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REVIEW

# Pathogenesis of the epigastric hernia

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**Abstract**
*Purpose* Epigastric herniation is a rather common condition with a reported prevalence up to 10 %. Only a minority is symptomatic, presumably the reason for the scarce literature on this subject. Epigastric hernias have specific characteristics for which several anatomical theories have been developed. Whether these descriptions of pathological mechanisms still hold with regard to the characteristics of epigastric hernia is the subject of this review.
*Methods* A multi-database research was performed to reveal relevant literature by free text word and subject headings 'epigastric hernia', 'linea alba', 'midline' and 'abdominal wall'. Reviewed were studies on anatomical theories describing the pathological mechanism of epigastric herniation, incidence, prevalence and female-to-male ratio and possible explanatory factors.
*Results* Three different theories have been described of which two have not been confirmed by other studies. The attachment of the diaphragm causing extra tension in the epigastric region is the one still standing. Around 1.6–3.6 % of all abdominal hernias and 0.5–5 % of all operated abdominal hernias is an epigastric hernia. Epigastric hernias are 2–3 times more common in men, with a higher incidence in patients from 20 to 50 years. Some cadaver studies show an epigastric hernia rate of 0.5–10 %. These specific features of the epigastric hernias (the large asymptomatic proportion, male predominance, only above umbilical level) are discussed with regard to the general theories.
*Conclusions* The epigastric hernia is a very common condition, mostly asymptomatic. Together with general factors for hernia formation, the theory of extra tension in the epigastric region by the diaphragm is the most likely theory of epigastric hernia formation.

**Keywords** Epigastric hernia · Pathophysiology · Aetiology

## Introduction

Epigastric hernias are the second most common type of linea alba abdominalis defects in adults [1, 2]. They occur from the xyphoid process to the umbilicus [3] and represent 1.6–3.6 % of all abdominal hernias and 0.5–5 % of all operated abdominal hernias [4].

Epigastric hernias are 2–3 times more common in men, with a higher incidence in patients from 20 to 50 years [3, 5]. Indeed, pathology studies report a prevalence of 0.5–10 %. These specific features of the epigastric hernias, such as the high proportion of asymptomatic occurrence, the male predominance and the fact that they only occur above umbilical level, cannot be explained by general theories on hernia formation alone. This collective review focussed on the pathophysiology of the epigastric hernia and its specific features. Another goal of this review is to condone a clear definition of an epigastric hernia.

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## Methods

A search was conducted to reveal relevant literature by free text word and subject headings 'epigastric hernia', 'linea alba', 'midline' and 'abdominal wall'. Databases used were Medline and Cochrane Library as well as books and guidelines. There was no date or language restriction. Studies were reviewed on anatomical theories describing the pathological mechanism of epigastric herniation, incidence, prevalence and female-to-male ratio and possible explanatory factors. Manuscripts were selected based on title and abstract. Two reviewers selected the reports independently, and search results were if necessary combined (JP/SN). Search was completed up to December 2011.

## Anatomy

The anterior abdominal wall aponeurosis consists of closely interwoven sheaths that pass freely from one side to the other side of the abdominal wall. Two types of these sheaths are present in the abdominal wall: the anterior rectus sheath and the posterior rectus sheath [6, 7].

The anterior rectus sheath forms the major and most conspicuous part of the anterior abdominal wall. This sheath contains three strata: the superficial stratum, the middle stratum and the deep stratum. The superficial stratum has tendinous fibres that run downward and laterally and, when followed, will come from the external oblique aponeurosis of the other side. In the middle stratum, the tendinous fibres run downward and medially at right angles to those of the superficial stratum; these are the tendinous fibres of the external oblique muscle of the same side. In the deep stratum, the tendinous fibres run upwards and medially, and these fibres originate from the anterior lamina of the internal oblique aponeurosis. Due to these anatomical structures, a triple criss-cross layer pattern is formed. The tendinous fibres in these three strata are bound together by loose tissue that facilitates their movement [6, 7].

In the posterior rectus sheath, a similar triple-layer pattern is seen. It is formed by the posterior lamina of the internal oblique aponeurosis together with two other strata, which originate from the transverses aponeurosis.

The triple-layer criss-cross pattern offers firmness to the texture of the aponeurosis in both the anterior and the posterior rectus sheaths and therefore makes them less liable for herniation. The tendinous fibres from all the strata of the anterior and posterior rectus sheaths decussate with the fibres from the opposite side. In the midline, these two decussating sheaths form the linea alba abdominalis. This crossing pattern can be seen on both the anterior and the posterior surfaces of the linea alba.

## Pathophysiology

### Pathological anatomy responsible for epigastric herniation, a theory by Askar (part I)

Askar suggested in 1978 that in 30–40 % of the general population, the fibres decussate once with the fibres of the opposite side in the abdominal wall. In the other 60–70 %, the fibres decussate two or three times. Askar assumes that epigastric herniation is found exclusively in patients with a single anterior and single posterior crossing pattern. This is probably a congenital factor predisposing to developing an epigastric hernia [1, 2, 6]. The three anatomical variants of the abdominal wall Askar describes are seen in Fig. 1. Askar states that epigastric herniation exclusively occurs in the first anatomical variation seen in Fig. 1 [6, 7].

### Evaluation of Askar's theory (part I)

Several studies tried to confirm part I of the theory described by Askar by taking biopsies from the linea alba. Despite the effort, the results from these studies could not confirm Askar's theory. For instance, Korenkov et al. investigated 93 cadavers and described that the fibres of the aponeurosis could not be allocated to any of the three anatomical variants Askar described [8]. These findings were confirmed by Axer et al. [9], who investigated 12 cadavers. To create more clarity, Korenkov et al. [8] presented a new classification system, which subdivided the

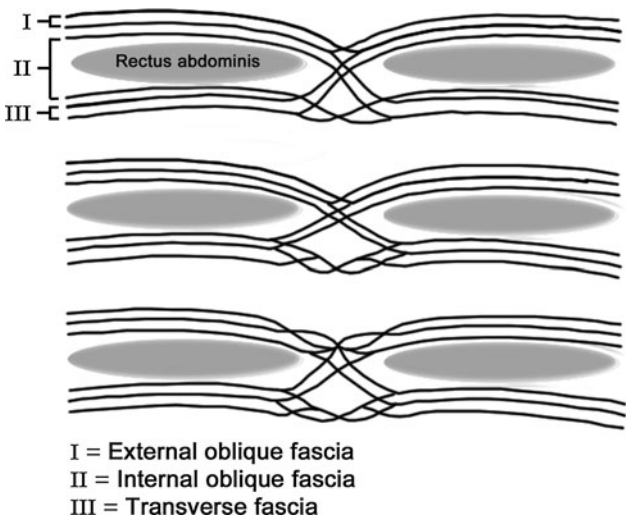


**Fig. 1** Different patterns of decussation at the linea alba abdominalis described by Askar

linea alba into a so-called weak type, an intermediate type and a compact type. However, they declared that there is not a clear cut-off point between these classifications. Therefore, the clinical relevance of this system is low.

Therefore, to recapitulate, both biopsy studies and post-mortem (or cadaver) studies do not confirm the first part of Askar's theory.

## Pathological anatomy responsible for epigastric herniation, a theory by Askar (part II)

Besides the lack of decussating in the linea alba, Askar suggests that another mechanism is responsible for the acquirement of an epigastric herniation. Originating from the sternocostal portion of the diaphragm, there are fine tendinous fibres. These fine fibres are viewed as the posterior aspect of the upper part of the midline aponeurosis. They descend vertically from under the costal margin to midway between the xiphoid and the umbilicus. This is where they curve medially towards the midline and the end by interlacing with the aponeurotic fibres in the posterior rectus sheath and with fibres in the middle tendinous intersection. Figure 2 shows these fibres from the diaphragm described by Askar [6, 7]. Askar supposes that an anatomic predisposition itself is not enough for the actual acquirement of an epigastric hernia.

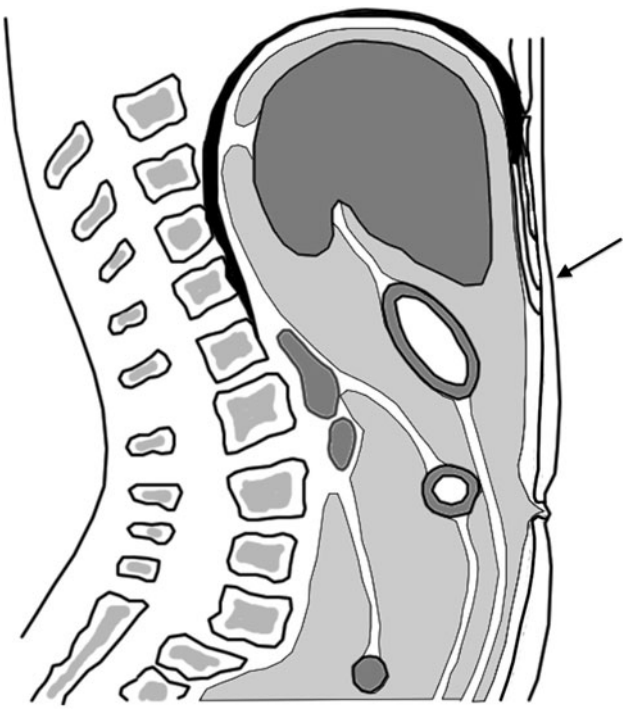

**Fig. 2** The arrow is showing the tendinous fibres from the diaphragm entering the linea alba abdominalis

Normally, the function of these tendinous slips, coming from the diaphragm, is to synchronize the movements of the diaphragm with those of the respiratory zone in the upper part of the anterior wall [6, 7]. They appear to be responsible for the occurrence of an actual epigastric hernia due to powerful contractions of the diaphragm, as occurs in a severe spasm caused by coughing or straining [6, 7]. In a case of powerful contraction, the tendinous slips would tighten in a harmful upward traction on the midline aponeurosis, mostly in the middle of the linea alba. Together with an outward traction on the aponeurosis by the middle tendinous insertion, caused by an accompanying forceful contraction of the rectus abdominis muscle, a force is created that is sufficient enough to tear a weak linea alba. The punctum maximum of this force is exactly in the middle between the xiphoid and the umbilicus, where epigastric hernias are usually seen.

## Evaluation of Askar's theory part II

The second part of Askar's theory is not strongly disputed in the current literature. The fact that all epigastric hernias occur in the midline and that the hernial orifice is transversely placed, either rhomboid or oval, strongly supports this suggestion [6].

However, the statement that the acquired herniation is due to a single strenuous event seems rather unlikely. Pathak et al. [10] show that it is more likely that the herniation is due to repeated strenuous exertion. Pathak et al. retrospectively investigated 133 patients with symptomatic abdominal wall hernias. In 89 % of the patients, a gradual onset of symptoms was reported. These patients could not link the abdominal wall hernia to a single strenuous event. Only a small group of 15 patients thought that the abdominal wall hernia occurred because of a single strenuous event, but this could not be confirmed by Pathak et al. For epigastric herniation, this is very difficult to investigate because many epigastric hernias present asymptomatic.

## Pathological anatomy responsible for epigastric herniation, a theory by Moschowitz

Moschowitz suggests that epigastric herniation is linked to the role of vascular lacunae at the linea alba of the anterior abdominal wall [11]. These vascular lacunae are formed when small blood vessels, which run between the transversalis fascia and the peritoneum, perforate the linea alba. As they pierce the transversalis fascia, a layer of fascial fibres is being pushed upwards and a space is created between the peritoneum and the fascia. During the periods of increased abdominal tension, parts of the pre-peritoneal fat derived from the falciform ligament are forced into the space along the blood vessels and pierce the aponeurosis.

Over a period of chronic and acute intermittent straining, the fascial defect enlarges and an epigastric hernia is formed. Only when the size of the hernia enlarges does the pre-peritoneal fat draw a pocket of peritoneum giving rise to a true hernial sac [4, 11]. Moschowitz theory is illustrated in Fig. 3.

### Evaluation of Moschowitz theory

The perforating vessel theory posed by Moschowitz has also been disputed. Primarily by Askar, who describes that idea that epigastric hernias start as a protrusion through a wide orifice for a blood vessel does not have a solid anatomic basis. On the other hand, Moschowitz reports two cases in which a perforating blood vessel was seen through the herniation [11]. Further support for this theory was also reported by Lang et al. [4] in a case report. The fact that these findings of a perforating vessel were only described in isolated cases makes this evidence not very strong. Indeed, these findings could be due to anatomic variations in rare cases.

The role of the falciform ligament reported by Moschowitz is unclear. Only a few case reports suggest that actual pre-peritoneal fat from the falciform ligament is herniated through the orifice in the linea alba [11–13]. In most cases, the hernia sac is empty or contains only a small portion of greater omentum [13]. However, it is possible that in rare cases the falciform ligament is partly herniated through the linea alba.

A summary of the pathophysiological theories thought to be responsible for epigastric herniation is seen in Table 1.

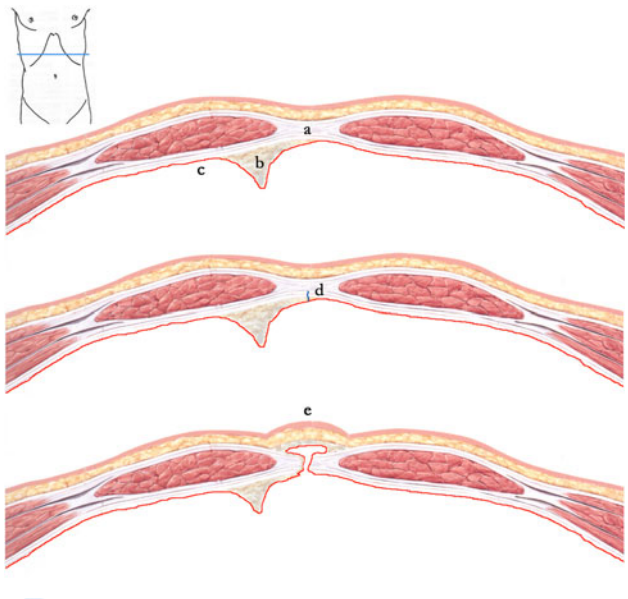


**Fig. 3** This figure was made with the use of the original picture of the abdominal wall from the prometheus anatomical atlas by Michael Schunke, Udo Schumacher and Erik Schulte 2010. *a* Linea alba, *b* falciform ligament, *c* peritoneum, *d* perforating vessel, *e* (true) epigastric hernia

### Microscopic studies

Recent microscopic anatomical studies show that not only the macroscopic anatomy of the linea alba is important in epigastric hernia pathology, but that the microscopic aspects should also be considered [14, 15]. Degeneration of collagen is associated with the requirement of several ventral hernias [14, 16]. In the abdominal wall, damaged collagen fibres are continuously repaired in the progress of proliferation and remodelling. The normal turnover of collagen is very slow, and it may take many years before the collagen is degraded and replaced [17]. Fachinelli et al. [14] showed that patients with ventral hernias have a significant less amount of total collagen in the abdominal wall compared to controls without ventral hernias. Especially the amount of type I collagen was about 20 % less than the amount of collagen in the controls.

In another study, Fachinelli et al. investigated the amount of elastin in patients with ventral hernias. Elastin gives tissues in the human body elasticity. Fachinelli et al. [15] reported that a significant higher amount of elastin is found in the abdominal wall in patients with ventral hernias. These findings suggest that not only anatomical diversity but also microscopic and histological factors plays a role in the acquirement of epigastric hernias.

It is likely that epigastric herniation is due to a tissue-destructive aspect, as suggested for all abdominal hernias by Cannon and Read. This is proved by higher blood levels of elastin-degrading activity and the lower level of proteinase inhibitors in patients with direct inguinal hernia [16, 17].

## Specific features of the epigastric hernia

### True or false hernias

In the literature the epigastric herniation is divided in a true and a false epigastric hernia. A false epigastric hernia consists of a protrusion of extraperitoneal fat through the linea alba without a peritoneal pouch. A true epigastric hernia consists not only of extraperitoneal fat but also of a protrusion of a peritoneal sac, with or without the presence of intestines or omentum [1, 2, 4]. Of course, patients who are operated in the past, by for instance laparoscopy, an incisional hernia have to be ruled out.

**Table 1** Condoned theories about epigastric herniation

| Theory | Origin | Described by | Years | Proofed theory | Current accepted theory |
|---|---|---|---|---|---|
| Single crossing | Congenital | Askar | 1978 | No | No |
| Forceful contraction | Acquired | Askar | 1978 | No, strong suggestion | Yes |
| Perforating vessel | Congenital | Moschowitz | 1917 | Yes, by case reports | Yes, in some rare cases |

## Incidence and prevalence

Epigastric hernias are the second most common type of linea alba abdominis defects in adults [1, 2] and occur mostly in patients from 20 to 50 years [3, 5]. They represent 1.6–3.6 % of all abdominal hernias and 0.5–5 % of all abdominal hernias operated [4]. Approximately 3–5 % of the population suffers from an epigastric hernia. From 1985 to 2008, the incidence of epigastric herniation increased significantly [18].

Due to the fact that an epigastric hernia is mostly a subclinical condition, post-mortem studies show a higher incidence than could be expected from the observed rate in the general population of 0.5–10 % [1]. Therefore, it is difficult to determine the exact amount of people suffering from this condition.

Some authors suggest that epigastric hernias are two to three times more common in men [3, 5]. This is contradicted in a recent study, which shows that this female–male distribution is probably outdated, although a rather small patient group was used [18]. In this study, the authors explain the levelling of gender occurrence by the emancipation of women, which leads to an increased amount of heavy physical work for women. The predisposition to other risk factors, such as smoking, for epigastric herniation is probably more equally distributed between gender over the years. Considering ethnicity, other authors studied the prevalence of external and epigastric hernias in African communities in Ghana. They found a 0.8–1 female-to-male ratio in patients with epigastric hernias, which confirms the equal distribution among gender in African patients [19]. Another study even found a higher incidence of epigastric herniation in Nigerian women [20]. This could be due to the fact that in some African countries heavy physical work is performed by women. Therefore, the higher incidence found in women in these African countries strengthens the theory of the epigastric hernia being an acquired lesion.

**Table 2** Epidemiological features of the epigastric hernia

| | |
|---|---|
| Incidence/prevalence (%) | 3–5 |
| Age of onset (years) | 20–50 |
| Female–male distribution (ratio) | 3:1 (probably outdated) |
| Subclinical presence | Majority of cases |

A summary of specific features of epigastric hernias is seen in Table 2.

## Infantile epigastric hernias

Some authors report infantile epigastric hernias being a rare condition or make no reporting of the condition at all [1, 5]. This is contradicted by Coats et al. [21], who found in a retrospective cohort of 40 patients under 18 years that epigastric hernias are responsible for 4 % of the infantile hernias.

The gender distribution in infantile epigastric hernias seems different compared to the distribution in adult life [1, 2]. Coast et al. [21] found a female-to-male ratio in infantile epigastric hernia of 5–3. Nearly half of the reported hernias were asymptomatic, and in 30 % of the investigated patients, the epigastric hernia was present at the time of birth. This implies that at least some infantile epigastric hernias have a congenital origin, while others might be acquired, especially in older children [21].

Although some authors report that elective repair of these abdominal wall defects appears justified, some series report that surgical intervention for infantile epigastric hernias is unnecessary [1, 21–23]. In Nyhus and Condon's Hernia, it is reported that it is unnecessary because infantile epigastric hernias resolve spontaneously, similar to infantile umbilical hernias. Therefore, surgical intervention should always be based on the age of the child and the severity of symptoms [1].

## Factors associated with epigastric herniation

There are some risk factors associated with epigastric herniation, such as uncoordinated, vigorous, synchronous contractions of the diaphragm-like extensive coughing and heavy training and lifting. It is thought that these increase the intra-abdominal pressure and therefore might result in a protrusion of pre-peritoneal fat or even peritoneum through the linea alba [1]. These conditions are associated with lung diseases and occupations in which heavy physical work is required, like athletes and soldiers [1, 2].

Epigastric herniation is also more common in patients with overweight or obesity [1, 10]. Several studies report that in obese patients, the recurrence rate after hernia repair, which also includes epigastric hernias repair, is

**Table 3** Probable and possible associated factors for epigastric herniation

| Probable associated factors | Possible associated factors |
|---|---|
| Extensive physical training | Smoking |
| Extensive coughing (lung diseases) | Chronic corticosteroid use |
| Overweight or obesity | Diabetes |
| | Old age |
| | Male gender |

higher. This is also probably due to the increased abdominal pressure in these patients, which results in more pressure on the linea alba abdominalis and/or the suture line after repair [1, 24, 25].

Similar to inguinal hernias, smoking increases the recurrence after repair of the epigastric hernia [27]. Biopsies of the abdominal wall in smokers show histological changes in collagen thickness [17]. Smoking increases the oxidative stress and thereby activates the neutrophil function. These neutrophils secrete potent tissue-destructive enzymes, such as collagenases and elastase. These processes make the collagen in the abdominal wall thinner and thus weaken the abdominal wall [1, 17, 26]. Despite this, currently there is no evidence suggesting that smoking is directly associated with a first epigastric herniation. Therefore, further research is needed to unravel the mechanism between smoking and epigastric herniation.

Another author poses that a risk factor for the acquirement of a hernia is chronic corticosteroid use. Studies found this to be associated with incisional hernias, but this is not proven for epigastric herniation [28]. However, chronic corticosteroid use weakens the abdominal wall and could therefore attribute to the development of epigastric hernias. Further studies are necessary to determine whether there is a causal link [14]. The same has to be done for risk factors reported for other hernias like diabetes, old age and male gender [16]. A summary of probable and possible factors associated with epigastric hernias is seen in Table 3.

## Conclusion

Different theories about epigastric herniation are described in this review. The oldest epigastric formation theory is the perforating vessel theory. This theory is still accepted and proved by some case reports. It is unlikely that the perforating vessel theory explains all epigastric hernias, because of the fact that during operative treatment, a perforating vessel is a rare phenomenon.

Secondly, the single crossing theory was deposited. As mentioned earlier, this theory was never proven in cadaver studies studying the linea alba. The single crossing theory is therefore not a commonly accepted theory in current times.

In the same time in history as the single crossing theory, the forceful contraction theory was described. This theory is also not proved, but there are strong suggestions that this theory contributes to the formation of an epigastric hernia.

In current times, other explanations for the formation of epigastric hernias are given. These are more general theories, such as the presence of less type I collagen, more elastin and higher intra-abdominal pressure. These explanations have their own accompanying risk factors like smoking, extensive physical training, extensive coughing as in some chronic lung diseases and obesity.

During the review of the literature on this subject, there was no clear definition of an epigastric hernia or the difference between a para-umbilical hernia and an epigastric hernia. Authors suggest that an epigastric hernia is a protrusion of extra peritoneal fat, with or without a peritoneal pouch, between the decussating fibres or through a perforating vessel lacunae of the linea alba abdominalis, occurring from the xyphoid to the umbilical ring, without the orifice in the linea alba being connected to the umbilical ring.

This review shows that the female–male distribution, with a higher prevalence in men, of the epigastric hernia is probably outdated. It seems that women catch up with men in the occurrence of risk factors, especially with smoking and obesity. It can well be that in the future the female–male distribution is equal. To the current knowledge, there is no anatomical substrate for the differences in prevalence between man and women.

More research is necessary for the epigastric hernia in children. It could well be that congenital infantile hernias are formed during labour. This is because of the great abdominal pressure the young abdominal wall has to endure during this process.

The limitations of our study merit some considerations. Firstly, this review has a collective study design. The groups are rather heterogenic, and therefore, it is difficult to determine explicit risk factors. Secondly, during the search, authors noticed that little research is done on the repair of solely epigastric hernias. Several studies describe repair for all ventral hernias or for epigastric hernias combined with umbilical or para-umbilical hernias. Therefore, patients' baseline characteristics and risk factor cannot be extracted and used for epigastric hernia alone. Further research for the repair and baseline characteristics is necessary to determine more possible risk factors. Of course in clinical practice, this could be hard because it is difficult to differentiate between a proximal para-umbilical hernia and an epigastric hernia. To increase knowledge concerning this topic, one could retrospectively follow patients with a peroperative proven epigastric herniation.

In conclusion, this review gives a clear view on the pathological aspects of the acquirement of an epigastric hernia. Knowledge of the pathology is essential to improve surgery and patient care towards a more specific approach for epigastric hernias.

**Conflict of interest** The authors declare that they have no conflict of interest.

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