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Massive haemoptysis

Medical management will usually arrest the bleeding

The amount of blood lost during haemoptysis is not easy to measure: it may be swallowed or aspirated, so any haemoptysis should be considered massive when it ceases to be a sign of underlying pulmonary disease and becomes a threat to life in its own right. The definition of massive haemoptysis varies between the expectoration of 200 ml and 1000 ml of blood in 24 hours.[1 2]

The volume of blood is, however, rarely critical, as the cause of death in patients with massive haemoptysis is asphyxiation rather than blood loss. The important factors are the rate at which the bleeding occurs and the patient's underlying lung function.[2 3] Massive haemoptysis is rare, accounting for less than 1·5% of all cases of haemoptysis.[4] In the past, tuberculosis and bronchiectasis were by far the most common causes,[3 5 6] but as their incidence has declined bronchitis and carcinoma of the lung have become more important.[2] Even so, the underlying cause of massive haemoptysis is "benign" in nearly three quarters of cases.[2 5 6]

Although there have been no prospective clinical trials, the accepted treatment for massive haemoptysis is rapid localisation of the site of bleeding and its surgical resection.[7] This view is based on three retrospective studies, which reported a mortality from massive haemoptysis of 78-85% in operable patients who did not receive surgery compared with less than 20% in those who did.[3 8 9] But similar studies have disputed these findings, quoting a mortality of 0-25% in patients who were managed medically.[1 2 5 6 10 11] In these reports many of the patients who died had inoperable conditions or had sudden haemoptysis that caused death too rapidly for surgery to be an option. Even in the operable patients surgery did not confer an advantage over conservative management.[1 2 5 6 11] We believe that in most cases of massive haemoptysis the bleeding can be stopped without thoracotomy, which should be an elective procedure for selected patients after medical management.

Patients with massive haemoptysis should generally be cared for in an intensive care or high dependency unit. Their management has five main objectives: to prevent asphyxiation, to localise the site of bleeding, to arrest the haemorrhage, to determine the cause of the haemorrhage, and to treat the patients definitively (with surgery if required). Patients should undergo chest radiography, and if the site of bleeding can be lateralised should lie head down on the side of the haemorrhage to prevent aspiration of blood into the healthy lung. Cough suppressants and sedatives should not be given; patients should be encouraged to clear their airways with gentle coughing. Blood should be transfused as necessary, and hypoxaemia should be corrected with high concentrations of oxygen through a face mask. Obvious causes of bleeding should be treated with specific measures (for example, reversal of anticoagulation, antituberculous treatment, and so on). In patients with bronchitis or bronchiectasis infection may precipitate haemoptysis, so all patients should be given broad range antibiotics intravenously. A patient with depressed consciousness or one in imminent danger of asphyxiation should be intubated, ventilated, and given adequate suction. A double lumen endotracheal tube may offer advantages over standard intubation provided that the individual lumina are large enough to allow clearance of the airways and the passage of a fibreoptic bronchoscope.[12]

Once the patient's condition is stable the site and cause of bleeding should be sought. The chest radiograph may not help, and bronchoscopy is always required. Traditionally, the rigid bronchoscope is preferred because suction and ventilation are easier with this. As most cases of haemoptysis subside spontaneously within four days[5 10] the procedure can be usually delayed until bleeding is stopped. At that stage fibreoptic bronchoscopy is preferable; a general anaesthetic is not needed, and it allows better visualisation of the bronchi. Even if the patient is still actively bleeding the fibreoptic bronchoscope may be passed through an endotracheal tube, allowing ventilation and suction to continue.[13] Local measures to control bleeding can also be tried, such as occlusion of the affected bronchus with a Fogarty catheter balloon.[14] Another possibility is to infuse thrombin and fibrinogen into the bronchi,[15] but no controlled trials have shown this to be more effective than lavage with iced saline.[6] Patients with persistent life threatening haemoptysis should be treated with bronchial artery embolisation, which should stop the bleeding (in the short term) in almost all patients.[16 17] Emergency surgery should be reserved for those patients with adequate lung function in whom the site of haemorrhage has been identified who continue to suffer massive haemoptysis despite the measures described.

When the bleeding has stopped the problem is to prevent recurrence. Individual patients will be given specific treatment (medical or surgical) according to their diagnoses, but should "prophylactic" resection of the bleeding site be carried out in patients with bronchitis or bronchiectasis or in whom no cause is found? We have little information in published work to answer this question. Three studies have found no recurrence of massive haemoptysis in medically treated

patients, but the numbers were small and the length of follow up only between one and five years.[5 18 19] As in many aspects of the management of massive haemoptysis no hard and fast rules have been proved to apply, and decisions will often depend on the preferences of the physicians or surgeons.

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Idiopathic dilated cardiomyopathy

Rational treatment awaits better understanding of pathogenesis

Idiopathic dilated cardiomyopathy is a chronic heart muscle disease that predominantly affects young men and causes dilatation and contractile dysfunction of the left or the right ventricle, or both.[1] In the United States its prevalence is around 20 per 100 000 population, and there is an incidence of six new cases per 100 000 a year[2]; exact figures for Britain are not available. The diagnosis is made by exclusion; it relies on showing the absence of coronary artery disease, valvular or pericardial disorders, and specific heart diseases.

The usual presentation is with features of heart failure, arrhythmia, or thromboembolism, and the duration of the presymptomatic phase of the illness is uncertain in most patients. For many years it has been thought that dilated cardiomyopathy may be a consequence of previous viral myocarditis. A syndrome very like human dilated cardiomyopathy may be produced in animals after they have had viral myocarditis, but the progression in humans of acute myocarditis to left ventricular dilatation and contractile failure has been described only in isolated case reports.[3]

Although the early natural course of the condition before the development of symptoms remains a matter of speculation, the clinical course after diagnosis is well documented. Published reports agree on the poor prognosis of dilated cardiomyopathy, with two fifths to a half of patients dying within two years after the diagnosis.[1 4] Some patients die in cardiogenic shock or pulmonary oedema, but most deaths seem to be sudden. In the early period after diagnosis sudden death usually occurs in association with progressive impairment of left ventricular function, but it is common at all stages of the disease and may occur in patients who have responded well to treatment with regard to their symptoms and the haemodynamic features of the disease and who have remained stable for some time.

The high incidence of sudden death suggests an arrhythmic cause, and indeed arrhythmias are common regardless of the duration of the disease (one fifth of patients develop chronic atrial fibrillation and two fifths have non-sustained ventricular tachycardia on 24 hour electrocardiographic monitoring).[5] The prognostic implications of ventricular arrhythmias remain controversial, however, and may be different at different stages of the disease. Non-sustained ventricular tachycardia is common in the first two years after diagnosis—the period during which most deaths occur—but these are generally in association with progressive heart failure (though details of the mode of death are frequently lacking in published work). Thereafter there is an annual mortality of about 4%, with most deaths apparently being sudden. The influence of ventricular arrhythmia on survival may be more important in later stages in patients whose heart failure is well controlled but who die suddenly.

Conventional treatment makes very little difference to survival and has no impact on the progression of the disease. Indeed our imperfect understanding of the disease processes that underlie dilated cardiomyopathy means that treatment remains empirical. In practical terms it is restricted to the relief of symptoms of heart failure with diuretics, angiotensin converting enzyme inhibitors, and (perhaps) digoxin and the prevention of thromboembolic complications with coumarin anticoagulants.[1] The only treatment option that has been shown to improve prognosis is heart transplantation, and patients with dilated cardiomyopathy account for more than half of all recipients of heart transplants.[6]

Treatment with antiarrhythmic agents has not been shown to improve prognosis in patients with dilated cardiomyopathy except in those patients who present with episodes of sustained ventricular tachycardia and those in whom the disease predominantly affects the right ventricle.[7] Although this group of patients is small, sustained monomorphic ventricular tachycardia of right ventricular origin is a prominent feature of their disease. Aggressive management of the arrhythmia, including drug treatment and surgical approaches, is warranted; if life threatening arrhythmias can be suppressed the prognosis is good, as left ventricular dysfunction is usually minimal.

The report of the cooperative north Scandinavian enalapril survival study (CONSENSUS) study of the effect of the angiotensin converting enzyme inhibitor enalapril in the treatment of older patients (mean age 70 years) with severe