EXPERIMENTAL USE OF A FREE PERICARDIAL PATCH IN THE REPAIR OF A DEFECT IN THE ESOPHAGUS OF DOGS

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SUMMARY

This paper discusses the feasibility of using a free pericardial patch in repairing defects of the esophagus. The experimental model used is the dog. A piece of the side wall of the esophagus is first excised. This defect in the esophagus is then covered with a free patch of pericardium. The animals are then sacrificed at sequential dates and the grafted site submitted for microscopic examination. The results show that a free graft of pericardium when used as a patch can prevent leakage of esophageal contents and allow healing of the defect without gross narrowing of the lumen.

INTRODUCTION

Esophageal surgery has, until recent years been fraught with a prohibitive morbidity and mortality. This exposure was well expressed in a paper written during the beginning of the century by James H. Saint,[1] who wrote "The latter (i.e. esophageal surgery) have so far been attended by a depressing mortality, so that some of the most stout-hearted surgeons often prefer to leave that anatomic relations of the viscus undisturbed rather than bring about almost certain death by surgical intervention"

However, the more optimistic views of Abel[2] in his Hunterian Lecture in 1926 are now being realized. With the advent of intravenous hyperalimentation, development of powerful antibiotics and anaesthetic advances, esophageal surgery, as shown by McKeown[3] is now a well established exercise with an acceptable morbidity and mortality.

Nevertheless, the problem of anastomotic leakage remains. Amongst other reasons, this has been blamed on the absence of a serosa and omentum to protect and reinforce the anastomosis. In an attempt to overcome this problem, Kleinsasser[4] evaluated the effectiveness of free peritoneal grafts in reinforcing esophageal anastomoses in dogs and concluded that the grafts were of no benefit. Goldsmith[5] showed that in dogs the omentum on a pedicle may help in seasing-off defects produced in the esophagus. Others, including McSwain[6] have used parietal pleural grafts and concluded that it may be effective in preventing leakage in an esophago-esophageal suture line.

This paper looks into the feasibility of using a free pericardial graft in the repair of defects of the esophagus.

MATERIALS AND METHODS

Twenty-five mongrel dogs were used in this investigation. They were anaesthetised with

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thiopentone, an endotracheal tube was inserted and respirations were then maintained with a fixed volume respirator pump. Under aseptic conditions, the left hemithorax was then entered through the bed of the sixth rib. The esophagus was dissected out and a strip of the side wall excised. The phrenic nerve was then dissected off the pericardium and a piece of the pericardium excised. This was later used to close the defect in the esophagus. In the first twenty dogs the size of the esophageal defect was 4.0 x 2.0 cm and the size of the pericardial graft was 5.0 x 3.0 cm. It was then found at post-mortem on the twentieth dog which died that the heart had herniated through the defect in the pericardium. As such, in all subsequent dogs, the size of the esophageal defect was reduced to 2.0 x 1.5 cm so that the pieces of pericardium removed could be reduced to 3.0 x 2.0 cm.

At the end of the procedure, the lung was inflated and the chest cavity then closed. No chest drainage tubes were used. Through a small abdominal incision, a Stamm-type feeding gastrostomy was then made.

Post-operatively the dogs were kept on a nil-by-mouth regime for five days. Nutrition during this period was maintained through the gastrostomy. Oral feeding on a standard diet of rice and dog-food pellets was then commenced on the sixth day when the gastrostomy tube was removed.

The dogs were sacrificed at sequential dates and a post-mortem performed. If the dog died before the planned date for sacrifice the post-mortem was done as well. At post-mortem the pleural cavity was inspected for evidence of infection. The esophagus together with the aorta and surrounding lung were excised en-bloc. The esophagus was then opened longitudinally along the side opposite the site of the defect. The grafted site was carefully inspected for any perforations. The circumference of the esophagus at the grafted site, 2 cm above and below it, was measured. The grafted area was then subjected to histological examination. The following results were obtained.

RESULTS

Of the total number of twenty-five dogs, sixteen dogs were alive at the time of planned sacrifice. Of the nine dogs which died, three died as a direct result of leakage of the graft. Post-mortem examination in these dogs revealed leakage at the grafted site associated with a large empyema of the thorax. Of the remaining six dogs which died, one died from a herniation of the heart through the defect in the pericardium. The remaining five dogs died from problems related to anaesthesia and bleeding. Post-mortem on these dogs showed the graft to be intact with no evidence of empyema in the chest.

The circumference of the esophagus was used as an indication of narrowing of the esophagus. Measurements were taken at the site of the original defect, 2 cm from the uppermost and 2 cm from the lowermost edge of the defect. In the first twenty dogs in which the size of the original defect was 4.0 x 2.0 cm, the average circumference of the esophagus was 4.6 x 4.3 x 4.3 cm. In the subsequent five dogs, where the size of the original defect was 2.0 x 1.5 cm, the average circumference of the esophagus was 4.3 x 4.0 x 4.0 cm. The last dog was sacrificed sixty-five days post-operatively and the circumference of the esophagus was 4.5 x 4.5 x 4.5 cm. Thus, irrespective of the size of the original defect created, there was no appreciable narrowing of the esophagus.

Histological Appearance

All the esophagi were then submitted for histological examination. At forty-eight hours (Fig. 1), the graft showed evidence of necrosis although macroscopically, there was no leakage of esophageal contents seen. At four days (Fig. 2), there was heavy inflammatory cellular infiltration and granulation tissue formation can be seen. At the same time the squamous epithelium from the surrounding normal esophagus can be seen to be growing over the granulation tissue. The surrounding lung with the visceral pleura (Fig. 3) can be seen to be closely adherent to the graft and was also involved in the inflammatory reaction. By one week, the grafted site had almost been covered over by the new growth of squamous epithelium. The surrounding inflammatory infiltration was now being replaced by fibrous tissue. At two weeks (Fig. 4) epithelization of the graft was complete. Fibrosis

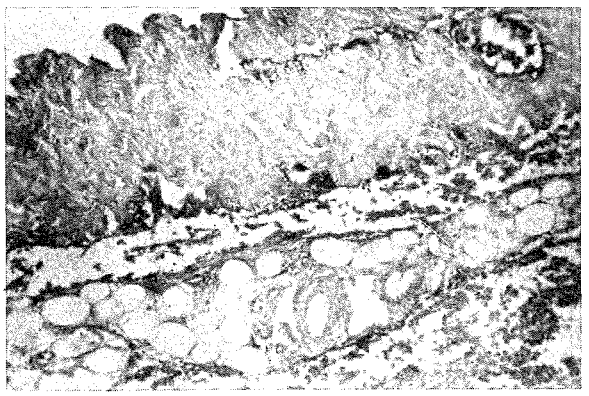

Fig. 1 48 hours postoperative. The graft shows evidence of necrosis.

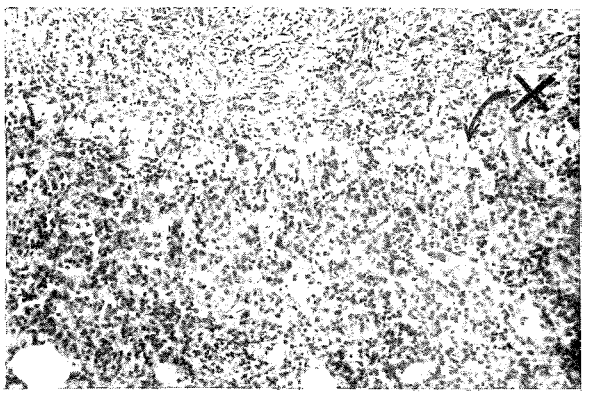

Fig. 3 One week following surgery. The lung tissue is invaded by inflammatory infiltrate and is seen in the bottom half of the micrograph. The visceral pleura is marked X and the graft lies above it.

Fig. 2 Four days following surgery. There is heavy inflammatory cellular infiltration of the graft. Squamous epithelium can be seen to be growing over the granulation tissue.

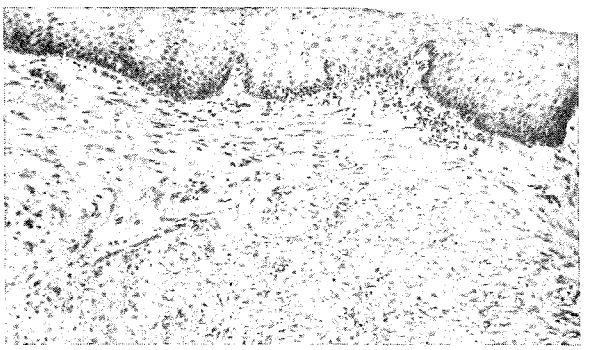

Fig. 4 Two weeks following surgery, marked fibrosis of graft with epithelization completed.

in the graft was more marked with the lung firmly adherent to its outer surface.

DISCUSSION

In this investigation, the experimental model used has been the purposeful creation of exaggerated defects in the esophagus in dogs. It has been previously demonstrated by Middleton[7] that such defects resulted in the death of the animal if they were left uncovered. The effectiveness of a free pericardial graft in preventing leakage of esophageal content in the pleural cavity is then evaluated.

Various autogenous grafts have been used experimentally in the past. Middleton[7] used a visceral pleural pattch for repair of large suture for defects in dogs. Micklos Roth[8] demonstrated that a lobe of the lung can also be used effectively to cover a defect in the side wall of the esophagus in dogs. In both these investigations, the graft was of a pedicled form and maintained their vascular connection with the donor site. Deaton[9] however, successfully used a free lung-pleura graft as a buttress for reinforcement of esophago-esophageal anastomoses in dogs. Adler[10] designed the use of a free pericardial graft which he then used to reinforce an anastomotic suture line using a thrombin fibrinogen coagulant.

In this investigation, we have used a free graft of pericardium which is then sutured to the edges of a defect in the esophagus. Histological analysis of the specimen has demonstrated an early necrosis of the graft within 48 hours and in fact, in the later stages, it may be the visceral pleura of the lung which begins to take on a more active part in reinforcing the patching procedure. Thus, although there is a lack of omentum in the pleural cavity, the visceral pleura may to some extent take over its function. As such, a free patch of pericardium sutured to a defect in the esophagus may serve as a mechanical and bacteriological barrier in the first 48 hours to prevent gross contamination of the pleural cavity thereby allowing the surrounding lung to then complete the sealing-off process while the graft then slowly deteriorates and becomes replaced by fibrosis. Although one would expect some narrowing of the esophagus as a result of the fibrosis, we have not been able to demonstrate this in our investigation.

CONCLUSION

We have demonstrated in this experiment that although a free patch of pericardium does not become revascularized when used to cover a defect in the esophagus, it acts as an effective mechanical and bacteriological barrier to the leakage of esophageal contents. The visceral pleura also plays an equally important part in sealing-off the esophageal defect thus preventing contamination of the pleural cavity.

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