Original Article

To Identify the Risk Factors Associated with Development of Anterior Abdominal Wall Hernia

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Abstract

Background: Numerous risk factors for the development of hernia have been identified, including abnormal collagen fiber organization and increased intra-abdominal pressure. **Objectives:** The purpose of this study is to investigate if one or both of these factors (arrangement of collagen fibers and increased intra-abdominal pressure) contribute to the formation of hernias. **Materials and Methods:** Adolescents and adults with anterior abdominal wall hernia were admitted to the hospital and underwent surgery after completing a standard preoperative evaluation protocol that included a detailed medical history, physical examination, and laboratory tests. Prior to anesthesia induction in the operation room, intra-abdominal pressure was measured using a Foley's catheter attached to a pressure transducer. According to their condition, the patients underwent routine surgery, either laparoscopic or open. Excision of the sac and submission to the histo-pathological examination for collagen analysis using two different stains, Masson's trichrome and Gomori's reticulin. Categorical variables were quantified using numbers and percentages (percent), whereas continuous variables were quantified using the mean, standard deviation, and median. The chi-square test was used to ascertain correlations between qualitative variables. $P < 0.05$ was considered statistically significant. **Results:** We considered a total of 200 patients, 50 of whom served as controls. Elevated intra-abdominal pressure has been identified as a risk factor for ventral wall hernia. Collagen fibers in patients with hernias were found to be disorganized at the microscopic level. Obesity and elevated intra-abdominal pressure were found to be statistically significant predictors of collagen disorder and the development of hernias. Hernias are more likely to occur as people age. Although chronic cough has been linked to metabolic disorders such as diabetes, hypertension, and kidney disease, we found no statistically significant link. **Conclusions:** Intra-abdominal pressure elevation, disordered collagen fiber arrangement, obesity and increasing age are risk factors associated with hernia development.

Keywords: Anterior abdominal wall hernia, collagen disorderliness, intra-abdominal pressure, intravesical pressure

Introduction

Approximately 5% of the world's population has a hernia, with 75% of hernias occurring in the inguinal region. Approximately two-thirds of these hernias are indirect hernia. Males experience groin hernia 25 times more frequently than females and have a 2:1 ratio of indirect to direct inguinal hernia. Females are more likely to develop femoral and umbilical hernias. In the United States of America, over 6 million hernias are repaired each year.[1]

Hernias in the anterior abdominal wall can be caused by a variety of factors. These risk factors may be modifiable or irreversible. We are powerless to alter our genetic mutations causing collagen disorders, previous surgical procedures, or developmental abnormalities. Constipation is a risk factor that can be mitigated through lifestyle modifications. Incisional hernias are associated with prior abdominal surgeries. Numerous risk factors have been proposed, including abnormal collagen fiber arrangement and increased intra-abdominal pressure.[2]

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Numerous studies have been conducted and discussed the risk factors for hernias. The purpose of this study was to determine whether one or both of these factors (collagen fiber arrangement and increased intra-abdominal pressure) contributed to the development of hernias.[3] Although numerous studies and publications have been conducted and published on the various risk factors for anterior abdominal wall hernias, only a few have established a link between intra-abdominal pressure and the development of hernias. Numerous researchers have attempted to understand the pathology of hernias. These studies did not examine the arrangement or orderliness of collagen fibers in hernia sac tissue, nor did they investigate the effect of intra-abdominal pressure on collagen fiber orderliness. This study aimed to fill knowledge gaps in this area.

Materials and Methods

Following approval by the institutional ethics committee, a case series analysis was conducted at a single institution over a 21-month period from January 2019 to September 2020. According to previous research, abdominal wall hernias accounted for 15%–18% of all surgical procedures.[4] 188 patients were required to achieve a 5.5% margin of error and a 5% level of significance using this value as a reference. To minimize the margin of error, 200 people were sampled. Adolescents and adults with anterior abdominal wall hernias (epigastric, umbilical and paraumbilical, incisional and groin) were admitted to the hospital and underwent surgery following a standard preoperative evaluation protocol that included a thorough history, physical examination, and laboratory testing.

Measurement of intra-abdominal pressure was determined prior to the operation theatre's induction of anesthesia. This was determined by connecting a Foley's catheter to a pressure transducer. The patient underwent routine surgery, either laparoscopic or open, depending on the severity of the condition. The sac was excised and analyzed for collagen by the histopathological examination. Two stains were used to visualize collagen fibers within the hernial sac.

1. Masson's trichrome stain: to show collagen fibers in tissue section. Collagen was stained blue and Nuclei were stained black. Muscle, cytoplasm, keratin were stained red.
2. Gomori's reticulin stain: to show reticulin fibers in the tissue. Reticulin fiber was stained black and other structures like coarse connective tissue fiber were stained brown pink. Nuclei were stained grey to black.

Postoperative care was standard, and patients were discharged once they were ambulatory and capable of consuming oral feeds, as well as voiding and passing stool.

Statistical analysis

Categorical variables were expressed in terms of numbers and percentages (%), whereas continuous variables were expressed as mean, standard deviation, and median. Correlations between qualitative variables were determined using the chi-square test. Statistical significance was defined as $P < 0.05$. The data were entered into an MS Excel spreadsheet and analyzed using SPSS software program, version 21.0.

Ethical consideration

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patients verbal and analytical approval before sample was taken. The study protocol and the subject information and consent form were reviewed and approved by a local ethics committee according to the document number 55 (including the number and the date in 13/11/2018) to get this approval.

Results

During the study period, 479 hernias were repaired. A total of 279 participants were excluded from the study due to the fact that 70 were under the age of 16 and 209 refused to consent. Thus, for statistical analysis, 200 patients were included in the study. In addition, we collected fascia transversalis tissue samples from 50 patients who underwent abdominal surgery for reasons other than hernia repair. The demographic characteristics of the study sample are summarized in Table 1.

Table 2 compares different study parameters per age group.

Table 3 compares study parameters with the type/location of hernia.

Table 4 compares the histology of hernia sacs (orderliness of collagen) to several demographic and clinical characteristics.

The relationship between intra-abdominal pressure and demographic and clinical variables is depicted in Table 5.

Discussion

We examined the factors that contribute to anterior abdominal wall hernia in this study. It was hypothesized that the disorderliness of collagen in the hernia sac was the molecular basis for hernia development. In addition, we investigated and evaluated the primary etiological factors, which included increased intra-abdominal pressure and body mass index (BMI). We discovered that people in their middle years (40–60 years) had the highest prevalence of ventral hernia, followed by the elderly, which is consistent with previous research.[5-7] Tensile strength of the fascia transversalis and rectus sheath decreases with age, possibly because these tissues contain less collagen than those of younger age groups.[6,7] Ventral hernia is associated with increasing age.

The majority of our patients suffered from inguinal hernias (52%). The average BMI of the patients was 25 kg/m^2. Obesity may be a risk factor for the development of ventral

Table 1: Sample's demographic characteristics

Demographic parameters	Frequency (*n*)	%
Sex		
Male	55	27.50
Female	145	72.50
Age distribution (years)		
16–20	17	8
20–40	26	13
40–60	123	62
>60	34	17
Body mass index (BMI) distribution (kg/m^2)		
<18.5 (underweight)	3	1.5
18.5–24.9 (ideal BMI)	78	39
25.0–29.9 (overweight)	106	53
>=30 (obese)	13	6.5
Type/location of hernia		
Epigastric	11	5.5
Incisional	20	10
Incisional + inguinal	1	0.5
Inguinal	104	52
Umbilical/paraumbilical	62	31
Umbilical/paraumbilical + inguinal	2	1
Distribution of complaints		
Swelling	182	91
Pain	57	28.5
Signs of obstruction	12	6
Associated factors/comorbidities		
None	47	23.5
Constipation	43	21.5
Diabetes	58	29
Chronic cough/COPD	16	8
Liver disease	12	6
Congenital defect	16	8
Hypertension or CVD	56	28
Renal disease	10	5
History of previous abdominal surgery		
Yes	40	20
No	160	80
Orderliness of collagen in hernia sac		
Non-orderly arranged	157	78.5
Orderly arranged	43	21.5
Intra-abdominal pressure (mm Hg)		
0–5 mm Hg	51	25.5
>5 mm Hg	149	74.5
Distribution of content of hernial sac		
Bowel	6	3
Bowel + other viscera	1	0.5
Omentum	139	69.5
Omentum + bowel	53	26.5
Omentum + other viscera	1	0.5

hernias. The same conclusion was reached by Ruhl and Everhart.[8] Obesity is almost certainly associated with an increase in intra-abdominal pressure. BMI had a statistically significant correlation with increased intra-abdominal pressure (P = 0.0003). Twenty-one of 40 patients who had previously undergone abdominal surgery developed an incisional hernia in our study. A weakness in the incision line may result in the development of a hernia. Previous research[9] has discovered a similar result. In addition, scar hernias may heal with an abnormal type of collagen.

Previously, it was believed that impaired collagen synthesis contributed to the development of ventral wall hernias. Collagen fibers were found to be arranged irregularly in hernia sac specimens collected from hernia patients in our study, most likely due to altered cross linkage, a deficient ratio of type 1 and type 3 collagen fibers, or a decrease in overall collagen fiber production.[10] On the contrary, hernia-free tissue specimens do not show disordered collagen tissue architecture. However, no study has been able to characterize the arrangement or organization of collagen fibers in patients with ventral hernia's hernia sac or fascia transversalis.

Our study enrolled 200 patients with hernia sacs and 50 patients who underwent hernia repair in addition to non-hernia abdominal surgery. Collagen fibers were distorted in 157 of the 200 patients, but not in any of the 50 control samples. A ventral wall hernia occurs as a result of a change in the tissue's collagen fibers.[11]

In 149 of 200 patients, intra-abdominal pressure was greater than 5 mm Hg, indicating that it is a risk factor for ventral wall hernia development. Chronic tissue strain has the potential to alter the tissue's structure. These changes, which manifest as an irregular arrangement of collagen fibers, can be caused by increased intra-abdominal pressure.[12] This may result in the formation of a hernia.

In middle-aged and elderly patients, the collagen fiber arrangement was found to be the most disorganized. Collagen fibers with a distorted structure were detected only in patients with ventral hernia and in the elderly, but not in a few younger patients.[13] Most likely as a result of a genetic predisposition, increased intra-abdominal pressure caused by strenuous exercise, constipation or irregular bowel habits, or some underlying malnutrition. When age groups were compared, we discovered that individuals' resting intra-abdominal pressure increased with age. This could be because BMI increases with age in the Indian population due to reduced physical activity, a sedentary lifestyle, or the presence of comorbid conditions such as diabetes and chronic obstructive pulmonary disease (COPD). Furthermore, as previously stated, BMI is inversely proportional to intra-abdominal pressure.

In all age groups, the omentum was discovered to be the primary component. In the elderly, the bowel was associated with the omentum. This may be the result of a chronic hernia. The bowel loop or other nearby viscera gravitates toward the hernia as it grows in size. Due to the absence of data on the duration and content of hernias, this hypothesis warrants further investigation.

The most disorganized collagen fibers in the hernia sac were found in umbilical/paraumbilical hernias, followed by incisional, inguinal, and epigastric hernias. However, we were unable to ascertain the reason for this occurrence. Intra-abdominal pressure elevation aided in the development of ventral wall hernia.[14] Umbilical/paraumbilical and

Table 2: Comparison of various study parameters across age groups

Clinical parameters	<20 (*n* = 17)	20–40 (*n* = 26)	40–60 (*n* = 123)	>60 (*n* = 34)	Total	*P* Value	Test performed
HPE of hernia sac (orderliness of collagen)							
Non-orderly arranged	4 (23.53%)	12 (46.15%)	110 (89.43%)	31 (91.18%)	157 (78.50%)	<.0001	Chi-square test, 58.5
Orderly arranged	13 (76.47%)	14 (53.85%)	13 (10.57%)	3 (8.82%)	43 (21.50%)		
Intraabdominal pressure							
0–5 mm Hg	14 (82.35%)	9 (34.62%)	24 (19.51%)	4 (11.76%)	51 (25.50%)	<.0001	Chi-square test, 35.759
>5 mm Hg	3 (17.65%)	9 (34.62%)	99 (80.49%)	30 (88.24%)	149 (74.50%)		
Content of hernia sac							
Bowel	0 (0%)	0 (0%)	3 (2.44%)	3 (2.44%)	6 (3%)	<.0001	Fisher exact test
Bowel + other viscera	1 (5.88%)	0 (0%)	0 (0%)	0 (0%)	1 (5.88%)		
Omentum	16 (94.12%)	23 (88.46%)	89 (72.36%)	11 (32.35%)	139 (69.50%)		
Omentum + bowel	0 (0%)	3 (2.44%)	30 (24.39%)	20 (58.82%)	53 (26.50%)		
Omentum + other viscera	0 (0%)	0 (0%)	1 (5.88%)	0 (0%)	1 (5.88%)		

HPE: Histo-pathological examination

Table 3: Comparison of study parameters with the type/location of hernia

	Epigastric (*n* = 11)	Incisional (*n* = 20)	Incisional + inguinal (*n* = 1)	Inguinal (*n* = 104)	Umbilical/paraumbilical (*n* = 62)	Umbilical/paraumbilical + inguinal (*n* = 2)	Total	*P* Value	Test performed
HPE of hernial sac									
Non-orderly arranged	7 (63.6%)	16 (80%)	1 (100%)	74 (71.15%)	57 (91.94%)	2 (100%)	157 (78.50%)	0.013	Fisher exact test
Orderly arranged	4 (36.36%)	4 (20%)	0 (0%)	30 (28.85%)	5 (8.06%)	0 (0%)	43 (21.50%)		
Intraabdominal pressure									
0–5 mm Hg	6 (54.55%)	3 (15%)	1 (100%)	33 (31.73%)	8 (12.90%)	0 (0%)	51 (25.50%)	0.003	Fisher exact test
>5 mm Hg	5 (45.45%)	17 (85%)	0 (0%)	71 (68.27%)	54 (87.10%)	2 (100%)	149 (74.50%)		
Content of hernia sac									
Bowel	0 (0%)	3 (15%)	0 (0%)	3 (2.88%)	0 (0%)	0 (0%)	6 (3%)	<.0001	Fisher exact test
Bowel + other viscera	0 (0%)	0 (0%)	0 (0%)	1 (0.96%)	0 (0%)	0 (0%)	1 (0.50%)		
Omentum	9 (81.82%)	3 (15%)	0 (0%)	88 (84.62%)	37 (59.68%)	2 (100%)	139 (69.50%)		
Omentum + bowel	2 (18.18%)	13 (65%)	1 (100%)	12 (11.54%)	25 (40.32%)	0 (0%)	53 (26.50%)		
Omentum + other viscera	0 (0%)	1 (5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (0.50%)		

HPE: Histo-pathological examination

Table 4: Comparison of histopathology of hernia sac (orderliness of collagen) to various demographic and clinical variables

HPE of hernial sac	Non-orderly arranged	Orderly arranged	Total	*P* Value	Test performed
Associated constipation					
No constipation (n = 157)	120 (76.43%)	37 (23.57%)	157 (100%)	0.174	Chi-square test, 1.848
Constipation (n = 43)	37 (86.05%)	6 (13.95%)	43 (100%)		
Associated diabetes					
Nondiabetic (n = 142)	105 (73.94%)	37 (26.06%)	142 (100%)	0.014	Chi-square test, 6.023
Diabetic (n = 58)	52 (89.66%)	6 (10.34%)	58 (100%)		
Associated chronic cough/COPD					
No chronic cough/COPD (n = 184)	142 (77.17%)	42 (22.83%)	184 (100%)	0.202	Fisher exact test
Chronic cough/COPD (n = 16)	15 (93.75%)	1 (6.25%)	16 (100%)		
Associated liver disease					
No liver disease (n = 188)	146 (77.66%)	42 (22.34%)	188 (100%)	0.468	Fisher exact test
Liver disease (n = 12)	11 (91.67%)	1 (8.33%)	12 (100%)		
Type of hernia (congenital/acquired)					
No congenital defect (n = 184)	153 (83.15%)	31 (16.85%)	184 (100%)	<.0001	Fisher exact test
Congenital defect (n = 16)	4 (25%)	12 (75%)	16 (100%)		
Associated hypertension or cardiovascular diseases					
No hypertension or CVD (n = 144)	104 (72.22%)	40 (27.78%)	144 (100%)	0.0002	Fisher exact test
Hypertension or CVD (n = 56)	53 (94.64%)	3 (5.36%)	56 (100%)		
Associated renal disease					
No renal disease (n = 190)	147 (77.37%)	43 (22.63%)	190 (100%)	0.123	Fisher exact test
Renal disease (n = 10)	10 (100%)	0 (0%)	10 (100%)		
Intra-abdominal pressure					
0–5 mm Hg (n = 51)	25 (49.02%)	26 (50.98%)	51 (100%)	<.0001	Chi-square test, 35.251
>5mm Hg (n = 149)	132 (88.59%)	17 (11.41%)	149 (100%)		
Body mass index (BMI)					
<18.5 (n = 3)	2 (66.67%)	1 (33.33%)	3 (100%)	0.0003	Chi-square test, 12.858
18.5–24.9 (n = 78)	52 (66.67%)	26 (33.33%)	78 (100%)		
25.0–29.9 (n = 106)	90 (84.91%)	16 (15.09%)	106 (100%)		
>=30 (n = 13)	13 (100%)	0 (0%)	13 (100%)		

HPE: Histo-pathological examination

Table 5: Comparison of relationship between intra-abdominal pressure and demographic and clinical variables

Intra-abdominal pressure	0–5 mm Hg	>5 mm Hg	Total	*P* Value	Test performed
Body mass index (BMI)					
<18.5 (*n* = 3)	2 (66.67%)	1 (33.33%)	3 (100%)	0.0003	Chi-square test, 12.858
18.5–24.9 (*n* = 78)	52 (66.67%)	26 (33.33%)	78 (100%)		
25.0–29.9 (*n* = 106)	90 (84.91%)	16 (15.09%)	106 (100%)		
>=30 (*n* = 13)	13 (100%)	0 (0%)	13 (100%)		
Content of hernial sac					
Bowel	0 (0%)	6 (4.03%)	6 (3%)	0.007	Fisher exact test
Bowel + other viscera	0 (0%)	1 (0.67%)	1 (0.50%)		
Omentum	45 (88.24%)	94 (63.09%)	139 (69.50%)		
Omentum + bowel	6 (11.76%)	47 (31.54%)	53 (26.50%)		
Omentum + other viscera	0 (0%)	1 (0.67%)	1 (0.50%)		
Associated chronic cough/COPD					
No chronic cough/COPD (*n* = 184)	49 (96.08%)	135 (90.60%)	184 (100%)	0.368	Fisher exact test
Chronic cough/COPD (*n* = 16)	2 (3.92%)	14 (9.40%)	16 (100%)		
Associated liver disease					
No liver disease (*n* = 188)	50 (98.04%)	138 (92.62%)	188 (100%)	0.303	Fisher exact test
Liver disease (*n* = 12)	1 (1.96%)	11 (7.38%)	12 (100%)		
Associated cardiovascular diseases					
No hypertension or CVD (*n* = 144)	43 (84.31%)	101 (67.79%)	144 (100%)	0.023	Chi-square test, 5.149
Hypertension or CVD (*n* = 56)	8 (15.69%)	48 (32.21%)	56 (100%)		

incisional hernias had the greatest effect. Almost all types of ventral hernias included the omentum. Because it is a free-floating structure beneath the rectus sheath, it has the best chance of protruding into the hernia sac. Long-standing inguinal hernias or incisional hernias were complicating factors.

Constipation increases intra-abdominal pressure, but the correlation between collagen fiber disorder and constipation is not statistically significant ($P = 0.174$). Almost all patients with chronic cough or respiratory illness had a hernia sac with disorderly collagen fibers. Although smoking has been shown to alter the collagen composition of various organs,[15] no statistically significant correlation was observed in this study ($P = 0.202$). The liver's role in protein metabolism is well-known.[16] Cirrhosis of the liver results in malnutrition as a result of impaired amino acid processing and thus collagen synthesis. Due to the heterogeneity of our cohort's comorbidity, it is difficult to prove or disprove this hypothesis.

Because children (0–15 years) were excluded from our study, hernias occurred in a very small proportion of patients with an anterior abdominal wall congenital defect. Furthermore, a congenital ventral wall hernia is detected and treated promptly. We enrolled all patients over the age of 16 and discovered several with an abnormal collagen fiber arrangement in the hernia sac. The likelihood of the fascia transversalis becoming distorted microscopically is

influenced by age and metabolic abnormalities. Although we discovered a statistically significant correlation between collagen disorderliness and congenital hernias, further research with younger age groups is required.

In ventral wall hernias, collagen disorder was associated with cardiovascular disease ($P = 0.0002$). Numerous recent researches[17] have discussed dysregulation of collagen type 1 metabolism in patients with hypertension-induced heart failure's cardiac muscles. In addition, hypertension has been linked to a decrease in the pliability of the anterior wall. This could be a result of changes in the collagen structure and the ratio of elastin to collagen in body tissues.

Our study discovered that the collagen fiber composition of all patients with renal disease was altered, but the underlying mechanism is unknown. This relationship was not statistically significant ($P = 0.123$).

A significant proportion of patients with elevated intra-abdominal pressure had an unorganized arrangement of collagen fibers in the hernial sac (0.0001). Increased intraabdominal pressure places mechanical strain on the collagen structure, resulting in a distorted collagen structure in the fascia transversalis. The correlation between hernia development and collagen disorderliness supports our study's fundamental hypothesis that increased intra-abdominal pressure results in the formation of hernias. There is no evidence for such a correlation in the literature.

Our study established that increased BMI is associated with an increased risk of anterior abdominal wall hernias. During microscopic examination of the hernial sac, the rectus sheath and fascia transversalis were discovered to be disordered in such individual's collagen structure.[18] This was a statistically significant result ($P = 0.0003$).

Age had a significant effect on both BMI and intra-abdominal pressure ($P = 0.003$). It was discovered that a sizable proportion of cases had both an increased BMI and increased intra-abdominal pressure. In a study of morbidly obese patients, increased intra-abdominal pressure was observed. Thus, both intra-abdominal pressure and body mass index are significantly associated with the development of ventral wall hernias. Our study found no correlation between intra-abdominal pressure and chronic cough or patients with COPD ($P = 0.202$).

We had 12 patients who required hernia surgery due to decompensated liver disease. In such patients, ascites increases intra-abdominal pressure, which may contribute to the development of a hernia. This correlation appears to be logical, but the *P*-value was insignificant ($P = 0.253$). Although numerous studies on the various risk factors for ventral wall hernias have been conducted and published, few have established a link between intra-abdominal pressure and hernia development. Numerous researchers have attempted to understand hernia pathology but have neglected to examine the arrangement or orderliness of collagen fibers in hernia sac tissue or the effect of intra-abdominal pressure on collagen fiber orderliness. The purpose of this study was to accomplish the same objective, namely to close knowledge gaps.

Conclusion

The purpose of this study was to ascertain the role of age, gender, BMI, and other risk factors in hernia development, as well as the orderliness of the collagen in the hernia sac. Intra-abdominal pressure elevation was identified as a significant risk factor for ventral wall hernia. On a microscopic level, hernia patients' anterior abdominal wall tissue (fascia transversalis and rectus sheath) is different from that of nonhernia patients. Collagen fibers are disorganized in hernia patients. It is possible that hernias are caused by disordered collagen fibers. Obesity and increased intra-abdominal pressure were found to be statistically significant predictors of collagen disorder and hernia development. Age has a statistically significant correlation with hernia development, indicating that age is a risk factor for hernia development. Although chronic cough has been associated with metabolic disorders such as diabetes, hypertension, and renal disease, we found no statistically significant association.

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Conflicts of interest

There are no conflicts of interest.

References

1. Mark AM, Michael JR. Hernias. In: Townsend CM, editor. Sabiston Textbook of Surgery. Gurgaon: Elsevier; 2016, p. 1092-3.
2. Beauchamp EM. Umbilical hernia. In: Townsend CM, editor. Sabiston Textbook of Surgery. Gurgaon: Elsevier; 2016, p. 1130-1.
3. Pans A, Albert A, Lapière CM, Nusgens B. Biochemical study of collagen in adult groin hernias. J Surg Res 2001;95:107-13.
4. Sangwan M, Sangwan V, Garg M, Mahendirutta P, Garg U. Abdominal wall hernia in a rural population in India—Is spectrum changing? Open J Epidemiol 2013;3:135-8.
5. Hosgor M, Karaca I, Ozer E, Suzek D, Ulukus C, Ozdamar A. Do alterations in collagen synthesis play an etiologic role in childhood inguinoscrotal pathologies: An immunohistochemical study. J Pediatr Surg 2004;39:1024-9.
6. Liem MS, van der Graaf Y, Zwart RC, Geurts I, van Vroonhoven TJ. Risk factors for inguinal hernia in women: A case-control study. The Coala Trial Group. Am J Epidemiol 1997;146:721-6.
7. Balamaddaiah G, Reddy SV. Prevalence and risk factors of inguinal hernia: A study in a semi-urban area in Rayalaseema, Andhra Pradesh, India. Int Surg J 2016;3:1310-3.
8. Ruhl CE, Everhart JE. Risk factors for inguinal hernia among adults in the US population. Am J Epidemiol 2007;165:1154-61.
9. Caglià P, Tracia A, Borzì L, Amodeo L, Tracia L, Veroux M, *et al.* Incisional hernia in the elderly: Risk factors and clinical considerations. Int J Surg 2014;12:164-9.
10. Öberg S, Andresen K, Rosenberg J. Etiology of inguinal hernias: A comprehensive review. Front Surg 2017;4:52.
11. Henriksen NA, Mortensen JH, Sorensen LT, Bay-Jensen AC, Ågren MS, Jorgensen LN, *et al.* The collagen turnover profile is altered in patients with inguinal and incisional hernia. Surgery 2015;157:312-21.

12. Wagner J, Herden U, Fischer L, Schild R, Vettorazzi E, Herrmann J, *et al*. Intravesical monitoring of intra-abdominal pressure after renal transplantation in children: A safety and feasibility study. Pediatr Transplant 2020;24:e13781.
13. Rodrigues Junior AJ, Rodrigues CJ, da Cunha AC, Jin Y. Quantitative analysis of collagen and elastic fibers in the transversalis fascia in direct and indirect inguinal hernia. Rev Hosp Clin Fac Med Sao Paulo 2002;57:265-70.
14. Manigrasso M, Anoldo P, Milone F, De Palma GD, Milone M. Case report of an uncommon case of drain-site hernia after colorectal surgery. Int J Surg Case Rep 2018;53:500-3.
15. Huang Q, Li CD, Yang YR. Role of the IL-33/ST2 axis in cigarette smoke induced airways remodelling in chronic obstructive pulmonary disease. Thorax 2021;76:743.
16. Mesejo A, Juan M, Serrano A. [Liver cirrhosis and encephalopathy: Clinical and metabolic consequences and nutritional support]. Nutr Hosp 2008;23:8-18.
17. McNulty M, Mahmud A, Spiers P, Feely J. Collagen type-I degradation is related to arterial stiffness in hypertensive and normotensive subjects. J Hum Hypertens 2006;20:867-73.
18. Lambert DM, Marceau S, Forse RA. Intra-abdominal pressure in the morbidly obese. Obes Surg 2005;15:1225-32.