Case Presentation :

Oesophageal Perforation

Prof. A.K. Sharma FRCS, FACS, FICS

Dr. G. Sharma, M.D., PH.D.

Dr. J.K. Shrestha M.B.B.S.

Perforation of the intrathoracic oesophagus is a life-threatening condition and requires prompt diagnosis and immediate surgical management. When a perforation of the intrathoracic oesophagus is untreated, gastric juices and bacterialaden oropharyngeal secretions leak into the chest cavity. Mediastinitis is chemical but within hours of the rupture, bacterial invasion occurs. If the rupture extends into the pleural space, the mediastinitis may be less severe but the pleural cavity becomes extensively infected. In either case, dehydration, bacteremia, and circulatory collapse usually develop within hours.

Factors affecting the outcome of oesophageal ruptures include the age and general condition of the patient, the location, size and cause of the treatment, and the pre-existing oesophageal disease. According to Postlethwait (1979) after 40 years of age, mortality from spontaneous rupture of the oesophagus increases progrssively with age. Blichert-Toft (1971), reporting results between 1944 and 1969, found that patients who had operations within 12 hours had a mortality of 22 percent and within next 12 hours, 36 percent. However, if treatment was delayed beyond 24 hours, the mortality rose to 64 percent. Primary repair produced a 34 percent mortality while nonoperative treatment resulted in an 80 percent rate of mortality.

Although it is agreed in general that management of the recently ruptured thoracic oesophagus is direct suture with drainage of the pleural space, there is disagreement as to the best method of managing late intrathoracic oesophageal perforations. Most authors concur

that tube drainage of the mediastinum and pleural cavity, administration of antibiotics and adequate nutrition are important components of oesophageal perforations with delayed recognition.

Material and Method

A 35-year old man was admitted in the dept. of surgery of Bir Hospital with complaints of severe pain in the right hemithorax and epigastrium, high fever and breathlessness since two days. On admission patient stated that he had accidentally swallowed a big piece of meat with bone about 36 hour prior to admission. 5-6 hour later he developed the above-mentioned complaints. On physical examination general condition of the patient was low. He was severely dehydrated and had cervical crepitus. His pulse rate was 132 per minute and blood pressure was 80/60 mm. of mercury. On auscultation of the chest the breath sounds were diminished on the right side. There was positive rebound tenderness all over the anterior abdominal wall. A postero-anterior view of chest X-ray revealed massive pneumo-hydrothorax in the right and a little shift of the mediastinum on the left side. A diagnosis of thoracic oesophageal perforation was made on the basis of above mentioned finding. After adequate rehydration and clinical examination of the blood, which revealed total leucocyte count to be 10,000 per cubic mm. neutrophils 86% and haemoglobin 13.7 gm. percent, blood grouping and cross matching was done and patient was taken to operation room. A right antero-lateral thoracotomy was performed through the 4th intercostal space. On exploration the pleural cavity contained about 500 ml. of purulent fluid with few food particles. The fluid was sent for culture and sensitivity and then aspirated with a suction tube. On examination of the oesophagus a 8 cm. perforation was visualized just below the azygos vein. A big piece of meat with bone was seen stuck in the perforated wall of the oesophagus. Part of it was located inside the oesophageal lumen; the foreign body was removed. The defect in the muscle layer was surgically extended longitudinally at each end of the perforation in order to visualize the mucosa. The muscle around the perforation was debrided and closed with interrupted 4-0 chromic catgut sutures; the muscle was closed with interrupted 3-0 chromic catgut sutures. The pleural space was cleansed and drained with two chest tubes, one in the second intercostal space anteriorly and another in the 8th intercostal space posteriorly. The chest cavity was closed in layers using chromic catgut for muscles and fascia and thread for skin. After this a gastrostomy was performed for temporary feeding of the patient. In the immediate postoperative period patient had slightly raised temperature for two days. The lung had expanded and there was no air and fluid in the pleural cavity, so the

upper drain was removed on the third and lower drain was removed on the sixth postoperative day respectively. On the 7th postoperative day patient again developed temperature; X-ray chest was done and fluid was visualized in the right pleural cavity again. Gastrografin follow-through of the oesophagus did not reveal any leakage from the suture line. As the quantity of the fluid remained same in spite of repeated aspiration, it was decided to put an intercostal drainage tube again for the draining of the exudation. After the drainage of the pus, temperature came down and the lung was totally expanded again, there was no discharge through the tube, so it was removed on the 13th postoperative day. All stitches were removed on the 12th P.O. day. On the 15th P.O. day patient again developed high temperature upto 102 F, but X-ray chest did not reveal any collection in the pleural cavity. The rise in temperature continued further but X-ray chest was negative; patient was investigated for Malaria, Kalazar, UTI, but nothing could be revealed. All reports on culture of the pleural fluid were also negative. Patient was treated with ampicillin and gentamycin throughout the whole postoperative period. Malecot's tube was removed on the 21st day from the stomach and patient was allowed semisolid diet orally. On the 26th P.O. day again the X-ray chest suggested collection of fluid in the pleural cavity, so again the cavity was drained; protein and blood were transfused. Gradually the temperature came down but did not become normal. The cavity was washed with antiseptic solutions daily. This continued for about one month. Ultimately the fever came down to normal. Gradually patient's general condition became fair and on the 66th day of operation he was discharged home. His immediate follow-up after discharge did not reveal any other complications. Soon he started to fulfil his full time duty of a tailor. In two years follow-up after operation, X-ray chest shows complete expansion of the lungs with normal pleural thickness and barium follow through a normal oesophagus.

Discussion

Despite awareness of the seriousness of intrathoracic oesophageal ruptures, delay in diagnosis is the major stumbling block to lowering morbidity and mortality. Early in the course of an oesophageal perforation the diagnosis may only be suspected from a characteristic history. However, once mediastinitis becomes established, chest or abdominal pain, dyspnea, fever, hydropneumothorax, or mediastinal emphysema are highly suggestive of a oesophageal rupture. Patients who are seen late after oesophageal rupture are dyspnoic, dehydrated, and very often hypotensive. Following fluid resuscitation and antibiotics the patient's condition improves and there is reluctance among the surgeons to repair the oesophageal rupture by operation. However

if an operation is not done, soilage of the mediastinum and pleural cavity progresses to severe empyema and the patient dies of the complications of uncontrolled sepsis. In patients with delayed recognition of nonmalignant intrathoracic oesophageal perforation, elimination of continued chemical and bacterial contamination can be achieved by primary closure of the oesophageal perforation. Although the oesophageal muscle is inflamed and necrotic, the mucosa usually remains viable and can hold sutures securely. The mucosal tear is often longer than the muscle tear so it is important to mobilize the oesophagus and to extend the muscle tear at each end of the perforation in order to define, debride, and close the mucosa accurately with interrupted sutures. The inner aspect of the oesophagus should be carefully inspected before closure of the rupture in order to rule out the presence of cancer, stricture or another tear. The muscle layer should be debrided and closed with interrupted sutures. Potential intrapleural dead space is eliminated by adequate pleural drainage, pulmonary decortication, if necessary the elective use of mechanical ventilation in order to maintain full expansion of the lung. Finally, the use of the total parenteral nutrition for maintenance of the protein and caloric needs of these catabolic patients and broadspectrum antibiotic is mandatory for increased survival.

Announcement From Resolution Sub-Committee

Any member wishing to bring any resolution in the
General Body meeting of N. M. A. during 12th
All Nepal Medical Conference, may kindly
send it to Chairman, Resolution
Sub-committee by the 1st week
of Falgun 2041.

Dr. N. G. Amatya
CHAIRMAN,
Resolution Sub-Committee.