# Ischaemic necrosis of subcutaneous colonic neoesophagus: an unusual complication of presternal hypertrophic scar

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## ABSTRACT

Hypertrophic scars and keloids not only pose aesthetic problems but also cause functional and anatomical dysfunction by leading to contractures and sometimes by compression of underneath structures. A 76-year-old man presented with progressive dysphagia of two months duration. Examination showed the unusual complication of a presternal hypertrophic scar, causing ischaemic necrosis of a subcutaneously-transposed colon, used for oesophageal reconstruction in a patient with inoperable carcinoma of the oesophagus. Such a complication of hypertrophic scar has never been reported in the literature.

Keywords: hypertrophic scar, keloid, oesophageal carcinoma, oesophagus, oesophagoplasty

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### INTRODUCTION

Hypertrophic scars and keloids are unique human dermal fibroproliferative disorders that occur following trauma, inflammation, surgery, burns and possibly, spontaneously. These lesions indicate excessive wound healing and occur as a result of excessive scarring caused by pathologically overabundant collagen deposition and irregular functioning of fibroblasts. These lesions cause concerns to the patient because of their unsightly appearance and also the functional problems by leading to contractures<sup>(1-3)</sup>.

#### **CASE REPORT**

A 76-year-old man, known hypertensive with ischaemic heart disease, presented with progressive dysphagia of two months duration. Examination did not reveal any abnormality except for a presternal hypertrophic scar (Fig. 1), which had developed following a thermal burn 33 years ago. Endoscopical biopsy revealed a poorly-differentiated, diffusely-infiltrating adenocarcinoma in the middle one-third of the oesophagus. Computed tomography (CT) of

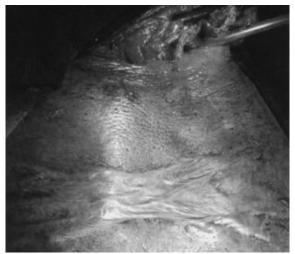


Fig. I Photograph of anterior chest shows a presternal hypertrophic scar.

the thorax and upper abdomen showed involvement of mediastinal structures by the growth, with enlarged mediastinal and coeliac group of lymph nodes. Cardiac evaluation showed stress test positive for exercise-induced ischaemia, putting the patient under ASA class IV risk.

Based on the poor prognosis of the disease and high risk of operative morbidity and mortality, feeding jejunostomy was planned under local anaesthesia. However, the patient did not agree for jejunostomy and expressed his desire to eat and swallow for rest of his life. Restoration of swallowing was a challenge in this high-risk patient, and the simplest option was oesophageal reconstruction by colonic transposition by the subcutaneous route, without oesophageal resection, thereby avoiding thoracotomy and intrathoracic anastomosis. The patient underwent transposition of well-vascularised segment of transverse colon to the subcutaneous route in front of the sternum and anastomoses to the cervical oesophagus (oesophago-colostomy) in the neck and to the jejunum (colo-jejunostomy) in the abdomen. The postoperative period was uneventful. The patient tolerated the oral feeds, which was started on the eighth postoperative day, well. He was discharged on the 13<sup>th</sup> postoperative day, after suture removal.

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Fig. 2 Intraoperative photograph shows viable colon, the upper end (arrow) of which corresponds exactly to the level of presternal hypertrophic scar.

Two weeks after the discharge, the patient presented with high-grade fever and tachycardia. There was foul-smelling discharge from the cervical wound with salivary leak. Based on the clinical diagnosis of cervical anastomotic leak and sepsis, an emergency exploration was done. Intraoperative findings were surprising. The part of the colonic conduit distal to the hypertrophic scar was completely necrosed and sloughed off, and the part proximal to it was viable (Fig. 2). The transposed colon was removed, the cervical oesophagus was closed and the jejunal opening was used to insert the feeding tube. The patient tolerated the jejunostomy feeds well, started on the third post-operative day, but could not mentally accept it. On follow-up two months after surgery, the patient was on full jejunostomy feeds.

# DISCUSSION

Restoration of swallowing in a patient with dysphagia due to malignant disease in the upper gastrointestinal tract, remains a surgical challenge. Techniques for bypassing the oesophagus were first described by Roux<sup>(4)</sup>, who used the jejunum, and Kelling<sup>(5)</sup> and Vulliet<sup>(6)</sup>, who suggested the use of the colon. Since then, the controversy has persisted as to the best mode of reconstruction, and the colon remains a durable option<sup>(7)</sup>. The major advantages of using the colon for oesophageal replacement are: a greater length of the viscus is available, mobilisation is easy and acid regurgitation with anastomotic ulceration rarely occurs<sup>(7)</sup>.

Based on a comprehensive evaluation of the disease type, patient age, heart and lung functions, nutritional status, and accompanying diseases, three colon transposition routes are available (anterosternal subcutaneous tunnel, retrosternal tunnel, and oesophageal bed passage), with each route having its advantages and disadvantages<sup>(8)</sup>. The anterosternal subcutaneous route is preferred in cases where a long graft for oesophageal replacement is required and intrathoracic anastomosis is not desirable, as in the present case. The advantages of this route are: thoracotomy is avoided, oesophageal anastomosis is easily performed, anastomotic failure in the subcutaneous space is not a serious problem, revision of the anastomosis is easily performed under local anaesthesia, and the mortality rate due to oesophageal anastomotic failure is diminished<sup>(9)</sup>.

The most serious complication of oesophageal reconstruction by colonic transposition is colonic segment necrosis and anastomotic leak<sup>(8)</sup>. This usually occurs in the first postoperative week and is preventable by giving attention to technical details like the colour of the colonic segment and bleeding of its cut ends before performing the anastomoses. This patient underwent meticulous anastomoses, with particular attention to the vascularity of the mobilised colon. The surgical team was anticipating good surgical outcome, ignorant of the consequences of the presternal hypertrophic scar on the transposed colon just underneath it. We suggest the possibility of compression of colonic vascular pedicle between the excessive scar tissue anteriorly and the sternum posteriorly, resulting in necrosis of the colonic conduit just distal to the scar tissue.

The surgical team never anticipated such a complication of burn scar contracture. This complication, in our opinion, could have been avoided by giving attention to the presternal hypertrophic scar before resorting to subcutaneous transposition of colon. The purpose of reporting this case is to avoid such complications in the future by giving significance to even "insignificant" clinical findings before planning the management.

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