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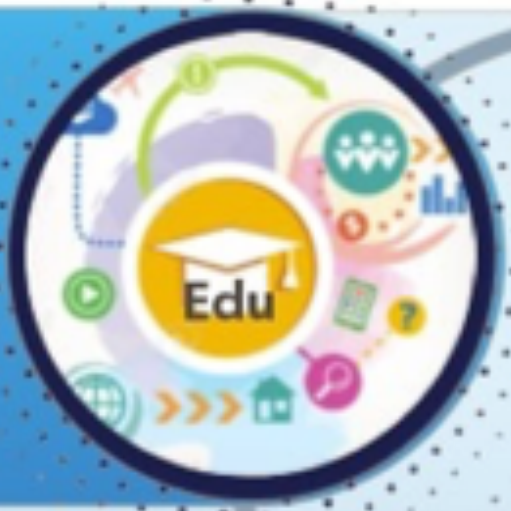

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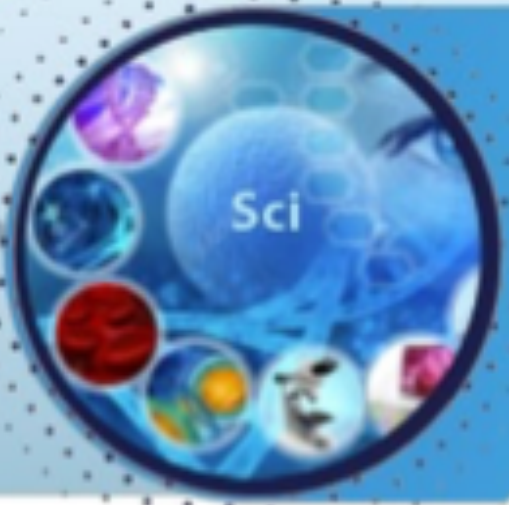

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Review Article

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Primary Purulent Mediastinitis

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ABSTRACT

This review article provides information on the causes of primary mediastinitis. Back in the 19th century, surgeons identified two ways for infection to penetrate the mediastinum: from the esophagus when it is perforated and from the cellular spaces of the neck. This corresponds to the division of mediastinitis into primary and secondary. Purulent mediastinitis is considered primary 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 of the purulent process to the mediastinum, in the presence of a primary purulent focus outside the mediastinal tissue. From a clinical point of view, it seems appropriate to single out postoperative mediastinitis in a special group. It is concluded that the relevance of primary mediastinitis remains and knowledge in this field of surgery is still in demand.

Of course, according to the mechanism of occurrence, mediastinitis, which develops as a complication 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 a specificity that is not noted in other variants of purulent mediastinitis. [41]

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

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

Without aiming at the analysis of the classifications of esophageal perforations proposed in the literature, the number of which is quite large, for a systematic 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 relationship between different types of esophageal injuries has changed significantly during the twentieth century, primarily due to the widespread use of endoscopic methods of examination of the gastrointestinal tract and breast surgery.

The ratio of these reasons in the largest modern statistics is quite stable. [11] Traumatic esophageal perforations account for 42-48%, [38] iatrogenic 30-40%, [16] disease-related 4-14% of cases. [29] Of the total number of injuries, the cervical spine accounted for 39.1%, the thoracic spine for 56.6%, and the abdominal spine for 4.3%. [20]

A fairly large number of injuries can lead to damage to the esophagus, which can be divided according to the direction of the impact of the traumatic agent - from the

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inside (foreign bodies, chemical and thermal burns, hydraulic and pneumatic shocks) and from the outside (wounds and closed trauma of the chest and neck, fracture of the cervical spine). [17]

The incidence of esophageal perforations from the total number of patients who complained of the presence of a foreign body in the esophagus is 0.9 - 1.6%. [40] Among all the causes of esophageal perforations, foreign body damage accounts for 16 to 48%. [44] Most often, these injuries are located in the cervical spine and the area of the first anatomical narrowing. [41]

The most common traumatic agent is bones (more often fish, less often poultry, and even more rarely meat). [3] Other foreign bodies that cause damage to the esophagus include sewing needles [42], coins, buttons and cufflinks, fruit pits [30], and a case of perforation of the esophagus in a child with corn chips. [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, can be different. Esophageal perforation can be caused by the direct impact of a foreign body on the wall of the esophagus. Another variant encountered is injury due to improper or inept medical care undertaken to remove the foreign body. [2]

Small, sharp foreign bodies, especially those located transversely, cause spastic contractions of the esophagus, leading to perforation such as a puncture wound. Double-edged bodies can cause the walls to dissect as they move through the esophagus. [36]

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

A foreign body that is not removed in time causes pressure ulcers in the esophageal wall (necrotizing esophagitis) with the subsequent formation of a perforation hole. Such a situation, in particular, is possible in persons with a cicatricial-narrowed esophagus due to a chemical burn, when large unchewed pieces of soft food, most often meat ("meat blockage"), act as a foreign body. With the slow development of perforation, some delimitation of the purulent focus is possible due to demarcation processes in the surrounding tissues. [7]

Non-perforating injuries lead to intramural abscesses, which can open both into the lumen of the esophagus and mediastinum and also lead to the development of esophageal phlegmon. [10]

The incidence of purulent mediastinitis with perforations by foreign bodies is up to 80%. [1] The rate of development and peculiarities of the course of purulent mediastinitis, which develops as a result of perforation of the esophagus by a foreign body, depending on the nature and mechanism of damage, the degree of contamination of the foreign body, and 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, there are descriptions in the literature of both the long-term stay in the periesophageal tissue of foreign bodies that have penetrated from the lumen of the esophagus, without the development of abscesses and the development of abscesses around these foreign bodies for months and even years after the initial injury. [46]

Perforations of the esophagus in 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 type are fatal due to the extreme severity of the patient's condition, due to both the resorptive effect of the cauterizing poison and the rapidly developing mediastinitis and other complications. [6]

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

The so-called hydraulic ruptures occur as a result of 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. They are usually large and located in the thoracic or abdominal regions. [13]

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

A "mixed" hydropneumatic shock is possible when opening containers with highly carbonated liquids - wine, beer, soft drinks - with the teeth or near the open mouth. Pneumatic and hydropneumatic ruptures damage

the posterior pharyngeal wall and the initial part of the esophagus. [30]

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

According to various authors, the incidence of esophageal injury in neck injuries ranges from 2 to 10.8 percent, and in chest injuries, it is less than 1 percent. Injuries account for no more than 5% of all mechanical injuries to the esophagus. Often, injuries to the esophagus during wounds are combined with injuries to other vital anatomical formations - trachea, larynx, thyroid gland, and large vessels, which very often leads to the death of victims at the site of injury. The most likely chance of damage to the esophagus is due to blast trauma. [26]

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

Cases of esophageal injury in closed chest and neck trauma are even rarer than in wounds and are also rarely isolated. In patients admitted to hospitals with closed thoracic and thoracoabdominal trauma, they are 0.3%; Among those who died from these injuries - 0.45%, and among all mechanical injuries of the esophagus in the largest of the statistics - 0.56%. A rupture of the esophagus in an unstable closed fracture of the III-IV thoracic vertebrae is described. [24]

The likelihood of developing disseminated mediastinitis when the esophagus ruptures due to a closed injury are very high. [32]

The most common and misunderstood cause is spontaneous rupture of the esophagus, a disease also known as Boerhaave syndrome, barogenic rupture of the esophagus, esophageal apoplexy, etc. More than 80% of cases of Boerhaave syndrome are seen 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 have been described. A certain importance is attached to previous changes in the esophagus - peptic esophagitis, cardiospasm, hiatal hernia, etc. As the main etiological factor of spontaneous esophageal perforation, a sharp increase in intra-abdominal followed by intraesophageal pressure, usually caused by vomiting, is usually considered. Therefore, a significant number of cases of Boerhaave syndrome develop in connection with the state of alcohol intoxication. [34]

Other producing factors include heavy lifting, defecation, childbirth, tearful cough, and status asthmaticus. Some authors emphasize the morphological differences between the Boerhaave and Mallory-Weiss syndromes, since if the latter initially damages the mucous membrane, then with a spontaneous rupture of the esophagus, the integrity of the muscle layer is initially violated, others, on the contrary, consider these conditions to be different forms of the same emetogenic syndrome. Spontaneous rupture of the esophagus most often occurs in the lower third, although again cases of rupture of all compartments have been noted. [11, 18, 49]

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

Among the diseases of the esophagus, violation of the integrity of its walls is observed in tumors, chronic peptic and acute (including drug) ulcers, diverticula, syphilis, and tuberculosis. [23, 27, 43]

The rarest condition is the perforation of the artificial esophagus, which is based on two factors - peptic, as a consequence of reflux of gastric contents into the graft, and ischemic - due to atherosclerosis of the vessels feeding the graft in elderly patients, or mechanical trauma of the intrathoracic intestine. [4]

Violation of the integrity of the esophagus is also possible with the pathology of other organs of the neck and mediastinum. Thus, cases of lung abscesses and pleura breaking through into the esophagus and destruction of the esophageal wall by a lung tumor have been described. [15] There are reports of esophageal perforations of aortic dissecting aneurysms. [33] Esophageal perforations have been described as complications of strumitis and thyroiditis, tuberculous mediastinal lymphadenitis, and as a consequence of the disintegration of syphilitic gums mediastinum. [21]

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

Instrumental impacts, 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 incidence of esophageal damage during instrumental exami-

nations and manipulations reached 8-10%, and with the use of some types of instruments (Graefe's coin extractor, Weiss fish bone extractor, etc.) it was so high that it forced to completely abandon their use. [9,16]

Unfortunately, instrumental injuries of the esophagus are still observed during various diagnostic examinations (all types of endoscopic examinations of the upper gastrointestinal tract, bronchoscopy, mediastinoscopy), therapeutic manipulations (tracheal intubation, cardio dilatation, esophageal bougienage, insertion of various probes and stents, etc.), as well as during prolonged stay of probes and stents in the esophagus. [9,35]

Instrumental injuries of a healthy esophagus are large, linear in shape, and localized more often in the cervical and upper thoracic regions. Instrumental lesions of the cicatricial esophagus are usually small and located in the thoracic region, predominantly below the strictures. There are double perforations of the esophagus, both at the same and at different levels. [35]

Injuries to the esophagus during esophagoscopy were noted with the use of both rigid and flexible appliances of all designs, generations and companies. [2,9]

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

Circumstances that increase the likelihood of esophageal perforation on endoscopic examination are as follows:

- Patient-related - wide pear-shaped sinuses of the pharynx, low esophageal muscle tone (more often observed in the elderly, especially in women), restless behavior, pronounced gag reflex, arthritic changes of the cervical and thoracic vertebrae with the presence of osteophytes on the anterior surface, the presence of diffuse or nodular goitre;

- related to the doctor performing the examination - inadequate selection and performance of anesthesia, fatigue, haste, insufficient experience; related to the equipment - poor visibility, bevelled optics, long steerable part (in fiber devices of old designs).

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

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

Penetrating injuries of the esophagus during therapeutic esophagoscopy - sclerotherapy for esophageal varicose veins, endoscopic polypectomy with diathermocoagulation, endoscopic electrosurgical dissection of cicatricial stricture of the esophagus - are described. [7,41,47]

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

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

Cases of esophageal injury have been described as a complication of diagnostic mediastinoscopy and transcollar diagnostic mediastinotomy. [9] The typical site of injury 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 esophageal perforation during pleural drainage due to empyema [39] and esophageal rupture with intussusception and prolapse of the stomach into the right pleural cavity during laparoscopy. [24]

Iatrogenic ruptures are noted as complications of bougienage strictures of various etiologies and strictures of esophageal anastomoses after various surgical interventions. Some techniques, in particular blind bougienage, are now almost completely abandoned due to fre-

quent perforations. The use of bougienage techniques on the conductor string and retrograde bougienage per thread made it possible to reduce the number of perforations, but not eliminate them. [1]

Changes in the paraesophageal tissue, which develop as a result 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 fairly high (up to 45%) incidence of mediastinopheural and tracheoesophageal fistulas. [41]

Iatrogenic lesions of the esophagus are rare and rare. As a rule, the cause of rupture is a rough and hasty insertion of a tube to empty the stomach in conditions of pathological (coma) or drug (action of muscle relaxants) atony of the esophagus. Probe perforations with subsequent development of purulent mediastinitis of the artificial esophagus have been described.

In the past, when rubber tubes were used for long-term nasogastric intubation, there have been cases of esophageal perforation in the form of pressure ulcers. Focal necrotic changes in the mucosa are noted as early as 3-4 days after the rubber probe in the esophagus. With the use of silicone probes, no such complications have been described.

Perforations caused by balloon probes used for closed gastric hypothermia, stopping bleeding from esophageal varicose veins, intraesophageal balloon manometry, and transesophageal echocardiography are more common. 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 it is in the esophagus, pressure ulcer of the esophageal wall when the balloon stays in an inflated state for a long time, the patient's attempt to remove the probe with the inflated balloon on his own. [41]

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

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

As a rule, these lesions, which are not noticed at the time of injury under general anesthesia, are usually diagnosed late, with the inevitable development of mediastinitis.

Esophageal injuries occur quite often with various methods of palliative bougienage, recanalization and endoprosthetic for esophageal tumors.

Esophageal injuries in the remote period after endoprosthetics. They occur when the prosthesis is located at an angle to the axis of the esophagus and the size of the tube bell does not match 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 incidence of perforations during stent placement 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 care, in particular, laser recanalization. [50]

Esophageal injuries in the treatment of achalasia cardia by cardio dilation have been observed with the use of all types of cardio dilators. Ruptures were caused by exceeding the permissible pressure in the cylinders (360 mm Hg) and the rate of pressure increase, and when using a Stark dilator, a high rate of expansion of its jaws. Esophageal ruptures during cardio dilatation were noted in 2%. The probability of developing mediastinitis with such injuries is close to 100%, the mortality rate is more than 80%. Long-term retention of grafts after anterior cervical spine corrodesis.

The frequency and speed of the development of purulent mediastinitis in iatrogenic injuries of the esophagus depend on the size of the defect, contamination of the esophagus and the wounding object, the severity of preexisting changes in the periesophageal tissue, and, finally, the timing of diagnosis and the start of adequate treatment.

The development of purulent-fibrinous inflammatory changes in mediastinal tissue in case of esophageal injury occurs in an average of 6-8 hours. Since the defect is usually significant in instrumental injuries, purulent mediastinitis develops faster and more often than in other types of injuries. On the contrary, when the esophagus is damaged under anesthesia, the duration of the serous inflammation stage is slightly increased.

It is the development of purulent mediastinitis that determines the course and prognosis of esophageal dam-

age. The average mortality rate for primary perforative mediastinitis reaches 30 - 40%.

Ruptures of the upper respiratory tract as a cause of purulent mediastinitis are quite rare. Very often, this injury leads to the rapid death of the victim due to gross disorders of external respiration. In cases of timely and successful medical care, the incidence of infectious complications is significantly lower compared to esophageal perforations due to lower contamination of the cellular spaces. Cases of mediastinitis due to tracheal rupture during traumatic intubation, long-term mechanical ventilation with excessively high pressure in the packing cuff, and the use of inadequate intubation tubes have been described.

Mediastinitis can develop after cold and gunshot wounds to the neck and mediastinum without damage to internal organs. In blind contaminated wounds, when there was an "immediate and constantly recurring infection of the mediastinal tissue", 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 suppuration of the hematoma - is most often observed after sternum fractures. There is even a description of the development of purulent mediastinitis after an iatrogenic fracture of the sternum, which occurred during successful resuscitation measures.

A rare cause of primary purulent mediastinitis is mediastinal infection due to the extravasal migration of central venous catheters. Such cases have been described during chemotherapy or long-term parenteral nutrition.

Thus, as can be seen from the presented review, the causes of purulent mediastinitis are diverse. Along with domestic traumatic injuries of the esophagus, the frequency of iatrogenic factors leading to the development of such a formidable 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 hand.

Conflict of Interest – None

Ethical aspect – the article is of a review nature and the information presented has a cited reference to primary sources.

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REFERENCE:

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.

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.

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.

4. Akman C., Kantarci F., Cetinkaya S. Imaging in mediastinitis: a systematic review based on aetiology. Clin. Radiol. 2004;59(7):573–585.

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.

7. Babaiarova ShU, Okhunov AO, Komarin AS. [Activity of the NO-system in the lung after pneumonectomy of various volumes]. Patol Fiziol Eksp Ter. 2012 Jan-Mar;(1):29-32.

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.

9. Biffl 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.

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.

12. Bryant A.S., Cerfolio R.J. Esophageal trauma. Thorac. Surg. Clin. 2007;17(1):63–72.

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.

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).

15. De Freitas RP, Fahy CP, Brooker DS, Primrose WJ, McManus KG, McGuigan JA, Hughes SJ. Descend-

ing necrotising mediastinitis: a safe treatment algorithm. Eur Arch Otorhinolaryngol. 2007 Feb;264(2):181-7.

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. Dzian A, Stiegler P, Smolár M, Hamzik J, Mistuna D. Posterior mediastinotomy as an unordinary method of mediastinal drainage in a patient with descending necrotizing mediastinitis: a case report. Thorac Cardiovasc Surg. 2013 Mar;61(2):175-7.

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.

20. Heath BJ, Bagnato VJ. Poststernotomy mediastinitis is treated by omental transfer without postoperative irrigation or drainage. J Thorac Cardiovasc Surg. 1987 Sep;94(3):355-60.

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.

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.

23. Karimov KhIa, Babadzhanov BD, Okhunov AO, Atakov SS, Kasymov UK, Ibragimov NK, Mukhitdinov UM, Rikhsibekov SN, Rakhmatov AN, Kutlimuratov Kh. Khirurgicheskie aspekty nerespiratornoĭ deiatel'nosti legkikh pri ikh ostrykh gnoĭno-destruktivnykh zabolevaniiakh [Surgical aspects of non-respiratory activity of the lungs during acute pyonecrotizing diseases]. Lik Sprava. 2004 Jan-Feb;(1):38-40.

24. Karnath B, Siddiqi A. Acute mediastinal widening. South Med J. 2002 Oct;95(10):1222-5.

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.

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.

29. Lin J., Jimenez C.A. Acute mediastinitis, mediastinal granuloma, and chronic fibrosing mediastinitis: a review. Semin. Diagn. Pathol. 2022;39(2):113–119.

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.

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.

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.

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.

35. Okhunov AO, Babadzhanov BD, Kasymov UK, Atakov SS, Ibragimov NK, Rikhsibekov SN, Rakhmatov AN, Mukhitdinov UM. Sovremennye printsipy antibakterial'noĭ terapii gnoĭno-septicheskikh zabolevaniĭ [Modern principals of antibacterial therapy of suppurative-septic diseases]. Lik Sprava. 2003 Oct-Nov;(7):70-3.

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.

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.

38. Petrone P., Kassimi K., Jimenez-Gomez M., et al. Management of esophageal injuries secondary to trauma. Injury. 2017;48(8):1735–1742.

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.

40. Puerta Vicente A., Priego Jiménez P., Cornejo López M.Á., et al. Management of esophageal perforation: 28-year experience in a major referral centre. Am. Surg. 2018;84(5):684–689.

41. Randjelović T, Stamenković D. Medijastinitis-dijagnostika i lecenje [Mediastinitis--diagnosis and therapy]. Acta Chir Iugosl. 2001;48(3):55-9.

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.

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.

44. Schraufnagel DP, Mubashir M, Raymond DP. Non-iatrogenic esophageal trauma: a narrative review. Mediastinum. 2022 Sep 25;6:23.

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.

46. Tripp HF, Paape KL, St Martin WH. Descending necrotizing mediastinitis. J La State Med Soc. 2002 Nov-Dec;154(6):319-21.

47. Turcanu L, Tănase D, Oţetea-Stemper G, Vilics D. Mediastinite la copil [Mediastinitis in children]. Rev Pediatr Obstet Ginecol Pediatr. 1989 Oct-Dec;38(4):361-7.

48. Vural FS, Girdwood RW, Patel AR, Zigiriadis E. Descending mediastinitis. Asian Cardiovasc Thorac Ann. 2012 Jun;20(3):304-7.

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.

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.

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BIRLAMCHI YIRINGLI MEDIASTINIT

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ABSTRAKT

Ushbu ko'rib chiqish maqolasida birlamchi mediastinit sabablari haqida ma'lumot berilgan. XIX asrda xirurglar infektsiyaning mediastinumga kirishining ikkita usulini aniqladilar: u teshilganda o'chirishdan va bo'yinning uyali bo'sh joylaridan. Bu mediastinitning birlamchi va ikkilamchi bo'linishiga to'g'ri keladi. Yiringli mediastinit, agar u kasalliklarning asosiy yiringli asoratlari va mediastinal organlarning shikastlanishi sifatida rivojlanadigan bo'lsa, birlamchi hisoblanadi. Ikkilamchi yiringli mediastinit vositachilik to'qimasidan tashqarida birlamchi yiringli fokus mavjud bo'lganda, yiringli jarayonning mediastinumga kontakt yoki metastatik tarqalishi bilan rivojlanadi. Klinik nuqtai nazardan, maxsus guruhda postoperativ mediastinitni tanlab olish maqsadga muvofiq ko'rinadi. Xulosa qilish mumkinki, birlamchi mediastinitning dolzarbligi qolmoqda va bu jarrohlik sohasidagi bilimlar hali ham talabga ega.

Tayanch so'zlar: yiringli mediastinit, ezofagaal shikastlanish, mediastinit uchun xisoblangan tomografiya, jarrohlik infektsiyasi, torakal jarrohlik.

ПЕРВИЧНЫЙ ГНОЙНЫЙ МЕДИАСТИНИТ

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АБСТРАКТ

В данной обзорной статье приводятся сведения относительно причин возникновения первичных медиастинитов. Еще в 19 веке хирурги выделяли два пути проникновения инфекции в средостение: из пищевода при его перфорации и из клетчаточных пространств шеи. Это соответствует подразделению медиастинитов на первичные и вторичные. Первичным гнойный медиастинит считается в том случае, если он развивается как первичное гнойное осложнение заболеваний и травм органов средостения. Вторичный гнойный медиастинит развивается при контактном или метастатическом распространении на средостение гнойного процесса, при наличии первичного гнойного очага вне медиастинальной клетчатки. С клинической точки зрения представляется целесообразным выделить в особую группу - послеоперационные медиастиниты. Делается заключение о том, что актуальность первичных медиастинитов сохраняется и знания в данной области хирургии все еще востребованы.

Ключевые слова: гнойный медиастинит, повреждение пищевода, компьютерная томография при медиастините, хирургическая инфекция, торакальная хирургия.