Intrapleural streptokinase: the answer to community acquired pleural infection?

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Approximately 40% of patients with community acquired pneumonia develop a parapneumonic effusion, despite the availability of appropriate antibiotics. This effusion may become secondarily infected, denoted by changes in fluid chemistry (fall in pH and glucose, rise in LDH), organisms on gram staining or culture, and extensive fibrin deposition. Untreated it can result in an empyema with frank pus and fibrous organisation in the pleural space. Effective treatment requires adequate and easy drainage of the fluid. Untreated infections need surgery compared with one quarter of the control group. No clinically significant systemic fibrinolytic complications using smaller individual doses (250 000 IU) and smaller doses at more purified forms of streptokinase has resulted in less anaphylaxis, with significantly reduced haemorrhagic complications such as anaphylaxis, intrapleural haemorrhage, and systemic fibrinolysis have limited its use in the past. However, recent experience with intrapleural streptokinase in community acquired pleural infection was started at the time of diagnosis, rather than at tube instillation times (2–4 hours). The efficacy of streptokinase in promoting drainage of loculated effusions has been suggested by several recent studies, but these involved small patient numbers and were uncontrolled.

In this issue of Thorax Davies et al. report the first prospective randomised trial of early intrapleural streptokinase versus control saline flushes through intercostal drains in patients with an infected parapneumonic effusion or empyema. The streptokinase treated group drained significantly more fluid than the control group, showed significantly greater radiographic improvement, and none needed surgery compared with one quarter of the control group. No clinically significant systemic fibrinolytic effect or haemorrhagic problems were encountered.

An important aspect of this study is that streptokinase was started at the time of diagnosis, rather than at tube drainage failure due to loculation and fibrinous plugging. Patients who fail to respond to treatment with streptokinase already have considerable pleural thickening at surgery. Thus, if streptokinase is to shorten treatment times and prevent surgery it must be initiated before the fibrinopurulent stage is advanced and organisation occurs.

Placement of the catheter under ultrasound guidance allows better positioning, particularly in multiloculated effusions where it can be placed in the largest loculation. The catheters used by radiologists are frequently much smaller than chest drains traditionally used at the bedside, with improved patient comfort and a more controlled rate of fluid drainage. However, even with the use of radiologically guided placement, suction and saline flushes, the addition of intrapleural streptokinase significantly enhances drainage and reduces the need for surgery.

The findings of Davies et al. allow us to re-evaluate the management of patients with parapneumonic effusions. All patients with suspected pneumonia should be examined clinically and radiologically for the presence of an effusion. Prompt diagnostic thoracocentesis should be performed where fluid is detected. If frankly purulent fluid is obtained, there is direct or indirect laboratory evidence of an infected effusion, or there is localisation on imaging, then a drain should be inserted, preferably under ultrasound guidance. Streptokinase should be administered daily (250 000 IU), even if the effusion is not multiloculated, and continued until the volume drained is <100 ml/24 hours. Patients should be discussed with a thoracic surgeon and those who fail to respond to this treatment should then be considered for surgical intervention.

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