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FOR PUBLICATION OF PAPER ENTITLED:
**ETIOLOGICAL FACTORS LEADING TO PURULENT
MEDIASITIS**

In volume 18 of World Bulletin of Public Health (WBPH),
January, 2023



Impact
Factor:
7.635



ISSN (E):
2749-3644



Sharof
Editor In-Chief





ETIOLOGICAL FACTORS LEADING TO PURULENT MEDIASTINITIS

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Article history:	Abstract:
Received: November 24 th 2022 Accepted: December 26 th 2022 Published: January 30 th 2023	This review article provides information on the causes of primary mediastinitis. Back in the 19th century, surgeons identified two ways of infection penetration into the mediastinum: from the esophagus during its perforation and from the cellular spaces of the neck. This corresponds to the division of mediastinitis into primary and secondary. Primary purulent mediastinitis is considered if it develops as a primary purulent complication of diseases and injuries of the mediastinal organs. Secondary purulent mediastinitis develops with contact or metastatic spread to the mediastinum of the purulent process, in the presence of a primary purulent focus outside the mediastinal tissue. From a clinical point of view, it seems appropriate to allocate a special group - postoperative mediastinitis. It is concluded that the relevance of primary mediastinitis is preserved and knowledge in this field of surgery is still in demand.

Keywords: purulent mediastinitis, esophageal damage, computed tomography in mediastinitis, surgical infection, thoracic surgery.

Of course, according to the mechanism of occurrence, mediastinitis, which develop as complications of various surgical interventions, can be both primary and secondary. The course and clinical manifestations of the purulent process in the mediastinum in operated patients have specifics that are not noted with other variants of purulent mediastinitis. [41]

Primary purulent lesion of the mediastinal fiber suggests exogenous infection. The most common cause of primary purulent mediastinitis is perforation of hollow organs located in the mediastinum - the esophagus and upper respiratory tract, and the former are observed much more often than the second. [14]

Esophageal ruptures are the "classic" cause of purulent mediastinitis. [12]

Without aiming at analyzing the classifications of esophageal perforations proposed in the literature, the number of which is quite large, to systematize further presentation, we will adhere to the division of the main groups of causes leading to a violation of the integrity of the esophageal wall.

The ratio between different types of damage to the esophagus underwent significant changes during

the twentieth century, primarily due to the widespread use of endoscopic methods for the study of the gastrointestinal tract and operations on the organs of the breast.

The ratio of these reasons in the largest modern statistics is quite stable. [11] Traumatic perforations of the esophagus account for 42-48%, [38] iatrogenic 30-40%, [16] caused by diseases of 4-14% of cases. [29] Of the total number of injuries, the cervical region accounts for 39.1%, thoracic - 56.6%, abdominal - 4.3%. [20]

A sufficiently large number of injuries can lead to damage to the esophagus, which can be divided in the direction of exposure to the traumatic agent - from the inside (foreign bodies, chemical and thermal burns, hydraulic and pneumatic shocks) and from the outside (wounds and closed trauma to the chest and neck, fracture of the cervical spine). [17]

The frequency of esophageal perforations from the total number of patients complaining of the presence of a foreign body in the esophagus is 0.9 - 1.6%. [40] Among all the causes of esophageal perforations, damage by foreign bodies ranges from 16 to 48%. [44] Most often, these lesions are localized in

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the cervical region and in the zone of the first anatomical narrowing. [41]

The most common traumatic agent is bones (more often fish, less often poultry and even less often meat). [3] Among other foreign bodies that cause damage to the esophagus, sewing needles [42], coins, buttons and cufflinks, fruit pits are mentioned [30], a case of perforation of the esophagus in a child with corn chips is described. [50] Damage by large foreign bodies (bones, dentures) is more common in adults in a state of intoxication. [30]

The mechanism of perforation of the esophagus when a foreign body enters may be different. Perforation of the esophagus may be due to the direct effect of a foreign body on the wall of the esophagus. Another option encountered is damage due to improper or inept medical care taken to remove the foreign body. [2]

Small sharp foreign bodies, especially those located transversely, cause spastic contractions of the esophagus, which leads to perforation of the type of puncture wound. Double-edged bodies can cause dissection of the walls when moving along the esophagus. [36]

Large sharp foreign bodies directly tear or pierce the wall of the esophagus. Such injuries are usually multiple, small in size and accompanied by a large number of abrasions and hemorrhages. [28]

In time, an unremoved foreign body causes a pressure sore of the esophageal wall (necrotic esophagitis) with the subsequent formation of a perforation hole. This situation, in particular, is possible in persons with a scar-narrowed esophagus due to a chemical burn, when large unchewed pieces of soft food act as a foreign body - more often meat ("meat blockage"). With the slow development of perforation, some delimitation of the purulent focus is possible due to demarcation processes in the surrounding tissues. [7]

Injuries of a non-through nature lead to the occurrence of intramural abscesses, which can open both into the lumen of the esophagus and into the mediastinum, and lead to the development of esophageal phlegmon. [10]

The frequency of development of purulent mediastinitis with perforations by foreign bodies is up to 80%. [1] The rate of development and features of the course of purulent mediastinitis, which develops as a result of perforation of the esophagus by a foreign body, depend on the nature and mechanism of damage, the degree of contamination of a foreign body, pre-existing changes in the paraesophageal tissue, but there are no absolute regularities here and it seems to us that it is ideologically incorrect to distinguish "more" and "less" dangerous perforations. [39]

In addition, in the literature there are descriptions of both a long stay in the paraesophageal tissue of foreign bodies introduced from the lumen of the esophagus, without the development of abscesses, and the development of abscesses around these foreign body's months and even years after the primary injury. [46]

Perforations of the esophagus with chemical burns due to chemical necrosis of the wall are possible both in the acute stage and in the stage of rejection of necrotic masses. Most cases of the first kind are fatal due to the extreme severity of the condition of patients, due to both the resorptive effect of cauterizing poison, and the rapidly developing mediastinitis and other complications. [6]

With the development of perforation in the stage of rejection of necrotic masses, the prognosis is more favorable, although it remains serious, since cicatricial-inflammatory changes in the paraesophageal tissue have time to occur, because of which perforation, as a rule, occurs in the pleural cavity, and common mediastinitis does not develop. [8]

The so-called hydraulic breaks occur due to an acute increase in pressure on the walls of the pathologically altered esophagus, when trying to "push" a stuck piece of food by taking a large amount of liquid. Usually they are large and are located in the thoracic or abdominal regions. [13]

The mechanism of occurrence of pneumatic ruptures of the esophagus is a direct blow of a jet of compressed air or gas with careless (and sometimes just hooliganism) handling of production compressors, cylinders with compressed gases, etc., as well as with an idle shot in the mouth. [45]

"Mixed" hydropneumatics shock is possible when opening with teeth or near the open mouth containers with strongly carbonated liquids - wine, beer, soft drinks. With pneumatic and hydropneumatic ruptures, the posterior pharyngeal wall and the initial part of the esophagus are damaged. [30]

With all these mechanisms of damage to the esophagus, the rapid development of a common purulent process in the mediastinal fiber is inevitable.

The frequency of damage to the esophagus in neck wounds, according to various authors, ranges from 2 to 10.8%, with chest wounds - less than 1 percent. Among all mechanical damage to the esophagus, the share of injuries accounts for no more than 5%. Often, damage to the esophagus with wounds is combined with damage to other vital anatomical formations - the trachea, larynx, thyroid gland, large vessels, which very often leads to the death of victims at the site of injury. The highest probability of damage to the esophagus with an explosive injury. [26]



Since most of the wounded undergo immediate surgical treatment, mediastinitis usually does not have time to develop.

Cases of damage to the esophagus with a closed chest and neck injury are even more rare than with wounds and are also practically not isolated. In patients admitted to hospitals with closed thoracic and thoracoabdominal trauma, they are 0.3%; among those killed from these injuries - 0.45%, and among all mechanical damage to the esophagus in the largest of the statistics - 0.56%. A rupture of the esophagus with an unstable closed fracture of the III-IV thoracic vertebrae is described. [24]

The likelihood of developing common mediastinitis with rupture of the esophagus due to a closed injury is very high. [32]

The most common and most misunderstood cause so far remains spontaneous rupture of the esophagus - a disease known in the same way as Burhave syndrome, barogenous rupture of the esophagus, esophageal apoplexy, etc. More than 80% of cases of Burhave syndrome are observed in men over 50 years of age, although the disease occurs at any age, even in infants. [19]

Cases of esophageal rupture in the early postoperative period after interventions on the abdominal organs are described. A certain importance is attached to the previous changes in the esophagus - peptic esophagitis, cardiospasm, hernia of the esophageal opening of the diaphragm, etc. As the main etiological factor of spontaneous perforation of the esophagus, a sharp increase in intra-abdominal, and then intraesophageal pressure, due, as a rule, to vomiting, is usually considered. Therefore, a significant number of cases of Burhave syndrome develop in connection with the state of alcohol intoxication. [34]

Among other producing factors are weightlifting, defecation, childbirth, irritable cough, asthmatic status. Some authors emphasize the morphological differences between Burhave and Mallory-Weiss syndromes, since if the mucous membrane is initially damaged during the latter, then with a spontaneous rupture of the esophagus, the integrity of the muscular layer is initially violated, others, on the contrary, consider these conditions to be different forms of one emetogenic syndrome. Spontaneous rupture of the esophagus most often occurs in the lower third, although again there are cases of ruptures of all departments. [11, 18, 49]

The significant size of the esophageal defect with spontaneous rupture leads to a rapid progression of mediastinitis, accompanied by a lethality of 80%. [31]

Among the diseases of the esophagus, a violation of the integrity of its walls is observed in

tumors, chronic peptic and acute (including medicinal) ulcers, diverticula, syphilis, tuberculosis. [23, 27, 43]

A rare condition is the perforation of the artificial esophagus, which is based on two factors - peptic, because of reflux of gastric contents into the graft, and ischemic - due to atherosclerosis of the vessels that feed the graft in elderly patients, or mechanical trauma to the antenthoracally conducted intestine. [4]

Violation of the integrity of the esophagus is also possible with the pathology of other organs of the neck and mediastinum. So, cases of breakthrough into the esophagus of abscesses of the lungs and pleura and destruction of the esophageal wall by a lung tumor are described. [15] There are reports of esophageal perforations of a dissecting aortic aneurysm. [33] There are descriptions of esophageal perforations as complications of strumitis and thyroiditis, tuberculous mediastinal lymphadenitis, as well as a consequence of the breakdown of syphilitic gums of the mediastinum. [21]

Except for extremely rare perforations of acute ulcers, damage to the esophageal wall in all the above diseases occurs slowly, gradually, accompanied by a pronounced inflammatory-demarcating reaction of the surrounding tissues, because of which common forms of purulent mediastinitis are practically not observed, more likely the occurrence of esophageal-respiratory and esophageal-pleural fistulas.

Instrumental effects, as before, are one of the main causes of esophageal perforations, in some statistics they account for more than 50% of all mechanical damage. [37]

At the beginning of the twentieth century, the frequency of damage to the esophagus during instrumental studies and manipulations reached 8-10%, and when using some types of tools (Graef coin extractor, Weiss fish bone extractor, etc.) was so great that it forced to completely abandon their use. [9,16]

Unfortunately, even now instrumental damage to the esophagus is observed in various diagnostic studies (all types of endoscopic examinations of the upper gastrointestinal tract, bronchoscopy, mediastinoscopy), therapeutic manipulations (intubation of the trachea, cardiodilation, bougienation of the esophagus, establishment of various probes and stents, etc.), as well as with a long stay in the esophagus of probes and stents. [9,35]

Instrumental lesions of a healthy esophagus are large, linear in shape and localized more often in the cervical and upper thoracic regions. Instrumental injuries of the cicatricially altered esophagus are usually small in size and are located in the thoracic region, mainly below strictures. There are double perforations



of the esophagus, both at one and at different levels. [35]

Damage to the esophagus during esophagoscopy is noted when using both rigid and flexible devices of all designs, generations, and firms. [2,9]

The frequency of esophageal damage when performing esophagoscopy with a rigid apparatus, the range of application of which is currently mainly limited to the removal of foreign bodies of the esophagus, is estimated at 0.22 - 0.51%. At the same time, it is sometimes impossible to determine what was directly the traumatic agent - the device itself, the foreign body or the effect was combined. It is now recognized that rigid esophagoscopy should be performed under general anesthesia with the use of muscle relaxants and artificial ventilation, especially when removing wedged foreign bodies and with a cicatricial-narrowed esophagus. [1,2,9,16]

Circumstances that increase the likelihood of perforation of the esophagus during endoscopic examination are as follows:

- associated with the patient - wide piriformis sinuses of the pharynx, low tone of the muscles of the esophagus (more often observed in the elderly, especially in women), restless behavior, pronounced gag reflex, arthritic changes in the cervical and thoracic vertebrae with the presence of osteophytes on the front surface, the presence of diffuse or nodular goiter.

- associated with the doctor performing the study - inadequate choice and performance of anesthesia, fatigue, haste, insufficient experience; related to the equipment - poor visibility, beveled optics, long controlled part (in fiber devices of old designs).

The uppermost segment of the posterior wall of the cervical region is most often damaged, which is associated with elements of violence during the passage of the lower pharyngeal constrictor, directly under which in the zone of the pharyngeal-esophageal transition there is a zone of weakness of the muscular membrane of the posterior wall (the place of formation of Zenker diverticula). In the thoracic region, which is damaged 3 to 5 times less often than the cervical region, the right wall suffers more often. [3, 5, 22, 46,50]

Biopsy of the esophageal mucosa during esophagoscopy with conventional biopsy forceps - the manipulation is relatively safe, perforations with it occur extremely rarely - with excessive capture of the inflammatory-altered mucosa (esophagitis) at the border of the visual field. In contrast, puncture biopsy of the esophagus through the unchanged mucosa is associated with a high probability of perforation and should not be used. [21,35]

End-to-end damage to the esophagus during therapeutic esophagoscopy is described - sclerotherapy for esophageal varicose veins, endoscopic polypectomy with diathermocoagulation, endoscopic electrosurgical dissection of the cicatricial stricture of the esophagus. [7,41,47]

In 40 - 85% of observations, esophageal ruptures are not diagnosed during endoscopic examination or immediately after it. [1,2,9,16]

The size of the esophageal defect when the esophagus is perforated with an endoscope usually corresponds to or exceeds the diameter of the apparatus. Such injuries are characterized by the formation of a false course and a fairly rapid development of mediastinitis and / or other complications (if the false course penetrates into the pleural or abdominal cavity). [16]

Cases of esophageal damage are described as a complication of diagnostic mediastinoscopies and transcollar diagnostic mediastinotomy. [9] A typical site of damage in such cases is the left tracheobronchial angle, where the esophagus is located directly behind the left main bronchus. [24] In the literature there are casuistic descriptions of perforation of the esophagus during drainage of the pleural cavity for empyema [39] and rupture of the esophagus with intussusception and prolapse of the stomach into the right pleural cavity during laparoscopy. [24]

Iatrogenic ruptures are noted as complications of bougienation of esophageal strictures of various etiologies and strictures of esophageal anastomoses after various surgical interventions. Some methods, in particular "blind bougienation", are now almost completely abandoned due to frequent perforations. The use of the techniques of bougiening on a conductor string and retrograde bougienation per thread made it possible to reduce the number of perforations, but not to eliminate them completely. [1]

Changes in the paraesophageal tissue, developing because of a chemical burn of the esophagus, in the case of mechanical damage on the one hand, contribute to the delimitation of the purulent process, on the other hand, lead to a high (up to 45%) frequency of mediastinopleural and tracheoesophageal fistulas. [41]

Iatrogenic damage to the esophagus probes is rare. The cause of rupture, as a rule, is a rough and hasty probe to empty the stomach in conditions of pathological (coma) or drug (action of muscle relaxants) esophageal atony. Perforations with a probe are described with the subsequent development of purulent mediastinitis of the artificial esophagus.

In the past, when used for prolonged nasogastric intubation of rubber probes, there were cases of perforation of the esophagus by the type of



pressure sore. Focal necrotic changes in the mucosa are noted already on the 3-4th day of the stay of the rubber probe in the esophagus. When using silicone probes, such complications are not described.

More often there are perforations caused by balloon probes used for closed hypothermia of the stomach, stopping bleeding from varicose dilated veins of the esophagus, intraesophageal balloon manometry, transesophageal echocardiography. The mechanisms of rupture of the esophagus by balloon probes are different - too rapid inflation of the balloon, erroneous inflation of the gastric balloon of the probe when in the esophagus, pressure sore of the esophageal wall with a prolonged stay of the balloon in a swollen state, the patient's attempt to independently remove the probe with the inflated balloon. [41]

Ruptures of the upper segment of the esophagus and the laryngeal part of the pharynx in the region of the pear-shaped sinuses during intubation of the trachea are observed with a frequency of 0.01 - 0.006%. A number of circumstances can contribute to the occurrence of this complication. Among them are the circumstances associated with patients: a short neck, long teeth, osteochondrosis of the cervical spine; and circumstances related to the peculiarities of intubation: hasty manipulations in cardiac arrest, asphyxia, regurgitation of gastric contents, aspiration of vomit, rough manipulations of an inexperienced specialist.

More often, the damaging agent is the blade of the laryngoscope, less often - the distal ends of the endotracheal tube or conductor.

As a rule, unnoticed at the time of injury under general anesthesia, these injuries are usually diagnosed late, with the inevitable development of mediastinitis.

Quite often, damage to the esophagus occurs with various methods of palliative bougienage, recanalization and endoprosthetics for esophageal tumors.

Damage to the esophagus in the distant period after endoprosthetics. occur when the prosthesis is located at an angle to the axis of the esophagus and the size of the socket of the tube does not correspond to the diameter of the unaffected esophagus. Most often, perforations are noted when the tumor is localized in the middle thoracic region, due to the constant compression of the tumor between the aortic arch, the prosthesis, and the spine.

The frequency of perforations during the installation of stents reaches 7%, and the formation of esophageal-tracheal fistulas with a prolonged stay of the stent in the lumen of the esophagus is 39%, which makes it necessary to give preference to other methods of palliative aids, in particular laser recanalization. [50]

Damage to the esophagus in the treatment of achalasia of cardia by cardiodilation was observed when using all types of cardiac dilators. The ruptures were caused by exceeding the permissible pressure in the cylinders (360 mm Hg) and the rate of increase in pressure, and when using the Stark dilator - a high rate of expansion of its branches. Esophageal ruptures during cardiodilation were noted in 2%. The probability of developing mediastinitis with such injuries is close to 100%, the mortality rate is more than 80%. Prolonged stay of grafts after anterior corporodesis of the cervical spine.

The frequency and rate of development of purulent mediastinitis in iatrogenic lesions of the esophagus depends on the size of the defect, the contamination of the esophagus and the wounding object, the severity of the pre-existing changes in the paraesophageal tissue and, finally, the timing of diagnosis and the beginning of adequate treatment.

The development of purulent-fibrinous inflammatory changes in the mediastinal fiber with an injury to the esophagus occurs on average after 6-8 hours. Since the defect is usually significant in instrumental lesions, purulent mediastinitis develops faster and more often than with other types of damage. With damage to the esophagus under anesthesia, the duration of the stage of serous inflammation, on the contrary, increases slightly.

It is the development of purulent mediastinitis that determines the course and prognosis of esophageal damage. The average mortality rate for primary perforative mediastinitis reaches 30 - 40%.

Ruptures of the upper respiratory tract as a cause of the development of purulent mediastinitis are quite rare. Very often, this damage leads to the rapid death of the victim due to gross violations of external respiration. In cases of timely and successful provision of medical care, the frequency of infectious complications is significantly lower compared to perforations of the esophagus due to lower contamination of the cellular spaces. Cases of mediastinitis development due to rupture of the trachea during traumatic intubation, prolonged artificial ventilation of the lungs with excessively high pressure in the tamponing cuff, the use of intubation tubes of inadequate size are described.

Mediastinitis can develop after cold and gunshot wounds to the neck and mediastinum without damaging internal organs. In blind contaminated wounds, when there was a "direct and constantly renewed infection of the mediastinal fiber", on the contrary, diffuse rapidly progressive purulent mediastinitis developed, the outcome of which, as a rule, was fatal. [26]



The development of mediastinitis after a closed chest injury without damage to internal organs - due to supuration of the hematoma - is most often observed after fractures of the sternum. There is even a description of the development of purulent mediastinitis after an iatrogenic fracture of the sternum, which arose during successful resuscitation measures.

A rare cause of the development of primary purulent mediastinitis is infection of the mediastinum, which occurs due to extravasal migration of central venous catheters. Such cases are described during chemotherapy or prolonged parenteral nutrition.

Thus, as can be seen from the presented review, the causes of purulent mediastinitis are diverse. Along with household traumatic injuries of the esophagus, the frequency of iatrogenic factors that lead to the development of such a terrible complication remains impressive. This, in turn, on the one hand, indicates a high probability of occurrence of purulent mediastinitis in the clinical practice of doctors and the need to take timely measures to prevent its development, on the other.

Conflict of Interest – No

Ethical aspect - the article is of an overview nature and the information provided has a cited reference to primary sources.

Funding is not.

BIBLIOGRAPHY:

1. Abdulrahman H, Ajaj A, Shunni A, El-Menyar A, Chaikhouni A, Al-Thani H, Latifi R. Blunt traumatic esophageal injury: unusual presentation and approach. *Int J Surg Case Rep.* 2014;5(1):16-8. doi: 10.1016/j.ijscr.2013.10.015. Epub 2013 Nov 14. PMID: 24394856; PMCID: PMC3907204.
2. Abdulrahman H., Ajaj A., Shunni A., et al. Blunt traumatic esophageal injury: unusual presentation and approach. *Int. J. Surg. Case Rep.* 2014;5(1):16–18. - PMC – PubMed.
3. Aiolfi A., Inaba K., Recinos G., et al. Non-iatrogenic esophageal injury: a retrospective analysis from the National Trauma Data Bank. *World J. Emerg. Surg.* 2017;12:19. - PMC – PubMed.
4. Akman C., Kantarci F., Cetinkaya S. Imaging in mediastinitis: a systematic review based on aetiology. *Clin. Radiol.* 2004;59(7):573–585. – PubMed.
5. Alisher O.Okhunov, Kakhramon X.Boboev, Azizbek F.Valijonov, & Shirina A. Valijonova. (2022). Principles of diagnosis and treatment of acute purulent-destructive lung diseases. *World Bulletin of Public Health*, 7, 1-2. Retrieved from <https://scholarexpress.net/index.php/wbph/article/view/526>.
6. Athanassiadi KA. Infections of the mediastinum. *Thorac Surg Clin.* 2009 Feb;19(1):37-45, vi. doi: 10.1016/j.thorsurg.2008.09.012. PMID: 19288819.
7. Babaiarova ShU, Okhunov AO, Komarin AS. [Activity of the NO-system in lung after pneumectomy of various volumes]. *Patol Fiziol Eksp Ter.* 2012 Jan-Mar;(1):29-32. Russian. PMID: 22629857.
8. Biancari F., Saarnio J., Mennander A., et al. Outcome of patients with esophageal perforations: a multicenter study. *World J. Surg.* 2014;38(4):902–909. – PubMed.
9. Biffi W.L., Moore E.E., Feliciano D.V., et al. Western trauma association critical decisions in trauma: diagnosis and management of esophageal injuries. *J. Trauma Acute Care Surg.* 2015;79(6):1089–1095. – PubMed.
10. Bobokulova, Sh A., and A. O. Okhunov. "Acute purulent-destructive lung diseases as consequences of endotheliitis after COVID-19." *Journal Of Education and Scientific Medicine* 2.3 (2022): 56-61.
11. Bohanes T, Neoral C. Akutní mediastinitida [Acute mediastinitis]. *Rozhl Chir.* 2011 Nov;90(11):604-11. Czech. PMID: 22442869.
12. Bryant A.S., Cerfolio R.J. Esophageal trauma. *Thorac. Surg. Clin.* 2007;17(1):63–72. – PubMed.
13. Cedeño A, Echeverría K, Vázquez J, Delgado A, Rodríguez-Ortiz P. Intrathoracic esophageal rupture distal to the carina after blunt chest trauma: Case-report. *Int J Surg Case Rep.* 2015;16:184-6. doi: 10.1016/j.ijscr.2015.08.021. Epub 2015 Sep 3. PMID: 26492358; PMCID: PMC4643336.
14. Cross M.R., Greenwald M.F., Dahhan A. Esophageal perforation and acute bacterial mediastinitis: other causes of chest pain that can be easily missed. *Medicine.* 2015;94(32) (Baltimore) - PMC – PubMed.
15. De Freitas RP, Fahy CP, Brooker DS, Primrose WJ, McManus KG, McGuigan JA, Hughes SJ. Descending necrotising mediastinitis: a safe treatment algorithm. *Eur Arch Otorhinolaryngol.* 2007 Feb;264(2):181-7. doi: 10.1007/s00405-006-0174-z. Epub 2006 Sep 29. PMID: 17009018.
16. Dickinson K.J., Blackmon S.H. Endoscopic techniques for the management of esophageal perforation. *Oper. Tech. Thorac. Cardiovasc. Surg.* 2015;20(3):251–278.
17. Dubose J.J., Scalea T.M., O’Conner J.V. In: *Trauma*. 9th ed. Feliciano D.V., Mattox K.L., Moore E.E., editors. McGraw Hill; New York:



2021. Trachea, bronchi, and esophagus; pp. 589–598.
18. Džian A, Stiegler P, Smolár M, Hamzik J, Mistuna D. Posterior mediastinotomy as an unordinary method of mediastinal drainage in patient with descending necrotizing mediastinitis: a case report. *Thorac Cardiovasc Surg.* 2013 Mar;61(2):175-7. doi: 10.1055/s-0031-1295576. Epub 2012 Jan 3. PMID: 22215493.
19. Garrana SH, Buckley JR, Rosado-de-Christenson ML, Martínez-Jiménez S, Muñoz P, Borsa JJ. Multimodality Imaging of Focal and Diffuse Fibrosing Mediastinitis. *Radiographics.* 2019 May-Jun;39(3):651-667. doi: 10.1148/rg.2019180143. Epub 2019 Apr 5. PMID: 30951437.
20. Heath BJ, Bagnato VJ. Poststernotomy mediastinitis treated by omental transfer without postoperative irrigation or drainage. *J Thorac Cardiovasc Surg.* 1987 Sep;94(3):355-60. PMID: 3626597.
21. Jarboui S, Jerraya H, Moussi A, Ben Moussa M, Marrakchi M, Kaffel N, Haouet K, Ferjaoui M, Zaouche A. Médiastinite nécrosante descendante odontogénique [Descending necrotizing mediastinitis of odontogenic origin]. *Tunis Med.* 2009 Nov;87(11):770-5. French. PMID: 20209836.
22. Kanlerd A, Mahawongkajit P, Achavanuntakul C, Boonyasatid P, Auksornchart K. Successful management of 72-h delay-detected blunt esophageal injury with trans-gastric primary repair; a case report and literature review. *Trauma Case Rep.* 2023 Jan 6;43:100755. doi: 10.1016/j.tcr.2023.100755. PMID: 36654763; PMCID: PMC9841267.
23. Karimov KhIa, Babadzhonov BD, Okhunov AO, Atakov SS, Kasymov UK, Ibragimov NK, Mukhitdinov UM, Rikhsibekov SN, Rakhmatov AN, Kutlimuratov Kh. Khirurgicheskie aspekty nerespiratornoï deiatel'nosti legkikh pri ikh ostrykh gnoïno-destruktivnykh zabollevaniïakh [Surgical aspects of non-respiratory activity of the lungs during acute pyonecrotizing diseases]. *Lik Sprava.* 2004 Jan-Feb;(1):38-40. Russian. PMID: 17051711.
24. Karnath B, Siddiqi A. Acute mediastinal widening. *South Med J.* 2002 Oct;95(10):1222-5. PMID: 12425517.
25. Kim J.J., Han J.W. Delayed diagnosis of thoracic esophageal rupture due to blunt abdominal trauma without chest trauma: a case report. *J. Cardiothorac. Surg.* 2022;17(1):228. - PMC - PubMed.
26. Kircheva DY, Vigneswaran WT. Successful primary repair of late diagnosed spontaneous esophageal rupture: A case report. *Int J Surg Case Rep.* 2017;35:49-52. doi: 10.1016/j.ijscr.2017.03.038. Epub 2017 Apr 1. PMID: 28437673; PMCID: PMC5403789.
27. Kluge J. Die akute und chronische Mediastinitis [Acute and chronic mediastinitis]. *Chirurg.* 2016 Jun;87(6):469-77. German. doi: 10.1007/s00104-016-0172-7. PMID: 27138268.
28. Lin J, Jimenez CA. Acute mediastinitis, mediastinal granuloma, and chronic fibrosing mediastinitis: A review. *Semin Diagn Pathol.* 2022 Mar;39(2):113-119. doi: 10.1053/j.semmp.2021.06.008. Epub 2021 Jun 15. PMID: 34176697.
29. Lin J., Jimenez C.A. Acute mediastinitis, mediastinal granuloma, and chronic fibrosing mediastinitis: a review. *Semin. Diagn. Pathol.* 2022;39(2):113–119. – PubMed.
30. Lin YY, Hsu CW, Chu SJ, Chen SC, Tsai SH. Rapidly propagating descending necrotizing mediastinitis as a consequence of intravenous drug use. *Am J Med Sci.* 2007 Dec;334(6):499-502. doi: 10.1097/MAJ.0b013e3180a5e911. PMID: 18091375.
31. Marty-Ane CH, Alauzen M, Alric P, Serres-Cousine O, Mary H. Descending necrotizing mediastinitis. Advantage of mediastinal drainage with thoracotomy. *J Thorac Cardiovasc Surg.* 1994 Jan;107(1):55-61. PMID: 8283919.
32. Marupov, I., Bobokulova, S., Okhunov, A. ., Abdurakhmanov, F., Boboev, K. ., Korikhonov, D. ., Yakubov, I. ., Yarkulov, A. ., Khamdamov, S. ., & Razzakov, S. . (2023). How does lipid peroxidation affect the development of pneumosclerosis: experimental justification. *Journal Of Education and Scientific Medicine,* 1(1), 2-7. Retrieved from <https://journals.tma.uz/index.php/jesm/article/view/368>.
33. Mihos P, Potaris K, Gakidis I, Papadakis D, Rallis G. Management of descending necrotizing mediastinitis. *J Oral Maxillofac Surg.* 2004 Aug;62(8):966-72. doi: 10.1016/j.joms.2003.08.039. PMID: 15278861.
34. Novakov IP, Safev GP, Peicheva SE. Descending necrotizing mediastinitis of odontogenic origin—personal experience and literature review. *Folia Med (Plovdiv).* 2010 Jul-Sep;52(3):13-20. doi: 10.2478/v10153-010-0002-5. PMID: 21053669.
35. Okhunov AO, Babadzhonov BD, Kasymov UK, Atakov SS, Ibragimov NK, Rikhsibekov SN,



- Rakhmatov AN, Mukhitdinov UM. Sovremennye printsipy antibakterial'noï terapii gnoïno-septicheskikh zabollevaniï [Modern principals of antibacterial therapy of suppurative-septic diseases]. *Lik Sprava*. 2003 Oct-Nov;(7):70-3. Russian. PMID: 14723141.
36. Okhunov AO, Kasymov AK. [Some pathogenic aspects of changes in non-respiratory function of the lungs in sepsis]. *Lik Sprava*. 2006 Oct-Nov;(7):45-7. Russian. PMID: 17312887.
37. Omura T., Asieri M., Bischof K., et al. Primary repair of a delayed presentation thoracic oesophageal gunshot injury: a report of two cases. *Trauma Case Rep*. 2017;12:45–47. - PMC – PubMed.
38. Petrone P., Kassimi K., Jimenez-Gomez M., et al. Management of esophageal injuries secondary to trauma. *Injury*. 2017;48(8):1735–1742. – PubMed.
39. Port J.L., Kent M.S., Korst R.J., et al. Thoracic esophageal perforations: a decade of experience. *Ann. Thorac. Surg*. 2003;75(4):1071–1074. – PubMed.
40. Puerta Vicente A., Priego Jiménez P., Cornejo López M.Á., et al. Management of esophageal perforation: 28-year experience in a major referral center. *Am. Surg*. 2018;84(5):684–689. – PubMed.
41. Randjelović T, Stamenković D. Mediastinitis--dijagnostika i lečenje [Mediastinitis--diagnosis and therapy]. *Acta Chir Iugosl*. 2001;48(3):55-9. Croatian. PMID: 11889988.
42. Roh J.Y., Kim I., Eom J.S., et al. Successful stenting for bronchial stenosis resulting from blunt airway trauma. *Intern. Med*. 2018;57(22):3277–3280. - PMC – PubMed.
43. Sancho LM, Minamoto H, Fernandez A, Sennes LU, Jatene FB. Descending necrotizing mediastinitis: a retrospective surgical experience. *Eur J Cardiothorac Surg*. 1999 Aug;16(2):200-5. doi: 10.1016/s1010-7940(99)00168-2. PMID: 10485421.
44. Schraufnagel DP, Mubashir M, Raymond DP. Non-iatrogenic esophageal trauma: a narrative review. *Mediastinum*. 2022 Sep 25;6:23. doi: 10.21037/med-21-41. PMID: 36164360; PMCID: PMC9385875.
45. Torba M, Baumbach SF, Gjata A, Buci S, Faber E, Subashi K. Verletzungen der Speiseröhre nach stumpfem Thoraxtrauma. Eine Fallpräsentation und Literaturübersicht [Esophageal injury following blunt thoracic trauma. A case report and review of the literature]. *Unfallchirurg*. 2012 Dec;115(12):1123-5. German. doi: 10.1007/s00113-012-2256-2. PMID: 23052701.
46. Tripp HF, Paape KL, St Martin WH. Descending necrotizing mediastinitis. *J La State Med Soc*. 2002 Nov-Dec;154(6):319-21. PMID: 12518725.
47. Turcanu L, Tănase D, Oțetea-Stemper G, Vilics D. Mediastinite la copil [Mediastinitis in children]. *Rev Pediatr Obstet Ginecol Pediatr*. 1989 Oct-Dec;38(4):361-7. Romanian. PMID: 2518582.
48. Vural FS, Girdwood RW, Patel AR, Zigiriadis E. Descending mediastinitis. *Asian Cardiovasc Thorac Ann*. 2012 Jun;20(3):304-7. doi: 10.1177/0218492311434088. PMID: 22718719.
49. Wiesemann S, Schmid S, Haager B, Passlick B. Mediastinitis: Klinik und Behandlungsoptionen [Mediastinitis: Clinical Presentation and Therapy]. *Zentralbl Chir*. 2015 Oct;140 Suppl 1:S8-15. German. doi: 10.1055/s-0035-1557779. Epub 2015 Sep 9. PMID: 26351767.
50. Yajima K, Neyatani H, Takahashi T. [Descending necrotizing mediastinitis resulting from acute epiglottitis; report of a case]. *Kyobu Geka*. 2014 Aug;67(9):860-3. Japanese. PMID: 25135420.