Histol Histopath (1986) 1: 89-92

Histology and Histopathology

Peritoneal fine structure of inguinal hernia: a scanning electron microscope study

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Summary. Mesothelial cells of the normal human peritoneum of the anterior abdominal wall are covered with numerous surface microvilli. These cells become partially denuded inside the sacs of direct and indirect inguinal hernias and so lose the protective property the microvillar covering may impart on them. These mesothelial cells of hernial sacs also acquire an extensive surface coat of fibrin-like material, presumably due to the loss of that protective property, which may as a result subject them to adhesions. There is a considerable collagen build-up in the subserosal fibrous tissue of sacs of both direct and indirect inguinal hernias. Such a build-up is at variance with the accepted current surgical concept which suggests a defect in collagen synthesis, rather than a build-up, as the cause of direct hernia.

Key words: SEM - Hernia - Peritoneum - lnguen

Introduction

Changes in the fine structure of the peritoneum of inguinal hernial sacs have been studied by the use of the scanning electron microscope (reported here) and the transmission electron microscope (to be published). The aim of the two studies, which supplement each other, was to elucidate how cells in general, and peritonea] mesothelium in particular, cope with mechanical trauma. Also, helpful data pertaining to adhesion formation are presented.

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In order to provide coniparative data, normal human peritoneum of the anterior abdominal wall has also been studied.

Materials and methods

Surgically excised hernial sacs from **10** cases of direct and indirect inguinal hernia were processed for scanning electron microscopy. Patients were of different ages and of both sexes. Small pieces of normal peritoneum from the anterior abdominal wall were also obtained and similarly processed to serve as control data.

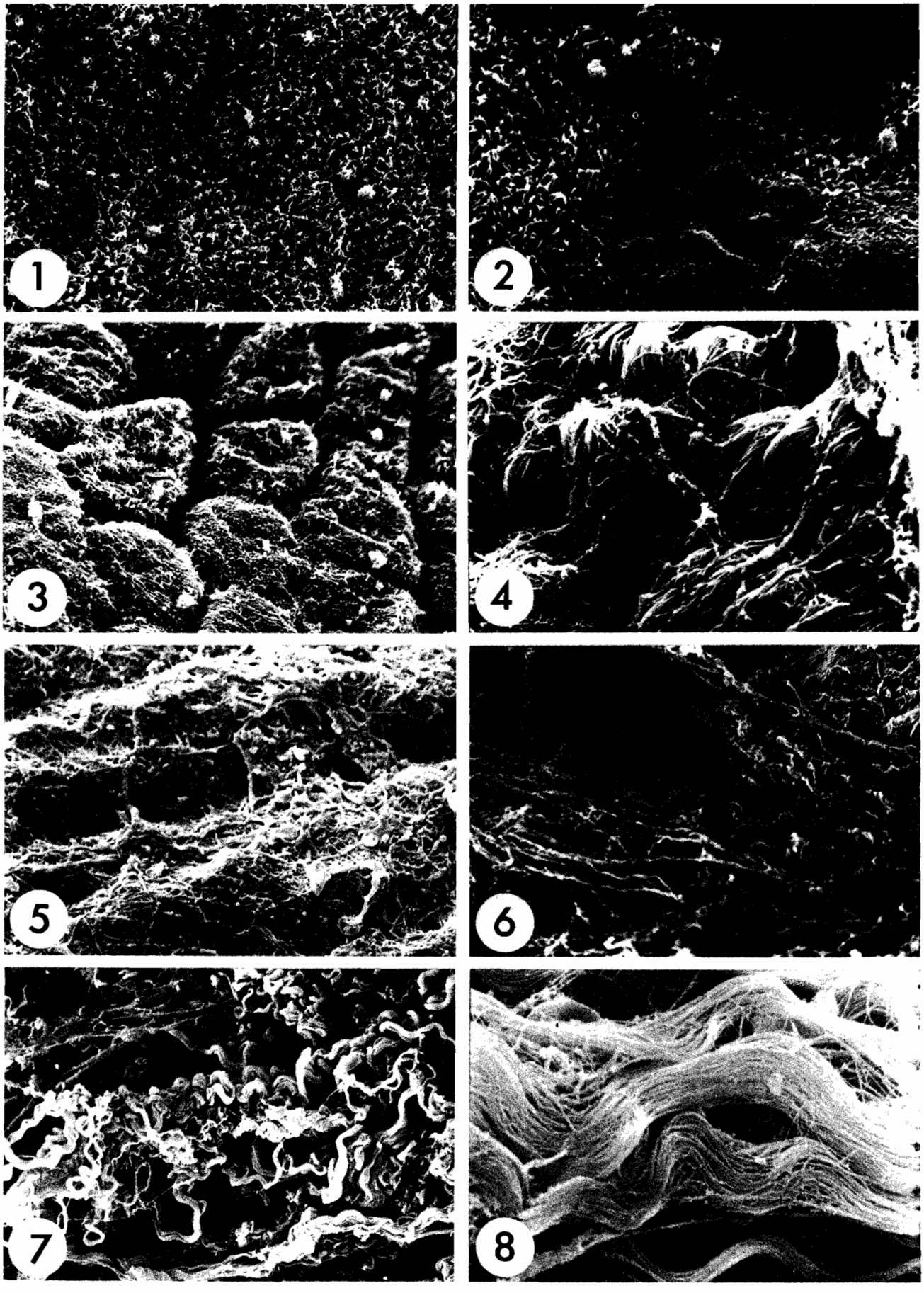
Tissues were briefly rinsed in normal saline solution immediately following surgical removal. Prolonged and/or vigorous rinsing which may have resulted in disruption to cell morphology was avoided.

Tissues were fixed in **2.5%** glutaraldehyde in **0.1** M sodium cacodylate buffer at pH 7.4 and 4° C for one hour and dehydrated in ascending grades of acetone. After critical point drying and gold coating, specimens were viewed in a Siemens Autoscan at **20KV** and at various magnifications.

Results

Results obtained from direct and indirect hernias were essentially similar, thus no attempt is made here to report them separately.

It is evident that the free surface of the normal peritoneum of the anterior abdominal wall is covered with, more or less, equally distributed microvilli (Fig. 1). Cell outlines are barely discernable, presumably due to protein precipitation within the peritonea1 serous fluid which adhered to large areas of the cell surface during fixation (Leak and Rahil, 1978).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Highpower investigation of the apical plasma membrane of the mesothelial cells at the base of the microvilli did not reveal the presence of stomata as reported in normal peritoneum of laboratory animals (Baradi and Rao, 1976; Leak and Rahil, 1979).

Peritoneum of hernial sacs showed scattered areas denuded of microvilli where mesothelial cells appeared bare (Fig. 2). Occasionally, mesothelial cells rounded up and retracted, creating abnormally wide deep cleavage in the intercellular spaces resulting in cell outlines becoming apparent (Fig. 3). This obviously had led to unobstructed exchange of material between the subserosal fibrous tissue and the cavity of the hernial sacs. It seems that these, more or less, spherical mesothelial cells of the hernial sacs carried somewhat fewer apical microvilli. Frequently these spherical mesothelial cells started acquiring a coat of fibrin-like material on their free surface. The density of this material was not extensive enough to obscure the identity of the mesothelial cells involved (Fig. 4). However, in many sacs, the fibrin-like material became so dense as to form a complete sheet over the mesothelial cells masking their outlines entirely (Fig. 5).

In normal peritoneum, mesothelial cells overlaid a subserosal bed of delicate loose fibrous tissue where thick bundles of collagen were not encountered (Fig. 6). In hernial sacs (direct and indirect) collagen build-up into thick bundles was extensive and present in all sacs examined (Figs. 7, 8).

Discussion

Andrews and Porter (1973) and Schwarz (1974) demonstrated, morphologically and histochemically, the presence of a glycocalyx stainable with ruthenium red on top and on the sides of mesothelial microvilli. These authors suggested that the peritoneal serous exudate becomes entrapped in small shock-absorbing cushions created by the microvilli being held together by the strong water-binding capabilities of the negatively charged acid glycosaminoglycans of the microvillar glycocalyx. They postulated that this arrangement protects the mesothelial cells from surface friction and ensures their integrity against adhesion formation. From a surgical viewpoint, traumatizing the microvillar protective covering alone without undue interference with the entire mesothelial cell would be sufficient to result in adhesions. Removal of the protective cushions, created by the microvillar glycocalyx, would subject the underlying cells to surface damage that may result in an inflammatory response leading to tissue adhesion.

It is well known that even the slightest of a wide variety of traumas to serosal membranes may result in adhesion (Bengt, 1956) and that trauma and bacterial infection predispose to adhesion formation (Bridges and Whitting, 1964). Peritoneum subject to trauma responds with an inflammatory reaction. Serofibrinous exudate leads to a surface fibrin coat which causes adhesions (Glucksman, 1966).

In view of these facts and judging from the micrographs presented here, it is not unreasonable to assume that mesothelial cells of hernial sacs lose their microvilli as a result of mechanical trauma (Fig. 2), prior to acquiring a surface fibrin coat (Figs. 4, 5) which may subject them to adhesions.

Mesothelial cells are known to produce a fibrinolytic agent which tends to remove fibrin deposits from their surface, thus preventing adhesions. This fibrinolytic agent is a plasminogen activator which converts the enzyme precursor plasminogen into the fibrin-splitting protease, plasmin (Myhre-Jensen et al., 1969). The mesothelial cells of hernial sacs appear to lose their ability to produce the fibrinolytic agent necessary for their protection against surface fibrin which may lead to adhesion. Indeed, we have encountered peritoneal adhesions inside the hernial sacs using transmission electron microscopy (to be published).

Our failure to demonstrate the presence of stomata in the apical cell membrane at the base of the mesothelial microvilli may not necessarily mean that normal human peritoneum is free of such crater-like pores that span the whole height of the mesothelial cell.

Fig. 1. Normal peritoneum of the anterior abdominal wall. Mircrovilli are equally distributed over the free surface of the mesothelial cells, the outlines of which are not easily discernible. × 3,000

Fig. 2. Peritoneum of hernial sac. Denuded areas are seen where microvilli are lacking over the free surface of the mesothelial cells. The outline of the latter are obscure. × 3,000

Fig. 3. Peritoneum of hernial sac. Mesothelial cells are retracted, creating abnormally wide and deep cleavage in the intercellular spaces such that cell outlines have become apparent. Microvilli are reduced in number over the free surface of the mesothelial cells. × 2,000

Fig. 4. Peritoneum of hernial sac. Fibrin-like material covers the free surface of the mesothelial cells. Cell outlines are still discernible. × 2,000

Fig. 5. Peritoneum of hernial sac. Fibrin-like material becomes dense on top of the mesothelial cells covering the cells completely and masking their view. × 2,000

Fig. 6. Normal peritoneum of the anterior abdominal wall. Subserosal fibrous tissue under the mesothelial cells is loose and delicate with no evident collagen bundles in sight. × 500

Fig. 7. Peritoneum of hernial sac. Subserosal fibrous tissue under the mesothelial cells. Collagen build-up is apparent with many bundles in view. × 500

Fig. 8. Peritoneum of hernial sac. A higher power view of subserosal fibrous tissue under the mesothelial cells, giving a clear picture of the considerable thickness of collagen bundles. This is unlike the normal peritoneum of the anterior abdominal wall. × 3,000

Our access to normal human peritoneum material was restricted to only very small areas. A survey study of the entire peritoneum would be needed in order to prove whether stomata are present or not. Permeability studies reviewed by Leak and Rahil (1978) leave no doubt as to the existence of stomata in laboratory animals and to their vital physiological significance.

Our results have consistently shown a build-up in collagen synthesis in the subserosal fibrous tissue of sacs of direct and indirect hernias (Figs. 6, 7, 8). It is not possible to judge from the present study whether such a build-up took place before or after herniation. The current accepted surgical concept is that direct inguinal hernias are acquired as the result of a developed weakness of the *transversalis fascia* in Hesselbach's area and that such hernias may be related to hereditary or acquired defects in collagen synthesis or turnover (Dunphy et al., 1983). Even if collagen build-up did occur as a result of, and not before herniation, it is difficult to imagine how a hereditary defect in collagen synthesis would manifest itself locally in the inguinal area rather than systemically all over the body. In that respect our results are clearly in contradiction with the current surgical concept.

Acknowledgements. Thanks to Mrs Eva Smialowska for technical assistance and to Professor D. Gareth Jones for reading the manuscript.

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Accepted October 15, 1985