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Odontogenic Putrefactive-Necrotic Phlegmon Complicated by Mediastinitis and Sepsis in a 4-Year-Old Child: A Case Report

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Abstract. The aim of this case report was to describe the clinical course, diagnostic findings, staged surgical management, intensive-care treatment, and postoperative recovery of a 4-year-old child with odontogenic putrefactive-necrotic phlegmon complicated by mediastinitis and sepsis. The patient was admitted to the surgical department of the Municipal Children's Clinical Hospital No. 3, Bishkek, Kyrgyz Republic, on 23 October 2025, with complaints of pain and swelling of the soft tissues of the left submandibular region, limited mouth opening, difficulty swallowing, and an increase in body temperature up to 38°C. The source of infection was the destroyed teeth 74 and 75 of the lower jaw on the left side. Upon admission, leukocytosis of 14.27×10^9 per litre was detected, a left shift of neutrophils up to 75.82%, and an increase in the erythrocyte sedimentation rate up to 35 millimetres per hour. Primary incision and drainage of the phlegmon of the submandibular space with evacuation of 30–40 millilitres of pus provided temporary improvement. By 27 October, the condition sharply deteriorated with the spread of the process to the deep fascial spaces of the neck and the mediastinum; the procalcitonin level reached 0.47 nanograms per millilitre, supporting the diagnosis of sepsis in the context of clinical deterioration and organ dysfunction. An emergency cervical mediastinotomy was performed with evacuation of 100–150 millilitres of pus from the pretracheal, paraoesophageal, and prevertebral spaces. Comprehensive treatment included antibacterial therapy with ceftriaxone and metronidazole, detoxification therapy, and intensive monitoring in the intensive care unit. Positive dynamics with wound cleansing and the formation of granulations allowed for the placement of secondary sutures on 5 November, followed by successful healing and discharge of the patient. The results of the observation emphasise the importance of early diagnosis, multi-stage surgical debridement, and comprehensive intensive therapy in widespread odontogenic infections in children. The presented clinical case may assist paediatric maxillofacial

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surgeons, paediatricians, and intensive care physicians in recognising clinical progression and the need for timely escalation of treatment in complicated odontogenic infections.

Keywords: odontogenic infection, descending mediastinitis, sepsis, paediatric patient, cervical mediastinotomy.

Odotogeninė puvimo nekrotinė flegmona 4 metų vaikui, kuri komplikavosi mediastinitu ir sepsiu: atvejo aprašymas

Santrauka. Pateikiame klinikinio atvejo aprašymą – 4 metų vaiko, sergančio odontogenine puvimo nekrotine flegmona, kuri komplikavosi mediastinitu ir sepsiu, klinikinę eigą, diagnostinius duomenis, etapais vykdytą chirurginį gydymą, intensyviąją terapiją ir sėkmingą pooperacinį atsigavimą. 2025 m. spalio 23 d. vaikas buvo paguldytas į Kirgizijos Respublikos Biškeko 3-iosios savivaldybės vaikų klinikinės ligoninės chirurgijos skyrių, skundėsi skausmu, jo kairės apatinio žandikaulio srities minkštieji audiniai buvo patinę, negalėjo praverti burnos, buvo sunku ryti. Kūno temperatūra iki 38 °C. Infekcijos šaltinis buvo suirę kairės pusės apatinio žandikaulio 74 ir 75 krūminiai dantys.Priėmimo metu buvo nustatyta leukocitozė – $14{,}27\times10^9$ vienetų litre, neutrofilų poslinkis į kairę iki 75,82 % ir eritrocitų nusėdimo greitis padidėjęs iki 35 milimetrų per valandą. Atlikus pirminį požandikaulinės ertmės flegmonos pjūvį ir drenažą, išsiurbus 30–40 mililitrų pūliųvaiko būklė laikinai palengvėjo. Spalio 27 d. vaiko būklė smarkiai pablogėjo, procesui išplitus į giliąsias kaklo fascijų ertmes ir mediastinumą; prokalcitonino lygis pasiekė 0,47 nanogramų mililitre, o tai, atsižvelgiant į klinikinį pablogėjimą ir organų disfunkciją, patvirtino sepsio diagnozę. Buvo atlikta skubi kaklo mediastinotomija, kurios metu iš pretrachealinės, paraezofaginės ir prevertebralinės ertmių buvo pašalinta 100–150 mililitrų pūlių. Kompleksinis gydymas apėmė antibakterinę terapiją ceftriaksonu ir metronidazolu, detoksikacijos terapiją ir stebėjimą intensyviosios terapijos skyriuje. Teigiama dinamika dėl žaizdos valymo ir granuliacijų susidarymo leido lapkričio 5 d. užsiūti antrines siūles, po to žaizda sėkmingai užgijo ir berniukas buvo išrašytas iš ligoninės. Stebėjimo rezultatai pabrėžia ankstyvos diagnozės, daugiapakopio chirurginio žaizdos valymo ir visapusiškos intensyviosios terapijos svarbą plačiai išplitusių odontogeninių infekcijų atvejais vaikams. Pateiktas klinikinis atvejis gali padėti vaikų veido ir žandikaulių chirurgams, pediatrams ir intensyviosios terapijos gydytojams atpažinti šios sunkios būklės klinikinę eigą ir laiku nuspręsti dėl gydymo intensyvinimo sudėtingų odontogeninių infekcijų atvejais.

Raktažodžiai: odontogeninė infekcija, nusileidžiantis mediastinitas, sepsis, vaikas pacientas, kaklinė mediastinotomija.

Introduction

This clinical observation is particularly relevant because odontogenic infections in children may lead to severe complications, including deep neck infection, mediastinitis, and sepsis. Anatomical and physiological features of childhood, including immature fascial barriers and the immaturity of the immune system, may facilitate rapid spread of purulent-inflammatory processes. The clinical importance of odontogenic infections in paediatric practice is related to their variable presentation and potential progression to vital anatomical structures.

Previous studies have identified clinical and systemic factors associated with severe infection. Joachim et al. [1] found that older age and higher leukocyte counts at admission were associated with prolonged hospitalisation in children with odontogenic infections. Ismailov et al. [2] linked deficiencies in dental infrastructure and sterilisation capacity with a higher burden of purulent-inflammatory complications, while Segal et al. [3] showed the broader importance of infection control in vulnerable paediatric surgical patients. Microbiological studies have also demonstrated the contribution of anaerobic organisms and Gram-positive cocci to maxillofacial infections [4–7], supporting early microbiological assessment and appropriate antibacterial therapy.

Severe odontogenic infections may extend into the deep fascial spaces of the neck and mediastinum [8]. Evsen et al. [9] described sepsis and descending necrotising mediastinitis as major life-

threatening complications, particularly when surgical treatment is delayed. Ho et al. [10] identified involvement of multiple fascial spaces and mediastinitis as factors associated with a severe course and repeated surgical intervention. Biomarkers may complement clinical assessment: Abdullaev and Babokhujaev [11] reported associations between pro-inflammatory cytokine levels, including interleukin-8, and the severity of multiple organ dysfunction, while Gregoriano et al. [12] supported the use of procalcitonin for monitoring systemic infection and guiding antibiotic treatment. Chuliev [13] also emphasised the importance of early decompression of the infectious focus combined with aetiotropic antibacterial therapy in severe paediatric purulent-inflammatory disease.

Despite advances in diagnosis and treatment, paediatric deep neck infections remain difficult to manage because progression may be rapid and the threshold for escalation from local drainage to more extensive surgical decompression is not always straightforward [14]. The absence of unified paediatric algorithms for multidisciplinary management of odontogenic deep neck infection and mediastinal involvement therefore remains clinically relevant.

The present case is informative because an initially local odontogenic phlegmon in a 4-year-old child progressed within several days to deep neck and mediastinal involvement despite primary drainage, requiring repeat surgery and intensive-care management. The clinical sequence illustrates practical warning signs of progression and the need for repeated reassessment when the response to initial source control is incomplete.

The aim of this case report was to describe the clinical course and management of odontogenic putrefactive-necrotic phlegmon complicated by mediastinitis and sepsis in a 4-year-old child. The objectives were to characterise the clinical progression and diagnostic findings, to describe the indications and outcomes of staged surgical intervention and intensive therapy, and to assess recovery on the basis of clinical improvement, laboratory dynamics, and postoperative wound healing.

Materials and Methods

This retrospective case report analysed the inpatient course of a 4-year-old female patient with odontogenic putrefactive-necrotic phlegmon of the left submandibular region and deep fascial spaces of the neck, complicated by mediastinitis and sepsis. The patient was treated in the surgical department of the Municipal Children's Clinical Hospital No. 3, Bishkek, Kyrgyz Republic. The analysis covered the entire period of inpatient treatment from 23 October to 7 November 2025, including admission, clinical deterioration and the second stage of surgery on 27 October, intensive-care management, subsequent clinical improvement, secondary wound closure, and discharge. The source of odontogenic infection was the temporary molars 74 and 75 of the lower jaw on the left side. The total duration of inpatient treatment was 15 days, including 9 days in the intensive care unit.

Diagnosis of the disease included clinical examination with assessment of the general condition and local status of the maxillofacial region, as well as laboratory and instrumental methods of investigation. Clinical examination was performed with assessment of facial configuration, condition of the skin, palpation of regional lymph nodes, evaluation of the degree of mouth opening, and examination of the oral cavity with determination of the condition of the mucous membrane and causative teeth by percussion and palpation. The diagnosis of sepsis and assessment of the degree of multiple organ dysfunction were verified in accordance with the criteria of the international consensus Sepsis-3 [15], adapted for paediatric practice (paediatric Sequential Organ Failure Assessment (pSOFA)) [16]. An acute increase in the pSOFA score by ≥2 points was considered clinically significant. At the time of deterioration on 27 October, the pSOFA score was 4 points, comprising respiratory dysfunction (PaO_2/FiO_2 = 292; 2 points), renal dysfunction with a creatinine level of 74.4 μmol/L above the age-specific reference range (1 point), and arterial hypotension (1 point); the

liver, coagulation, and neurological components scored 0. These findings were interpreted together with the confirmed odontogenic infectious focus and inflammatory laboratory abnormalities.

Laboratory assessment included a complete blood count with leukocyte differential and erythrocyte sedimentation rate, urinalysis, serum biochemistry (total and indirect bilirubin, total protein, urea, creatinine, glucose, alanine aminotransferase, aspartate aminotransferase, and cholesterol), blood gas and electrolyte analysis, and serum procalcitonin. Venous and arterial samples were collected by using standard sterile techniques. The complete blood count was obtained by automated haematology analysis, while serum biochemistry was assessed by using standard photometric, colorimetric, and enzymatic methods. Blood gas analysis included pH, pCO_2, pO_2, sodium, potassium, chloride, glucose, and lactate; bicarbonate and base excess were reported as derived acid-base parameters. Procalcitonin was determined by immunochemiluminescence.

Instrumental assessment consisted of ultrasonography of the soft tissues of the neck using a high-resolution linear probe at 7–12 MHz in grey-scale and colour Doppler modes, with assessment of soft-tissue echostructure and the presence, size, and extent of fluid collections. Chest radiography was performed in the anteroposterior projection.

Surgical treatment included two stages of operative interventions. The first operation, incision and drainage of the phlegmon of the left submandibular space with removal of the causative teeth, was performed under premedication and local infiltration anaesthesia with a 1% solution of lidocaine hydrochloride in a volume of 15.0 ml. After preparation of the operative field with antiseptic solutions of chlorhexidine bigluconate 0.05% and povidone-iodine, an external submandibular approach was performed. The incision was made parallel to the lower border of the mandible, at a distance of 1.5 cm from it. The skin, subcutaneous fat, superficial fascia of the neck, and platysma muscle were dissected layer by layer. The facial artery and vein were isolated, transected between ligatures, and ligated. Blunt dissection was used to separate the tissues and enter the submandibular fascial space. The wound was irrigated with antiseptic solutions: hydrogen peroxide 3%, chlorhexidine bigluconate 0.05%, and isotonic sodium chloride solution 0.9%. Drainage was carried out by using a rubber tubular drain with "*Levomekol*" ointment. Removal of the causative teeth was also performed by using an elevator and forceps, followed by curettage of the sockets and irrigation with antiseptic solutions.

The second operation – cervical mediastinotomy with revision of the deep fascial spaces of the neck – was performed under general endotracheal anaesthesia with artificial ventilation of the lungs. For induction of anaesthesia, propofol 2% at a dose of 2–3 mg per kilogram of body weight, fentanyl 0.005% at a dose of 2–3 μg per kilogram of body weight, and muscle relaxation was achieved by administration of atracurium besilate 0.5 mg per kilogram. Maintenance of anaesthesia was carried out by inhalation of sevoflurane at a concentration of 1.5–2 volume%. After preparation of the operative field with iodopolyvinol solution, incisions were made along the anterior surface of the sternocleidomastoid muscle on the left and right sides from the mastoid process to the clavicle. The skin, subcutaneous fat, fasciae, and muscle layers were dissected layer by layer. The pretracheal, paraoesophageal, and prevertebral fascial spaces were sequentially opened with revision. A counter-aperture was created between the operative wounds, and a rubber strip drain was inserted. The wounds were tamponed with turundas soaked in hypertonic sodium chloride solution 10%. Additionally, the parapharyngeal space on the left was opened with the creation of a counter-aperture between the wound in the submandibular region and the anterior surface of the neck.

Conservative treatment included antibacterial, anti-inflammatory, detoxification, and symptomatic therapy. Antibacterial therapy at the first stage was carried out with amikacin at a dose of 60 mg intramuscularly three times a day; subsequently, the regimen was adjusted with the administration of ceftriaxone 1,300 mg intravenously once daily and metronidazole 32 mg intravenously twice daily.

Detoxification therapy was carried out by intravenous administration of isotonic sodium chloride solution 0.9% in a volume of 200–400 ml per day and Ringer's solution at 250 ml per day. Anti-inflammatory therapy included dexamethasone 0.4% at a dose of 4 mg intramuscularly once daily. Analgesia was provided with ketoprofen 3% at a dose of 6.3 mg intramuscularly twice daily. Haemostatic therapy included sodium etamsylate in the form of a 10% solution at a dose of 1 ml intramuscularly. Sedative therapy was carried out with diazepam 0.5% at a dose of 4 mg as indicated. In the intensive care unit, oxygen therapy with humidified oxygen via nasal cannulae, sanitation of the tracheobronchial tree, and correction of water-electrolyte balance and acid-base status were performed.

Placement of secondary sutures was performed under neuroleptanalgesia with administration of droperidol 0.25% at a dose of 1 ml and fentanyl 0.005% at a dose of 1 ml, and local infiltration anaesthesia with a 1% solution of lidocaine hydrochloride in a volume of 15 ml. Mobilisation of the wound edges was performed, followed by placement of interrupted sutures with 3-0 silk on the skin.

At the stage of rehabilitation and completion of inpatient treatment, a set of physiotherapeutic measures was developed to prevent the development of mandibular contracture and cicatricial deformities of the soft tissues. For the prevention of jaw contracture and cicatricial deformities, the patient was prescribed outpatient rehabilitation for a course of 10–14 days. The programme includes magnetotherapy to improve microcirculation, ultrasound therapy to stimulate regeneration and soften scars, as well as therapeutic physical exercises to restore the amplitude of mouth opening and train the masticatory muscles.

Analysis of the obtained results was carried out by means of comparison of clinical, laboratory, and instrumental data over the course of the disease. To ensure objectivity of the obtained data, the method of clinical-anamnestic analysis was applied with sequential comparison of the results of clinical, laboratory, and instrumental investigations over the course of the disease. Laboratory parameters were evaluated against age-specific reference intervals established within the Canadian Laboratory Initiative on Pediatric Reference Intervals (CALIPER) [17] and the reference ranges applicable to the diagnostic assays used. Descriptive statistics were not applied due to the study format as a clinical observation of a single case.

The study was conducted in accordance with the ethical principles of the WMA Declaration of Helsinki [18]. The patient's relatives provided written informed consent for examination, surgical treatment, and subsequent analysis of medical documentation for the purposes of scientific research.

Results

Clinical presentation at admission and primary diagnosis

Patient K. P., aged 4 years, born in 2021, was admitted to the surgical department on 23 October 2025 with complaints of pain and swelling of the soft tissues in the left submandibular, submental, and retromandibular regions, limited and painful mouth opening, difficulty swallowing, an increase in body temperature up to 38 °C, general weakness, malaise, sleep disturbance, and loss of appetite. From the medical history, it was established that, four days prior to admission, pain appeared in the region of teeth 74 and 75 of the lower jaw on the left side. No medical assistance was sought, and no treatment was provided. After two days, the condition worsened: progressive swelling of the soft tissues of the left submandibular region developed, pain on swallowing appeared, and the body temperature increased. The relatives brought the child to the surgical department of the children's clinical hospital, where the patient was examined by the on-duty resident doctor and hospitalised on an emergency basis.

On objective examination at admission, the general condition was assessed as of moderate severity. Body temperature was 38.0°C. The child was lethargic, irritable, and refused food intake.

The skin was pale, and increased sweating was noted. Heart rate was 120 beats per minute, arterial blood pressure was 95/55 mm Hg. Respiratory rate was 24 per minute, with vesicular breathing. The tongue was coated with a white deposit and was dry. The abdomen remained soft and painless, with preserved intestinal peristalsis.

On external examination, facial asymmetry was noted due to collateral swelling of the soft tissues in the left submandibular, submental, and retromandibular regions. The skin over the swelling was hyperaemic, tense, with locally increased temperature, and could not be pinched into a fold. On palpation, a diffuse painful infiltrate of dense consistency without clear boundaries was determined. Examination of the oral cavity revealed limitation of mouth opening to 1.0 cm. The mucous membrane of the alveolar process in the region of teeth 74 and 75 was oedematous and hyperaemic. The sublingual fold on the left protruded above the level of the floor of the oral cavity, and the tongue was displaced to the right. The mucous membrane over the fold was oedematous, hyperaemic, and sharply painful on palpation. The crown portions of teeth 74 and 75 were destroyed at the level of the gingiva; pathological mobility of the second degree was determined, and percussion of the teeth caused severe pain.

The results of the complete blood count at admission on 23 October 2025 revealed leukocytosis up to 14.27×10^9/L (normal $4.0–9.0\times10^9$/L). In the leukocyte differential, a left shift was noted: segmented neutrophils accounted for 75.82% (normal 47–72%), lymphocytes decreased to 9.44% (normal 18–40 %), monocytes increased to 14.36% (normal 3–10%), eosinophils accounted for 0.36% (normal 1–5%). The erythrocyte count was 4.13×10^{12}/L. Haemoglobin level decreased to 108 g/L (normal 120 g/L). Haematocrit was 32.4%. Platelet count was within normal limits – 262×10^9/L. The erythrocyte sedimentation rate was significantly increased to 35 mm/h (normal 2–15 mm/h). The results of urinalysis on 23 October 2025 showed absence of protein; in the sediment, squamous epithelium was determined in the amount of 5–7 cells per field of view, leukocytes were established as 10–12 per field of view.

On the basis of clinical data and examination results, the diagnosis was established: odontogenic putrefactive-necrotic phlegmon of the left submandibular region originating from teeth 74 and 75. The patient's relatives were offered surgery – incision and drainage of the phlegmon of the left submandibular space with removal of teeth 74 and 75. Written informed consent for the operation was obtained. On the same day, 23 October 2025, within two hours from the moment of admission, the operation was performed. Upon opening the submandibular fascial space, approximately 30–40 ml of pus of thick consistency with a pronounced putrefactive odour and the presence of gas bubbles was obtained. Visually, throughout the surface of the operative wound, areas of necrotically altered tissues of grey-green colour were identified. The wound was drained with a rubber tubular drain with "*Levomekol*" ointment. From the oral cavity side, teeth 74 and 75 were removed, and curettage of the sockets was performed. Postoperative diagnosis was as follows: odontogenic putrefactive-necrotic phlegmon of the left submandibular region originating from teeth 74 and 75.

In the postoperative period, antibacterial therapy with amikacin 60 mg three times daily intramuscularly, anti-inflammatory therapy with dexamethasone 0.4% at a dose of 4 mg, analgesia with ketoprofen 3% at a dose of 6.3 mg, detoxification therapy with 0.9% sodium chloride solution and Ringer's solution, haemostatic therapy with sodium etamsylate, and sedative therapy with diazepam as indicated were prescribed. In the early postoperative period, moderate positive dynamics were observed: body temperature decreased to subfebrile values of 37.5°C, and the pain syndrome was reduced. However, from 25 October, the patient's condition began to deteriorate. Complaints of cough with expectoration of mucous sputum and dyspnoea on exertion appeared. On auscultation of the lungs, moist rales were heard in the lower sections on the right. Body temperature increased to 38.5°C. The development of hospital-acquired pneumonia was suspected.

Disease progression and the second stage of surgical treatment

By 27 October 2025, the patient's condition had sharply deteriorated and was assessed as extremely severe, due to progression of the purulent-septic process and increasing symptoms of intoxication. Oedema and infiltration of the soft tissues spread beyond the submandibular region and involved the entire anterolateral surface of the neck on the left up to the level of the clavicle. The skin in the area of swelling was hyperaemic, tense, and warm to the touch. On palpation, a diffuse painful infiltrate of dense-elastic consistency was determined, occupying the submandibular, submental regions and the anterior and lateral triangles of the neck on the left. From the postoperative wound, scant turbid discharge with a sharp fetid odour was noted. Mouth opening remained limited and painful, and swallowing was difficult. From the sockets of the extracted teeth 74 and 75, pus with a putrefactive odour was discharged.

On 27 October 2025, at the peak of clinical deterioration, the pSOFA score was 4 points [12]. The assessment comprised respiratory dysfunction (PaO2/FiO2 = 292; 2 points), renal dysfunction with a creatinine level of 74.4 μmol/L above the age-specific reference range (1 point), and arterial hypotension (1 point); the liver, coagulation, and neurological components scored 0. In the context of the odontogenic infectious focus, leukocytosis of 12.6×10^9/L, procalcitonin of 0.31-0.47 ng/mL, and an erythrocyte sedimentation rate of 39 mm/h, these findings supported the diagnosis of sepsis in accordance with the Sepsis-3 criteria [15].

Ultrasound examination confirmed the presence of a purulent infiltrate with spread to the deep fascial spaces. On the basis of clinical and instrumental data, the diagnosis was established: odontogenic putrefactive-necrotic phlegmon of the submandibular region and deep fascial spaces of the neck, reactive mediastinitis, sepsis. A case conference with the participation of surgeons and an anaesthesiologist recommended emergency surgical intervention – cervical mediastinotomy with revision of the deep spaces of the neck under endotracheal anaesthesia. Emergency cervical mediastinotomy was aimed at debridement of the infectious focus, prevention of further spread of the inflammatory process, and reduction of the severity of systemic intoxication. The operative approach scheme and the main stages of the surgical intervention are presented in Figure 1.

Figure 1 presents the scheme of the operative approach and the main stages of the intervention. Bilateral incisions were made along the anterior surface of the sternocleidomastoid muscle from the mastoid process to the clavicle. Revision of the pretracheal, paraoesophageal, and prevertebral



Figure 1. Intraoperative findings during revision and opening of the deep fascial spaces of the neck in mediastinitis

Source: produced by the authors.

fascial spaces on the left was performed with evacuation of 100–150 ml of pus with gas bubbles; purulent tracks extended into the anterior and posterior mediastinum. Similar debridement was performed on the right. A counter-aperture was formed between the operative wounds with placement of a rubber strip drain; the wounds were tamponed with turundas soaked in 10% sodium chloride solution. Additionally, the parapharyngeal space on the left was opened with evacuation of 20–30 ml of pus, and a counter-aperture was created between the wound in the submandibular region and the anterior surface of the neck. Blood loss amounted to approximately 150 ml and was compensated by infusion of crystalloid solutions.

The postoperative diagnosis was established as: odontogenic putrefactive-necrotic phlegmon of the submandibular region and deep fascial spaces of the neck, reactive mediastinitis, sepsis. In the postoperative period, the patient received antibacterial therapy: ceftriaxone 1,300 mg intravenously once daily, metronidazole 32 mg intravenously twice daily. Detoxification therapy was intensified: 0.9% sodium chloride solution up to 400 ml/day and Ringer's solution 250 ml/day. The patient was transferred to the intensive care unit.

Dynamic observation in the intensive care unit

On examination in the intensive care unit on the evening of 27 October 2025, the patient's condition was assessed as extremely severe due to the purulent-septic process and pronounced intoxication. The skin and visible mucous membranes were pale, and the extremities were cool to the touch. Body temperature was 36.6 °C. Breathing was harsh on auscultation, with no rales heard. Heart sounds were muffled, rhythm regular, heart rate was 153 beats per minute, arterial pressure measured 100/37 mm Hg. The abdomen was of a normal shape, soft, and intestinal peristalsis was auscultated. Urination was spontaneous. The dressing was heavily soaked with wound discharge, and swelling of the soft tissues persisted. The child was conscious, the muscle tone was restored, and cough and swallowing reflexes were elicited. After debridement of the tracheobronchial tree, extubation was performed without complications. Humidified oxygen was administered via nasal cannulae, with oxygen saturation at 95%.

Chest radiography showed clear lung fields without focal or infiltrative changes; the lung roots were structured and not enlarged; pleural cavities were free. The cardiac shadow was not enlarged, and bony structures showed no pathology. The complete blood count revealed a decrease in haemoglobin to 95 g/l, leukocytosis of 12.6×10^{9}/l, erythrocytes 3.67×10^{12}/l, lymphocytes 12.3%, monocytes 1.2×10^{9}/l, granulocytes 9.9×10^{9}/l, haematocrit 26.8%. The procalcitonin level was 0.31 ng/ml, which, together with the clinical and laboratory findings, supported the diagnosis of sepsis.

On examination on 28 October 2025, the patient's condition remained severe. Consciousness was clear, with an adequate response to examination; pupils were equal, and the photoreaction was preserved. Appetite was reduced; fluid intake was adequate; no vomiting was observed. The skin and mucous membranes were pale pink; extremities were warm. No fever was noted. Breathing remained spontaneous and regular; on auscultation, it was harsh; oxygen saturation was 93%, respiratory rate was 20 per minute. Heart sounds were muffled, the rhythm was regular, with a heart rate (HR) of 60 beats per minute, and arterial pressure (BP) measuring 80/40 mm Hg. The abdomen remained soft and painless; urination was spontaneous. The postoperative wounds had moderate purulent discharge with a fetid odour and signs of tissue necrosis. Wound debridement was performed with antiseptic solutions, and an aseptic dressing with "*Levomekol*" ointment was applied. The condition of the wounds two days after cervical mediastinotomy is presented in Figure 2.

Figure 2 shows two wounds with tampons soaked in an antiseptic solution; soft tissue oedema persists, the skin around the site is pale pink, and local areas of tissue necrosis are identified. The dynamics confirm the necessity of supportive detoxification and antibacterial therapy, regular surgical wound debridement, and further observation in the intensive care unit.



Figure 2. Condition of postoperative wounds 2 days after cervical mediastinotomy and debridement of purulent foci

Source: produced by the authors.

The complete blood count of 28 October showed an erythrocyte sedimentation rate (ESR) of 39 mm/h, indicating a persistent pronounced inflammatory response. Biochemical analysis revealed hypoproteinaemia (total protein 52.6 g/l), bilirubin 8.16 mmol/l, urea 7.32 mmol/l, creatinine 74.40 μmol/L, glucose 6.7 mmol/l, aspartate aminotransferase (AST) 17.19 U/l, alanine aminotransferase (ALT) 15.12 U/l, cholesterol 3.1 mmol/l. The blood gas parameters were as follows: pH 7.427, pCO_2 38.1 mm Hg, pO_2 61.4 mm Hg, HCO_3^- 25.3 mmol/l, haematocrit 31%, sodium 137.8 mmol/l, potassium 4.48 mmol/l, chloride 103.3 mmol/l, lactate 0.9 mmol/l. Procalcitonin was 0.47 ng/ml, supporting the presence of an ongoing systemic inflammatory response in the context of persistent sepsis.

Positive dynamics of general condition and cleansing of postoperative wounds

By 30 October 2025, the patient's condition began to improve. Sleep and appetite improved, and the child became more active. Consciousness remained clear. The body temperature was 36.1°C, and the patient remained afebrile. Breathing was spontaneous and regular, slightly harsh on auscultation, and symmetrical on both sides. When breathing room air, oxygen saturation reached 97%, and no oxygen dependence was observed. Chest percussion produced a normal resonant note. The respiratory rate was 20 per minute. Heart sounds were muffled, and the rhythm was regular. The peripheral pulse was of satisfactory filling and tension. The heart rate was 70 beats per minute, whereas the arterial pressure measured 90/40 mm Hg. The abdomen was soft, painless, of normal shape, and not distended. The liver was palpated at the costal margin. Intestinal peristalsis was auscultated. Urination was spontaneous.

On examination of the local status, oedema of the soft tissues in the area of the postoperative wounds persisted; however, a tendency towards its reduction was noted. The dressing was intact and clean, with slight staining from wound exudate. The wounds began to clear of purulent exudate and necrotic masses; healthy granulation tissue was observed. The rubber drain was removed from the wounds due to an improved outflow and a decrease in the amount of discharge. The postoperative wounds were irrigated with antiseptic solutions of hydrogen peroxide 3% and chlorhexidine bigluconate 0.05%. A loose aseptic dressing with "*Levomekol*" ointment was applied. The process of wound cleansing on the seventh day after cervical mediastinotomy is presented in Figure 3.

Figure 3 shows a reduction in soft tissue oedema and cleansing of the wounds. The wounds were irrigated with antiseptic solutions (3% hydrogen peroxide and 0.05% chlorhexidine bigluconate). An



Figure 3. Clinical appearance of wound cleansing and well-formed granulation tissue in the deep fascial spaces of the neck

Source: produced by the authors.

aseptic dressing with "*Levomekol*" ointment maintained sanitary conditions and promoted the formation of granulations. The dynamics presented in the figure reflect gradual normalisation of local and systemic status: improvement of tissue perfusion and progression of granulation tissue formation, confirming the effectiveness of ongoing antibacterial, detoxification, and symptomatic therapy. A decrease in pain on palpation was noted; the child tolerated examination without pronounced discomfort.

In the following days, the patient's condition continued to improve. The body temperature remained normal, and signs of intoxication regressed. Appetite was restored, and the child resumed normal oral intake of food and fluids. Haemodynamic and respiratory parameters normalised. The postoperative wounds continued to cleanse; the amount of purulent discharge decreased significantly, and granulation tissue matured and progressively filled the wound defects. Dressings were performed daily with irrigation of the wounds using antiseptic solutions and application of "*Levomekol*" ointment. Antibacterial therapy with ceftriaxone and metronidazole, as well as detoxification and symptomatic therapy, were continued. The patient's general condition improved: the appetite increased, sleep normalised, and general weakness resolved.

Placement of secondary sutures and successful completion of inpatient treatment

By 5 November 2025, on the thirteenth day after primary incision and drainage of the phlegmon and on the ninth day after cervical mediastinotomy, the patient's condition was assessed as relatively satisfactory. Breathing remained spontaneous, regular, and symmetrical on both sides. Oxygen saturation on room air was 98%, the respiratory rate was 18 per minute. The heart sounds and rhythm were regular. The peripheral pulse was of satisfactory filling and tension. The heart rate was 68 beats per minute, whereas the arterial pressure measured 90/40 mm Hg. The abdomen was of a normal shape, soft, and painless. Urination was spontaneous, with adequate diuresis.

On examination of the local status, the dressing was intact, clean, and dry. The postoperative wounds had significantly decreased in size compared with the initial state. Abundant healthy granulation tissue completely filled the postoperative wound defects. The wound discharge was serous, scant, without pathological impurities or unpleasant odour. The wound edges became mobile, soft, and without signs of inflammation. Soft tissue oedema significantly decreased. Considering the favourable course of the wound process, the presence of mature granulations, and the absence of signs of inflammation, a decision was made to place secondary sutures to accelerate healing and improve the cosmetic outcome. Wound debridement was performed with antiseptic solutions, and an asep-



Figure 4. Placement of secondary sutures 9 days after incision and drainage of purulent foci

Source: produced by the authors.



Figure 5. Condition of the wounds 11 days after incision and drainage of purulent foci

Source: produced by the authors.

tic dressing with *"Levomekol"* ointment was applied. On the same day, under neuroleptanalgesia and local infiltration anaesthesia, secondary sutures were placed on the postoperative wounds. The wound edges were mobilised; interrupted sutures with 3-0 silk were placed on the skin at a distance of 1–1.5 cm between sutures without tissue tension. Haemostasis was achieved, and an aseptic dressing was applied. The process of placement of secondary sutures is presented in Figure 4.

Figure 4 shows the placement of secondary sutures 9 days after incision and drainage of purulent foci. The postoperative wounds had significantly decreased in size; healthy granulation tissue was observed, the discharge was scant and serous, without odour. Soft tissue oedema decreased, and the wound edges were soft and mobile. The dynamics indicated favourable healing and allowed for the placement of secondary sutures with an aseptic dressing and "*Levomekol*" ointment.

The postoperative period after the placement of secondary sutures proceeded smoothly, without complications. The sutures were intact, with no signs of wound edge dehiscence or inflammation. The wounds healed uneventfully after secondary closure. Dressings were performed daily with the treatment of the sutures using antiseptic solutions. By 7 November 2025, 11 days after incision and drainage of purulent foci and 2 days after placement of secondary sutures, the condition of the postoperative wounds had significantly improved. The sutures were intact, and the wound edges were closely apposed. Soft tissue oedema had almost completely regressed. Discharge from the drainage openings was scant and serous. The condition of the wounds at this stage is presented in Figure 5.

Figure 5 shows intact secondary sutures placed on the wounds of the submandibular region and the anterior surface of the neck. The wound edges are closely apposed; signs of inflammatory infiltration, hyperaemia, or suture dehiscence are absent. The skin has been treated with an antiseptic solution (brilliant green); discharge from the drainage openings is essentially absent. This visualisation confirms successful resolution of the purulent-septic process and transition of the wound process to the phase of consolidation and epithelialisation.

The general condition of the patient at discharge was satisfactory. The body temperature remained normal over the previous seven days, and signs of intoxication completely regressed. Appetite and sleep were restored. The child was active, played, and communicated with relatives. The mouth opening was almost completely restored, with a slight limitation up to 3.5 cm remaining, determined by postoperative cicatricial changes and requiring further rehabilitation. Swallowing was not impaired, and oral feeding was performed without difficulty. Haemodynamic and respiratory parameters corresponded to age norms. The patient was discharged from hospital with recommendations for further outpatient follow-up with a maxillofacial surgeon, paediatrician, and physiotherapist. Physiotherapeutic treatment was also prescribed for the prevention of mandibular contracture and cicatricial deformities of the soft tissues, including magnetotherapy, ultrasound therapy, and therapeutic physical exercises for the masticatory muscles.

Discussion

The present case demonstrates rapid progression of an odontogenic infection from a local submandibular phlegmon to the deep cervical spaces and mediastinum in a 4-year-old child. Primary incision, drainage, and removal of the causative teeth produced only temporary improvement. Subsequent spread of cervical oedema and infiltration, persistent fetid discharge, dysphagia, systemic deterioration, elevated inflammatory markers, and ultrasonographic evidence of deep-space involvement indicated that the infection was no longer confined to the initially drained submandibular compartment. These findings justified escalation to cervical mediastinotomy and revision of the deep fascial spaces.

This clinical course is consistent with previous descriptions of descending odontogenic infection. Ma et al. [19] and Lou et al. [20] emphasised the potential for deep neck infection to extend rapidly to the mediastinum, while Ogle [21] described the anatomical pathways that permit spread through the pretracheal, paraoesophageal, and prevertebral spaces. Ortiz and Espinoza [22] highlighted trismus and respiratory symptoms as warning signs of severe deep-space infection, and Escobedo et al. [23] reported poorer outcomes in diffuse than in localised disease. In the present case, delayed presentation, marked trismus, dysphagia, progressive cervical swelling, and later respiratory and haemodynamic instability were therefore clinically important signs of progression. The use of broad-spectrum antibacterial therapy together with surgical source control was also consistent with the management principles discussed by Pastene et al. [24], Eshiev et al. [25], and Moldalievich et al. [26].

Several prognostic features reported in the literature were reflected in this case without implying that findings from larger cohorts can be directly extrapolated to a single patient. Wang et al. [27] identified fever, involvement of multiple spaces, and leukocytosis as factors associated with complications, all of which were present here. Zamiri et al. [28] discussed laboratory abnormalities associated with prolonged hospitalisation, while Baroot et al. [29] emphasised anatomical characteristics of children that may facilitate the spread of infection. The four-day delay before presentation in the present case is also clinically relevant in light of the importance of early comprehensive treatment, as discussed by Varzhapetian and Kopchak [30] and Iodice et al. [31].

Published paediatric cases demonstrate that both operative and conservative strategies have been used, depending on the extent and behaviour of infection. Okumura et al. [32] and Marques et al. [33] described severe paediatric mediastinitis requiring intensive treatment and drainage, whereas

Lira et al. [34] and Yamasaki et al. [35] reported successful conservative management in selected cases with early presentation and a favourable response to antibiotic therapy. Demongeot et al. [36] further showed that the extent of purulent collections is relevant to the decision for surgical drainage. In the present patient, conservative management was not appropriate because the infection was associated with putrefactive purulent exudate, gas formation, visible tissue necrosis, extensive involvement of the deep neck spaces and mediastinum, and sepsis. These features, together with the large volume of purulent material found at reoperation, favoured an aggressive surgical strategy.

The clinical warning signs observed in this child also resemble severity markers reported in larger series [37, 38]. Fornari et al. [39] identified trismus, fever, swelling, and deep-space involvement as predictors of hospital admission, while Ullah et al. [40] reported dysphagia, trismus, tachycardia, and leukocytosis as common markers in hospitalised odontogenic infections. Lixandru et al. [41] showed that more complex antibacterial management is associated with more severe clinical courses and longer hospital stays. Matta [42], although describing sclerosing rather than infectious mediastinal disease, illustrates the importance of differential diagnosis in children with mediastinal involvement; in the present case, the odontogenic history and intraoperative purulent-necrotic findings supported an infectious origin.

Procalcitonin was used as an additional marker of systemic infection and treatment dynamics [43, 44]. Li et al. [45] and Postic et al. [46] showed that procalcitonin-guided strategies may help optimise antibiotic duration in paediatric infections; however, in this case treatment decisions were based on the overall clinical course, organ dysfunction, wound status, and laboratory dynamics rather than on a single biomarker. Multidisciplinary coordination between the surgical, anaesthesia, and intensive-care teams facilitated repeated clinical reassessment, emergency surgical escalation, respiratory and haemodynamic monitoring, adjustment of antibacterial therapy, serial wound debridement, and subsequent secondary closure once healthy granulation tissue had formed.

Overall, the case supports a low threshold for repeated assessment and escalation of source control when paediatric odontogenic infection is accompanied by rapidly progressive cervicofacial swelling, persistent trismus or dysphagia, respiratory or haemodynamic deterioration, and evidence of deep-space extension. Conservative treatment may be appropriate in selected paediatric cases, but the presence of necrosis, gas formation, extensive purulent collections, or systemic deterioration favours prompt surgical drainage.

Conclusions

This case illustrates the potential for a paediatric odontogenic infection to progress rapidly from a local submandibular phlegmon to deep neck and mediastinal involvement with sepsis. Temporary improvement after primary drainage was followed by clinical deterioration, requiring repeated assessment and escalation to cervical mediastinotomy. Staged surgical source control, antibacterial therapy, intensive-care monitoring, and subsequent wound management were followed by progressive clinical recovery and successful secondary wound closure.

From a practical perspective, rapidly progressive cervicofacial swelling, persistent trismus or dysphagia, and emerging respiratory or haemodynamic instability should prompt clinicians to suspect extension of infection into the deep neck spaces or mediastinum. In such cases, prompt reassessment, appropriate imaging, and timely surgical source control should be considered.

The principal limitation of this report is its single-case retrospective design, which precludes generalisation to all paediatric patients. In addition, the independent effectiveness of individual treatment components cannot be determined because surgical, antibacterial, detoxification, and supportive interventions were applied simultaneously. Future multicentre case series may help clarify risk factors, escalation criteria, and long-term functional outcomes in paediatric odontogenic mediastinitis.

Author contributions

B. U.: Conceptualization, Methodology, Investigation, Data Curation, Formal Analysis, Writing – Original Draft Preparation, Writing – Review & Editing.

D. T.: Methodology, Investigation, Data Curation, Validation, Writing – Original Draft Preparation, Writing – Review & Editing.

A. M.: Investigation, Resources, Formal Analysis, Validation, Visualization, Writing – Review & Editing.

A. A.: Project Administration, Supervision, Resources, Validation, Writing – Review & Editing.

I. S.: Conceptualization, Methodology, Formal Analysis, Supervision, Writing – Original Draft Preparation, Writing – Review & Editing.

References

1. Joachim MV, Abdo M, Alkeesh K, Oettinger-Barak O, Abozaid S, El-Naaj IA. Factors associated with prolonged hospitalizations from odontogenic infections in children. *J Clin Pediatr Dent*. 2022;46(6):40-44. doi:10.22514/jocpd.2022.024
2. Ismailov A, Arstanbekov M, Mamaev N, Jorobaev S, Zhenishbek P, Pakyrov ZK, Imetkul I. Medical and social aspects of hemocontact infections in dental institutions of Osh region, Kyrgyzstan. *Bulletin of Stomatology and Maxillofacial Surgery*. 2025;21(4):93-101. https://stomatology-mfsjournal.com/wp-content/uploads/2025/07/Volume-21-No.-4.pdf
3. Segal I, Kang C, Albersheim SG, Skarsgard ED, Lavoie PM. Surgical site infections in infants admitted to the neonatal intensive care unit. *J Pediatr Surg*. 2014;49(3):381-384. doi:10.1016/j.jpedsurg.2013.08.001
4. Kubanychbekov MK, Adambekov DA, Aldzhambaeva IS. The features of the microbial spectrum of obligate anaerobes in purulent phlegmons of odontogenic nature in maxillofacial pathology. *Health Care of Kyrgyzstan*. 2021;3:133-138. https://zdrav.kg/images/133-138.pdf
5. Lokes K, Kiptilyi A, Skikevych M, et al. Microbiological substantiation of the effectiveness of quercitin and its combination with ethylmethylhydroxypyridine succinate in the complex treatment of odontogenic phlegmon and maxillofacial abscesses. *Front Oral Health*. 2024;5:1338258. doi:10.3389/froh.2024.1338258
6. Boffano P, Brucoli M, Ruslin M, et al. A European multicenter retrospective study of hospitalized patients with severe odontogenic infections. *J Dent*. 2025;163:106183. doi:10.1016/j.jdent.2025.106183
7. Harizanova Z, Baltadjiev A. Anthropological study of dental indices in Bulgarian population. *Folia Med*. 2023;65(4):659-663. doi:10.3897/folmed.65.e84820
8. Izzotti A, Menini M, Pulliero A, et al. Biphosphonates-associated osteonecrosis of the jaw: the role of gene-environment interaction. *J Prev Med Hyg*. 2013;54(3):138-145.
9. Evsen EA, Candan M, Dur MP. Serious Complications and Treatment Strategies Associated with Odontogenic Infections. *Eurasian J Med*. 2023;55(1):142-149. doi:10.5152/eurasianjmed.2023.23378
10. Ho CY, Wang YC, Chin SC, Chen SL. Factors Creating a Need for Repeated Drainage of Deep Neck Infections. *Diagnostics (Basel)*. 2022;12(4):940. doi:10.3390/diagnostics12040940
11. Abdullaev SY, Babokhujaev AS. Prognostic and preventive approaches in complicated odontogenic inflammatory diseases. *American Journal of Medicine and Medical Sciences*. 2025;15(12):4371-4373. doi:10.5923/j.ajmms.20251512.38
12. Gregoriano C, Heilmann E, Molitor A, Schuetz P. Role of procalcitonin use in the management of sepsis. *J Thorac Dis*. 2020;12(Suppl 1):S5-S15. doi:10.21037/jtd.2019.11.63
13. Chuliev MS. Improving the principle of diagnostics and treatment of purulent inflammatory diseases in children based on classifications. *Science and Innovation International Scientific Journal*. 2024;3(3):150-155. doi:10.5281/zenodo.10868211
14. Imanov EA. Usage of modern medications in complex treatment of inflammatory periodontal diseases of children. *Azerbaijan Medical Journal*. 2019;2019(4):55-59.
15. Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA*. 2016;315(8):801-810. doi:10.1001/jama.2016.0287

16. Malik A, Taksande A, Meshram R. Pediatric Sequential Organ Assessment Score: A Comprehensive Review of the Prognostic Marker in the Pediatric Intensive Care Unit. *Cureus*. 2024;16(5):e60034. doi:10.7759/cureus.60034
17. Jung B, Adeli K. Clinical laboratory reference intervals in pediatrics: the CALIPER initiative. *Clin Biochem*. 2009;42(16-17):1589-1595. doi:10.1016/j.clinbiochem.2009.06.025
18. World Medical Association. 2024. WMA Declaration of Helsinki – Ethical Principles for Medical Research Involving Human Participants. https://www.wma.net/policies-post/wma-declaration-of-helsinki/
19. Ma C, Zhou L, Zhao JZ, et al. Multidisciplinary treatment of deep neck infection associated with descending necrotizing mediastinitis: a single-centre experience. *J Int Med Res*. 2019;47(12):6027-6040. doi:10.1177/0300060519879308
20. Lou Y, Sun Z, Ma H, et al. Odontogenic infections in the antibiotic era: approach to diagnosis, management, and prevention. *Infection*. 2024;52(2):301-311. doi:10.1007/s15010-023-02117-5
21. Ogle OE. Odontogenic Infections. *Dent Clin North Am*. 2017;61(2):235-252. doi:10.1016/j.cden.2016.11.004
22. Ortiz R, Espinoza V. Odontogenic Infection. Review of the Pathogenesis, Diagnosis, Complications and Treatment. *Res Rep Oral Maxillofac Surg*. 2021;5(2):055. doi:10.23937/2643-3907/1710055
23. Escobedo MF, Junquera LM, Megias J, García-San Narciso L, Fernández MJ, Junquera S. Mediastinitis of odontogenic origin. A serious complication with 80 years of history. *Br J Oral Maxillofac Surg*. 2021;59(6):683-689. doi:10.1016/j.bjoms.2020.09.004
24. Pastene B, Cassir N, Tankel J, et al. Mediastinitis in the intensive care unit patient: a narrative review. *Clin Microbiol Infect*. 2020;26(1):26-34. doi:10.1016/j.cmi.2019.07.005
25. Eshiev A, Asanov A, Moldaliev Z, Eshiev D, Arstanbekov M. Influence of environmental factors on the condition of adolescent dental hard tissues: Clinical and physicochemical analyses. *Jurnal Ilmiah Ilmu Terapan Universitas Jambi*. 2025;9(1):406-421. doi:10.22437/jiituj.v9i1.38513
26. Moldalievich EA, Esenalievna AR, Abdyrakmanovich ED, Muhtaralievich AN, Omuralievich EE. Evaluating bone tissue biotypes in the maxillary posterior region: A CBCT-based observational study with clinical case report in elderly patients. *Bulletin of Stomatology and Maxillofacial Surgery*. 2025;21(6):56-64. doi:10.58240/1829006X-2025.21.6-56
27. Wang Y, Li Z, Chen Y, et al. Evaluating the risk factors for complications of patients with oral and maxillofacial space infections: a systematic review and meta-analysis. *BMC Oral Health*. 2025;25(1):1115. doi:10.1186/s12903-025-06521-1
28. Zamiri B, Hashemi SB, Hashemi SH, Rafiee Z, Ehsani S. Prevalence of odontogenic deep head and neck spaces infection and its correlation with length of hospital stay. *Shiraz Univ Dent J*. 2012;13(1):29-35. https://applications.emro.who.int/imemrf/J_Dent_Shiraz_Univ_Med_Sci/J_Dent_Shiraz_Univ_Med_Sci_2012_13_1_29_35.pdf
29. Baroot YAM, Alotaibi WAM, Alotibi AAM, Zalah AME, Darraj OA, Al Aftan MNH, et al. Odontogenic orofacial infections in pediatrics: Pathogenesis, diagnostic challenges, and advanced interdisciplinary management in clinical practice. *Saudi J Med Public Health*. 2025;2(2):1480-1496. doi:10.64483/202522298
30. Varzhapetian SD, Kopchak AV. The concept of complex treatment of odontogenous phlegmons of maxillofacial area. *Ukrainian Dental Almanac*. 2023;(1):17-20. doi:10.31718/2409-0255.1.2023.03
31. Iodice G, Bocchino T, Casadei M, Baldi D, Robiony M. Evaluations of sagittal and vertical changes induced by surgically assisted rapid palatal expansion. *Journal of Craniofacial Surgery*. 2013;24(4):1210-1214. doi:10.1097/SCS.0b013e3182997830
32. Okumura T, Tetsuka N, Yamaguchi M, et al. A Case of Descending Necrotizing Mediastinitis in a Previously Healthy Child. *Case Rep Pediatr*. 2021;2021:3159092. doi:10.1155/2021/3159092
33. Marques DL, Rato C, Miguéis A, Miguéis J. Descending necrotising mediastinitis: a rare entity in children. *BMJ Case Rep*. 2024;17(3):e258304. doi:10.1136/bcr-2023-258304
34. Lira J, Santos J, Capela M, Rodrigues J, Cunha O, Carvalho I. Descending Mediastinitis in a Child: Diagnostic Challenges and a Different Treatment Approach. *Clin Pract*. 2021;11(3):505-508. doi:10.3390/clinpract11030066
35. Yamasaki Y, Nishi J, Nishikawa T, et al. Descending necrotizing mediastinitis secondary to retropharyngeal abscess in a child. *J Infect Chemother*. 2008;14(3):255-257. doi:10.1007/s10156-008-0606-3
36. Demongeot N, Akkari M, Blanchet C, et al. Pediatric deep neck infections: Clinical description and analysis of therapeutic management. *Arch Pediatr*. 2022;29(2):128-132. doi:10.1016/j.arcped.2021.11.011

37. Ristic L, Dakovic D, Postic S, Lazic Z, Bacevic M, Vucevic D. Clinical Characteristics of Abutment Teeth with Gingival Discoloration. *J Prosthodont*. 2019;28(1):e45-e50. doi:10.1111/jopr.12612
38. Ilic DV, Brkovic SV, Postic SD. Overdenture in terms of preparation and restoration of supporting teeth. *Vojnosanitetski Pregled*. 2020;77(10):1086-1092. doi:10.2298/VSP1803230164I
39. Fornari V, Souza MA, Dallepiane FG, Pasqualotti A, de Conto F. Maxillofacial infections of dental origin: Risk factors for hospital admission. *Braz J Oral Sci*. 2024;23:e243442. doi:10.20396/bjos.v23i00.8673442
40. Ullah M, Irshad M, Yaacoub A, Carter E, Cox S. Hospitalisations Due to Dental Infection: A Retrospective Clinical Audit from an Australian Public Hospital. *Dent J (Basel)*. 2024;12(6):173. doi:10.3390/dj12060173
41. Lixandru CI, Maniu I, Cernușcă-Mițariu MM, Domnariu CD. Oral infections - a retrospective study of patients treated in the Oral and Maxillofacial Surgery clinic of the Emergency County Clinical Hospital in Sibiu. *Med Pharm Rep*. 2024;97(3):380-389. doi:10.15386/mpr-2759
42. Matta R. Sclerosing mediastinitis in a child: A case report. *The Egyptian Journal of Chest Diseases and Tuberculosis*. 2019;68(1):111-113. doi:10.4103/ejcdt.ejcdt_87_18
43. Pesce P, Lagazzo A, Barberis F, et al. Mechanical characterisation of multi vs. uni-directional carbon fiber frameworks for dental implant applications. *Mater Sci Eng C Mater Biol Appl*. 2019;102:186-191. doi:10.1016/j.msec.2019.04.036
44. Baldi D, Longobardi M, Cartiglia C, et al. Dental implants osteogenic properties evaluated by cDNA microarrays. *Implant Dent*. 2011;20(4):299-305. doi:10.1097/ID.0b013e318225f22b
45. Li P, Liu J, Liu J. Procalcitonin-guided antibiotic therapy for pediatrics with infective disease: A updated meta-analyses and trial sequential analysis. *Front Cell Infect Microbiol*. 2022;12:915463. doi:10.3389/fcimb.2022.915463
46. Postic SD. Effects of calcitonin and calcium medication in treatment of edentulous osteoporotic mandible. *Vojnosanit Pregl*. 2013;70(6):576-579. doi:10.2298/vsp1306576p