Studies on the Biology and Treatment of Recurrent Inguinal Hernia:

II. Morphological Changes

ERLE E. PEACOCK, JR., M.D., JOHN W. MADDEN, M.D.

From the Department of Surgery, University of Arizona, Arizona Medical Center, and the Tucson Veterans Administration Hospital, Tucson, Arizona, 85724

The architectural structure of the endopelvic fascia in both inguinal areas has been studied in 20 patients with unilateral indirect inguinal hernia appearing after the age of 40 and unilateral recurrent inguinal hernia appearing at any age. Regardless of the hernia type, the endopelvic fascia is completely absent in the area of frank herniation. Approximately half the patients showed gross attenuation and thinning of the endopelvic fascia on the clinically normal side. Although the fascial defects in all hernia types were identical, the structure of peritoneal sacs differed significantly. Hernia sacs in primary indirect inguinal hernias were structurally stable suggesting a shape established during embryogenesis; peritoneal sacs from all recurrent hernias regardless of the position of the epigastric vessels had no structural stability and conformed to the fascial defect. Analysis of the epidemiological, experimental, and operative data suggest that the terms direct and indirect hernia have little biological significance. Congenital and acquired hernia seems to be terms which more accurately distinguish between the fundamental biological processes involved in the development of inguinal herniation. Therapeutic considerations can be based on an analysis of the biological factors involved.

INDIRECT INGUINAL HERNIA appearing first in a man over 40 years of age or recurrent indirect hernia following high ligation of the peritoneal sac by a competent surgeon are not readily explained by failure of the processus vaginalis to become obliterated during development of the inguinal region or by failure of the surgeon to identify and remove a peritoneal sac. Moreover, operative findings in infants suggest that approximately 40% of males pass into and through childhood with a patent processus vaginalis but less than half of these children develop clinically apparent herniation of peritoneal contents.[3,10-13,15] In some patients, therefore, factors other than a patent processus vaginalis must be important in development of an indirect inguinal hernia. Moreover, it is also possible that similar factors also play a role in the etiology of repeated recurrence of direct hernias following seemingly adequate reconstruction of the transversalis fascia.[2,9,14]

Study of factors producing inguinal hernias in rats revealed that a peritoneal-lined sac alone did not produce inguinal hernia under the conditions of the experiment.[1] Mechanical abnormalities in the muscular sphincter of the internal ring or metabolic abnormalities reducing structural integrity of endopelvic fascia on the medial wall of the internal ring or a combination of both were required for intestine to pass into a scrotal sac. Such data support the theory that indirect inguinal hernia in some patients is not simply the result of a patent processus vaginalis. Other factors must be important, particularly in late appearance of indirect hernias and in repeated recurrence of direct hernias.

Although the gross anatomical abnormalities present in inguinal herniation have been adequately described, few controlled observational studies have been performed. Comparing inguinal wall structures on both sides of patients with unilateral herniation should reveal structural and biochemical factors contributing to herniation

Supported by Grant AM 14047, National Institute of Arthritis and Metabolic Diseases, National Institutes of Health, Bethesda, Maryland, 20014.

Presented at the Annual Meeting of the Southern Surgical Association, December 3–5, 1973, Hot Springs, Virginia.

of peritoneum through the abdominal wall. Experimental data obtained from studying a rat model suggest that transversalis fascia in the medial wall of the internal ring might show abnormalities important in the pathogenesis of inguinal hernia[1] In order to extend these observations to humans, we designed a protocol comparing gross appearance and certain biochemical characteristics of transversalis fascia medial to the internal ring on both sides of patients with unilateral inguinal hernias. This paper reports the gross structural differences observed in the transversalis fascia of patients participating in the study. Biochemical data will be reported in a subsequent publication.

Methods

Patients at the Tucson Veterans Hospital and the University of Arizona Medical Center were admitted to the study if they had either a unilateral indirect inguinal hernia appearing after the age of 40 or a unilateral recurrent inguinal hernia of any kind without obvious cause for recurrence (infection, hematoma, early dehiscence, etc.). The decision to use the other side as a control required that only patients with unilateral hernias be studied. Although metabolic studies on connective tissue elsewhere in the body might have provided adequate biochemical controls and cadaver dissections in patients without a hernia might possibly have provided structural controls, our hypothesis was based upon multiple abnormalities (metabolic and/or structural) being required for a hernia to develop. Thus, inguinal fascia from the nonherniated side of the same individual seemed the only legitimate control if differences responsible for appearance of a hernia were to be identified. Tissues removed for biochemical or biophysical study had to be positively identified on both sides for comparisons to be valid. Particularly, in patients with recurrent hernias, an approach through unoperated upon tissue seemed imperative less scar tissue with relatively high turnover of collagen be included in the specimen.[4] Exploration through a classical inguinal incision did not seem advisable because studies in the rat model suggested that abnormalities might be found in transversalis fascia on the medial wall of the internal ring.[1] Positive identification of this tissue in exactly the same location on both sides posed difficult exposure problems through a typical inguinal incision. Finally, a wide view of the entire endopelvic fascia seemed desirable because recurrent hernias, in spite of adequate repair with local tissue and adjacent flaps, probably are the result of an abnormality extending over a wider area than inguinal exposure reveals. Such considerations made preperitoneal approach ideal in many respects.

Repair of inguinal hernia through a preperitoneal approach has not been as reliable for many surgeons as Nyhus and Harkins reported.[6] In addition to concern about being able to repair the hernia adequately, we reasoned that removal of even a small fragment of tissue from the medial wall of the internal ring on the normal side might produce an inguinal hernia. Thus, better results than recently reported seemed mandatory if preperitoneal exposure was utilized. For this reason, a complete reconstruction of the endopelvic fascia by means of a large onlay graft was developed to strengthen the entire pelvic floor after conventional repair of the transversalis fascia. Details of this rather complex reconstruction and the results in 25 patients over a five-year period will be presented subsequently.

Because preliminary results utilizing the new technique were favorable, both inguinal areas of patients in the study were exposed through a preperitoneal approach. After entering the preperitoneal space through a single transverse lower abdominal incision placed approximately two finger breadths above the symphysis pubis, the pelvic peritoneum was dissected out of the pelvis and retracted superiorly exposing the internal ring and medial transversalis fascia on both sides. This maneuver requires the dissection and delivery of the peritoneal sac on the herniated side. At the completion of the dissection, the sac was allowed to assume its natural shape by being placed upon a towel. The shape and structural stability of the sac were observed and photographed. After examining the internal ring on both sides, a photograph was taken to show fine structural details in the transversalis fascia. Variation in thickness of transversalis fascia and relationship of abnormalities in the fascia to the epigastric vessels were noted. Fascial architecture at the margin of defects and the parameter of the internal ring were studied carefully; transitional changes between normal fascia and areas where fascia was completely absent were noted and photographed. Biopsies of transversalis fascia were excised from the edge of the defect on the herniated side and from a similar position on the control side. After repair of the defect and the biopsy site, suture lines were observed for structural strain or attenuation of fascia secondary to tension produced by sutures prior to the fascial graft.

Results

Twenty patients were admitted to the study group. All were males and ranged in age from 40 to 65. There were four unilateral primary indirect hernias and 16 recurrent inguinal hernias. The morphologic observations on the herniated side only confirmed previous reports, but the architecture of the clinically normal side proved to be of major interest.[7] In each patient, the herniated side showed a well-defined hole in the transversalis fascia. Although the process producing the hole or the mech-

anism responsible for enlargement of the defect and extrusion of abdominal contents were not evident, our observations suggested that structural and metabolic abnormalities were limited to the site of the defect. Margins of the defect on the herniated side were definite and abrupt; normal appearing fascia was present at the margins in all cases. The uninvolved inguinal region in nine of the 20 patients appeared grossly normal. The transversalis fascia of the inguinal area blended smoothly with the transversalis fascia of the abdominal wall and no structural differences were noted. In contrast, on the clinically normal side, 11 of the 20 patients had marked attenuation of endopelvic fascia and significant reduction in thickness of connective tissue in the area of the internal ring. Moreover, the process clearly was medial to the cord and often beneath the epigastric vessels. Fascial abnormalities occurred on either side of the epigastric vessels and often on both sides. In each case, thinning and attenuation of fascia was confined sharply to the inguinal area. This gross pathological abnormality in the absence of an identifiable hernia suggests a metabolic abnormality of connective tissues localized to this area. Specimens from the edge of the hernia defect and specimens from the attenuated fascia are being analyzed biochemically and may expose the metabolic defect. Although data are not available, our findings suggest that as many as half of our patients might have developed a contralateral hernia with time.

Although the fascial architecture in both recurrent and in primary indirect hernias was comparable, the architecture of the peritoneal sac differed significantly. In primary indirect hernias, the peritoneal sac held its shape after removal from the internal ring. The neck and body of the peritoneal sac conform to the inguinal ring and also maintain their internal architecture regardless of the size or shape of external structures. The structural stability of the peritoneal sac from primary indirect inguinal hernias may have been established during embryogenesis or could be the result of extensive remodeling of dense connective tissue. Peritoneal sacs in recurrent direct or indirect hernias presented different structural characteristics. The sac in patients with recurrent hernias seemed to be a passively formed structure lacking internal stabilizing structure. Peritoneal protrusions seemed merely to conform to the size and shape of the opening in the transversalis fascia. Moreover, in patients with markedly attenuated transversalis fascia but without clinically apparent hernia, the peritoneum seemed entirely normal. Regardless of the state of attenuation of endopelvic fascia, there was no evidence that a similar process was occurring in the peritoneum; in no instance was a leading point or finger of peritoneum invading fascia before the fascia actually disappeared. These findings suggest that the peritoneum in recurrent hernias is nothing more than a passive space filler and does not induce or contribute anything more than a bougienage effect.

The recurrence of a hernia in one of our early patients contributed greatly to our understanding of the formation and importance of the peritoneal sac. At initial surgery, a typical fascial defect resulting in a large recurrent direct inguinal hernia was defined medial to the epigastric vessels. The sac, when delivered from the defect, did not maintain its shape; it "flowed" into the defect and could not be identified as a preformed sac once the peritoneum was delivered. As usual, the entire pelvic peritoneum was swept out of the pelvis during closure of the defect. Although the internal ring was larger than normal, there were no peritoneal or intraperitoneal structures protruding into the inguinal canal; no indirect hernia sac was present. Because of an identifiable technical error, the placement of the fascial graft around the cord structures was not mechanically accurate at the time of repair. Four weeks following his surgery, the patient developed pain and observed an inguinal mass superior and lateral to the previous hernia. The patient was re-explored through a groin incision and peritoneum was found protruding into, although not yet emerging from, the inguinal canal lateral to the epigastric vessels. The peritoneal sac was pushing the anterior wall of the inguinal canal ahead of it. According to conventional terminology, the patient had a primary indirect inguinal hernia. Although the hernia was lateral to the epigastric vessels and within the internal ring, the structure of the peritoneal sac and direction of the protrusion were typical of a direct hernia. The peritoneal sac was amputated and the defect repaired by suturing transversalis fascia (graft) to Cooper's ligament. The previously placed fascial graft was identified easily in the site transversalis fascia usually is found.[5] The graft provided abundant tissue of excellent strength which was brought without tension to Cooper's ligament. Thus, in this patient, failure to ligate a peritoneal sac could not have been the cause of recurrent inguinal hernia. The herniation, although recurrent in temproal sense, was primary in the area where it occurred and appeared to be the result of an uncorrected structural defect. Technical measures to prevent similar occurrences have been devised and will be reported elsewhere. The biological implications of these observations, however, are important.

Our observations on the structural changes in the inguinal region of patients in this study can be summarized as follows: the transversalis or endopelvic fascia was absent when an inguinal hernia was clinically apparent. Approximately half of the patients had significant structural changes present in the endopelvic fascia of the clinically normal side. Gross pathological changes included attenuation, thinning, or frank absence of fascia. Structural abnormalities were not related to the position

of the epigastric vessels. In contrast to the similarity in fascial architecture, peritoneal sacs in primary indirect and in recurrent hernias of all types differed markedly. The neck and body of primary indirect sacs appeared precast and retained their size and shape after delivery from the inguinal canal. Peritoneal sacs from recurrent indirect and direct hernias were formless bags without internal stabilization; recurrent and direct sacs conformed to the shape of the external environment and did not maintain their structural configuration following removal from the fascial defect.

Discussion

Observations reported in this paper strongly suggest that the usual classification of direct and indirect inguinal hernia has little biological or therapeutic significance. The fascial architecture in all hernias, whether primary indirect or recurrent direct and indirect, was identical. The only significant architectural differences occurred in the structure of the peritoneal sac. The inherent structural stability of primary indirect inguinal sacs suggests a peritoneal structure established during the development of the inguinal region. The absence of form in all other hernia sacs suggests that the peritoneum played a minor role in development of the hernias. The incidence of patent peritoneal sacs in infants and laboratory studies in rats support the concept that the presence of a peritoneal sac alone is not sufficient pathology to produce clinical herniation. Based on the biological information available, we suggest a new classification of inguinal hernias: congenital and acquired. Although in both congenital and acquired types multiple concurrent abnormalities must be present before herniation occurs, the pathological factors present differ significantly and have different therapeutic implications.

In the congenital type, a patent processus vaginalis is always present prior to herniation. If the patent sac constitutes the major abnormality, high ligation and removal of the peritoneal process will correct the clinical abnormality. This seems to be the case in the majority of congenital hernias. Patent sacs, alone, however, do not lead to herniation in all cases. In some patients, structural abnormalities of the internal ring, acquired attenuation of the transversalis fascia, or abnormal muscle function may accompany the congenital defect. This seems particularly true in patients developing primary indirect inguinal hernias in their middle years. Unless steps are taken to correct the concurrent defects, recurrence is frequent.

In acquired inguinal hernias, the peritoneum seems to play a minor role in the development of clinical herniation. Our morphological studies suggest the presence of a fundamental metabolic defect in collagen metabolism. Whether this defect is generalized but expressed only in the inguinal region because of local mechanical factors or is truly a local process is unknown. The gross fascial abnormalities reported in this paper could result from decreased collagen synthesis or increased collagen degradation. Whatever the cause, the end result is attenuation and ultimately absence of endopelvic fascia. Our data indicate that the fascial defect can occur with equal frequency on either side of the epigastric vessels. In patients with patent peritoneal sacs, the fascial abnormality leads to the development of indirect inguinal herniation. In patients without patent peritoneal processus, fascial dissolution may result in recurrent indirect or direct herniation. Thus, if acquired metabolic defects are present, indirect inguinal hernia can recur in spite of adequate ligation and removal of the peritoneal sac.

Permanent repair of an acquired hernia probably depends upon the extent of the abnormal metabolic process. If normal fascial tissues can be exposed, conventional surgical repair of the inguinal area will be permanent. If abnormal tissue is included in the repair or if the basic metabolic abnormality continues in the tissues used for reconstruction, recurrence is inevitable. Unfortunately, there are no simple laboratory tests to delineate which tissues have or will develop the metabolic abnormality. Until such tests are developed, the clinical behavior of the patients is the only guide to therapy. Postlethwait's data demonstrate that inguinal hernias can recur as long as 15 years following the initial surgery.[8] Within six months, all of the technical factors under the control of the surgeon should have played their roles; recurrence after six to nine months seems more likely the result of continuing disease rather than technical errors or selection of an inferior procedure. Based on the morphological data presented, we now assume that recurrent inguinal hernias are the result of a localized mesenchymal metabolic defect unless a positive technical mistake can be identified. Subsequent repair, therefore, should include the transplantation of normal fascial tissues from other areas of the body.

Classical inguinal herniorrhaphy performed by competent surgeons in cooperative patients solves the problem permanently in 96% of primary indirect hernias and approximately 70% of recurrent hernias. A continuing disease process rather than incompetent surgery may be responsible for the high incidence of recurrence. By discarding the conventional classification of direct and indirect hernia and concentrating on a classification with greater etiological and biological implications (congenital and acquired), the fundamental biological abnormalities may be identified accurately and useless repetition of operative procedures eliminated. For patients with recurrent inguinal hernia, creativity and curiosity may be more important attributes for the surgeon than pride and technical expertise.

References

1. Conner, W. T. and Peacock, E. E., Jr.: Some Studies on the Etiology of Inguinal Hernia. Am. J. Surg., (In Press).
2. Glassow, F.: Recurrent Inguinal and Femoral Hernia: 3,000 Cases. Can. J. Surg., **7**:284, 1964.
3. Guttman, F. M. and Ducharme, J. C.: Herniography and the Pediatric Contralateral Inguinal Hernia. Surg. Gynecol. Obstet., **135**:551, 1972.
4. Madden, J. W. and Peacock, E. E., Jr.: Studies on the Biology of Collagen during Wound Healing. III. Dynamic Metabolism of Scar Collagen and Remodeling of Dermal Wounds. Ann. Surg., **174**:511, 1971.
5. Mizrachy, B. and Kark, A.: The Anatomy and Repair of the Posterior Inguinal Wall. Surg. Gynecol. Obstet., **137**:253, 1973.
6. Nyhus, L. M., Condon, R. E. and Harkins, H. N.: Clinical Experiences with Preperitoneal Hernial Repair for all Types of Hernia of the Groin: With Particular Reference to the Importance of Transversalis Fascia Analogues. Am. J. Surg., **100**:234, 1960.
7. Nyhus, L. M. and Harkins, H. N.: Hernia Philadelphia, J. B. Lippincott Co., 280, 1964.
8. Postlethwait, R. W.: Causes of Recurrence after Inguinal Herniorrhaphy. Surgery, **69**:772, 1971.
9. Quillinan, R. H.: Repair of Recurrent Inguinal Hernia. Am. J. Surg., **118**:593, 1969.
10. Rowe, M. K., Copelson, L. W. and Clatworthy, H. W.: Patent Processus Vaginalis and Inguinal Hernia. J. Pediat. Surg., **4**:102, 1973.
11. Seaman, W. B.: The Case of the Hidden Hernia. Hosp. Pract., **8**:105, 1973.
12. Shackelford, G. D. and McAlister, W. H.: Inguinal Herniography. Am. J. Roentgenol. Radium Ther. Nucl. Med., **115**: 399, 1972.
13. Sparkman, R. S.: Bilateral Exploration in Inguinal Hernia in Juvenile Patients. Surgery, **51**:393, 1962.
14. Thieme, E. T.: Recurrent Inguinal Hernia. Arch. Surg., **103**: 238, 1971.
15. White, J. J., Haller, J. A. and Dorst, J. F.: Congenital Inguinal Hernia and Inguinal Herniography. Surg. Clin. N. Am., **50**:823, 1970.

Discussion

Dr. Gilbert S. Campbell (Little Rock): Dr. Read and Dr. Wagh, of the Surgery Department at Little Rock, have been interested for a number of years in connective tissue abnormalities in patients developing inguinal herniation.

They presented their findings at the European meeting of the Society of University Surgeons at Garmisch this past September. In brief, they have shown that the anterior rectus sheath is thinner in adults with inguinal herniation, especially those with direct inguinal herniation.

Its collagen content is diminished, especially in direct herniation. Fibroblasts cultured from the rectus sheath in men with inguinal herniation take longer to divide, and incorporate radioactive proline less actively than do the controls.

Purified collagen in the rectus sheath biopsies obtained from patients with direct hernia failed to precipitate normally at neutral pH, and contained 30 per cent less hydroxyproline than normal. Their results will be published, shortly.

I would have to say that I'm not sure how important collagen and some of these ultrastructural organelles are.