for PE includes the history, the physical examination, chest radiograph, electrocardiogram, V/Q scan¹¹ and angiography.¹² Intermediate-probability scans or low-probability scans with high clinical suspicion may be an indication for angiography. Diagnosis is also made by excluding other conditions that may mimic PE (DD: pneumonia, pneumothorax, myocardial infarction).

Heavy type or 'classic' symptoms of patients with PE without pre-existing cardiac or pulmonary disease are with descending frequency: dyspnoea (73%), pleuritic pain (66%), cough (37%), leg swelling (28%), haemoptysis (13%), palpitations (10%), wheezing (9%), angina-like pain (4%).¹³ The most common heavy type signs are: tachypnoea (> 20/min) (70%), rales (51%), tachycardia (> 100/min) (30%), deep venous thrombosis (11%), diaphoresis (11%), temperature > 38.5° (7%), Homan's sign (4%), pleural friction rub (3%), cyanosis (1%).¹³ Patients with severe pleuritic chest pain tend to have peripheral PE, which may lead to pulmonary infarction but only rarely results in haemodynamic compromise, whereas patients with massive PE often present with insidious worsening dyspnoea or, less commonly, syncope, presyncope, or cyanosis.

The majority of PE arise from thrombi formed in the deep venous system of the lower extremities.^{14,15} Any patient with DVT proximal to the calf has up to a 50%

chance of developing PE (usually clinically silent). Thrombus confined to the calf veins carries a low embolism risk (15–30%). The typical symptoms and signs of DVT are leg discomfort, swelling, leg tenderness, oedema, warmth, erythema, but none of these features are specific for DVT.

PE is more common in patients with tetraplegia than in those with paraplegia; and with complete rather than incomplete paralysis.¹⁶ Predictive factors for massive PE are: high level of injury, increased body mass index (BMI), and lack of spasticity.¹⁷ These patients therefore fall into the category of high-risk for PE.

Physical activity, such as transfer from bed or straining at stool may trigger the sudden onset of chest tightness, air hunger, and a sense of impending death. ECG changes seen with PE generally consist of QRS complex, ST segment and T wave abnormalities, and rhythm disturbances.¹⁸ The cause is most likely right ventricular strain and myocardial ischaemia.¹⁹ The DD of these findings on ECG is cardiac contusion.²⁰ Small and medium-sized pulmonary emboli seldom show specific ECG changes.

Pulmonary vascular obstruction leads to increased pulmonary vascular resistance and elevated pulmonary arterial and right ventricular pressure. This, in return, causes right ventricular dilatation and hypokinesia, tricuspid regurgitation, and ensuing low cardiac output. In addition to anatomical obstruction, PE causes release of neurohumeral vasoconstrictor factors, such as serotonin and thromboxan A₂.³

These cardiopulmonary abnormalities lead to the clinical manifestation of PE and to its most dire haemodynamic consequence—death due to right ventricular heart failure and cardiac shock.

ECG signs of right heart strain: 'S1-Q3-T3' pattern, right bundle branch block, P pulmonale, or right axis deviation may all be noted with PE.

ST segment changes and T wave inversion are signs of myocardial ischaemia. Among the rhythm disturbances, sinus tachycardia (defined as > 90 b.p.m.) occurs in 30–80% of patients with acute PE.^{21,22} Atrial fibrillation or flutter occur in 5–40%.¹³ SVT is a rare finding and occurs in less than 2%.¹⁸ In an earlier case report²³ SVT was described as a possible presentation of pulmonary embolism. However diagnostic confirmation was not obtained at the time the patient displayed the SVT. The assumption that PE had occurred and was responsible for the rhythm disturbance was based on the subsequent medical course, and a later work-up. In patients with pulmonary embolism general cyanosis is seen in 2–20% of cases, and is usually associated with massive pulmonary embolus. Just-Viera and colleagues²⁴ found that those patients who were in shock had 88% mortality, and those with cyanosis had 64% mortality. Shock and cyanosis at any time are considered grave prognostic signs. Cyanosis is most often seen with acute right-sided heart failure (cor pulmonale). However, it can also be seen with chronic cor pulmonale secondary to multiple pulmonary emboli, as was reported in 10 of 12 such patients by Owen and associates.²⁵

Our patient presented with SVT and general cyanosis. The combination of these two signs in the clinical presentation, together with ruling out other possible diagnoses, and a V/Q scan of intermediate probability, all indicate PE. Furthermore, the treatment of PE resulted in rapid resolution of the symptoms and signs associated with the onset of the SVT.

The PIOPED²⁶ (prospective investigation of pulmonary embolism diagnosis) study showed that most individuals who experienced PE will have a non-high probability V/Q scan. Obtaining a high-probability V/Q scan is almost always associated with the presence of PE. The PIOPED study investigated the correlation between abnormal V/Q scans and angiographically proven PE and developed guidelines on the sensitivity and specificity of lung scans. High-probability scans, had high positive predictive value for PE at angiography. Intermediate-, or even low-probability scans did not exclude PE at angiography, particularly when the clinical suspicion was very high. The majority of patients in PIOPED had non-high probability lung scans. Conversely no patient with low-probability scans and low clinical suspicion had PE at angiography.

Ventilation/perfusion (V/Q) lung scanning seems to be the best screening test for PE. Lung scanning provides a 'road map' for the radiologist in performing pulmonary angiography. Angiography is a highly specific investigation which most accurately confirms a PE.

Our patient fits into the high-risk category and suffered from PE, despite receiving 15000 U subcutaneous heparin per day as a prophylactic measure for the prevention of thromboembolic disease.

Conclusions

The occurrence of SVT in the acute paraplegic patient should alert the clinician to the possibility of a pulmonary embolus. Patients at high risk may develop a very non-specific clinical picture, making diagnosis difficult. The physician should have a high index of suspicion for the occurrence of PE in high-risk patients, whenever there is a sudden change in the clinical status. It should be noted, as with this patient, that the routine administration of an antithrombotic drug may afford some degree of PE prevention but that PE may occur in spite of the drug administration. Hence, the diagnosis of PE should always be suspected and the appropriate investigation must be carried out early.

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