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Management of patients with respiratory disease in oral and maxillofacial surgery.

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Abstract

Pulmonary infections can compromise respiratory function and pose a risk during inhalation anaesthesia. Allergic reactions of the airway, particularly anaphylactic shock, can be induced by a variety of drugs and may constitute a medical emergency. Diseases of the lower tract are typically referred to as pulmonary diseases. Acute and chronic pulmonary infections are of concern to the dental team since some of these infections are transmissible in the dental office[1].Patients with COPD and asthma have difficulty breathing, particularly when reclined, and they also pose a risk during inhalation anaesthesia. This review article discuss the management of patients with different respiratory disease in oral and maxillofacial surgery speciality.

Keywords: anaphylactic, respiratory, pulmonary

Introduction

The respiratory system includes the nasal and oral cavities: the sinuses and larynx as the upper airway, and the trachea, bronchi, bronchioles, and alveoli as the

lower airway. The respiratory system is basically responsible for O2and CO2 exchange between the blood and the external environment. This gas exchange takes place passively across partial pressure gradients within the terminal respiratory units (alveolar spaces). Maintenance of the mentioned partial pressure gradients is essential for ensuring adequate pulmonary gas exchange[1].

Pulmonary disorders can be divided into three broad categories: obstructive diseases, restrictive diseases, and disorders of the pulmonary vascular system. Fortunately, these categories, based on patho physiology, are generally equally useful in identifying an appropriate clinical approach to the problem. The obstructive disorders include a cluster of pulmonary diseases called "chronic obstructive pulmonary disease" (COPD). This is not a single disorder, but rather a collective term for a group of disorders that share the mechanism of airflow obstruction, but have distinct mechanisms and distinct definitions. The three most common disorders included under the label COPD include chronic bronchitis, emphysema, and chronic persistent asthma[1].

Epidemiology

Respiratory diseases are leading causes of death and disability in the world. About 65 million people suffer from chronic obstructive pulmonary disease (COPD) and 3million die from it each year, making it the third leading cause of death worldwide. About 334 million people suffer from asthma, the most common chronic disease of childhood affecting 14% of all children globally.

Pneumonia kills millions of people annually and is a leading cause of death among children under 5 years old. Over 10 million people develop tuberculosis (TB) and 1.4 million die from it each year, making it the most common lethal infectious disease. Lung cancer kills 1.6 million people each year and is the deadliest cancer. Globally, 4 million people die prematurely from chronic respiratory disease. At least 2 billion people are exposed to indoor toxic smoke, 1 billion inhale outdoor pollutant air and 1 billion are exposed to tobacco smoke. The truth is that many of us are naïve to these stark realities[2].

Mechanism of infection

Two routes exist for oral micro-organisms to reach the lower respiratory tract: hematogenous spread and aspiration. Hematogenous spread of bacteria is an inevitable adverse effect of some dental treatments and may occur even after simple prophylactic procedures. Nonetheless, this route of infection seems rare, and only 2 well documented case reports could be found in the literature. In both cases hematogenous spread was the most likely source of pulmonary infection with periodontal anaerobes[3,4].

In contrast, aspiration of material from the upper airway occurs in 45% of healthy subjects during sleep and in 70% of subjects with impaired consciousness[5]. It is probably the main cause of nosocomial infection along with aspiration of gastric contents.

Patho-physiological considerations

The interaction between Anesthesia and the respiratory system creates some specific problems which can aggravate the patho physiology of pulmonary diseases. Foremost among them is the reduction in functional residual capacity that occurs with Anesthesia.

Functional residual capacity is the volume of air left in the lung at the end of expiration. It is the volume of air that keeps alveolar units open and functional, so that the next inspiration will deliver air in a uniform fashion, allowing good matching between perfusion and ventilation and thus good oxygenation[6].

Asthma is a disorder presenting as an increased responsiveness of the trachea bronchial tree to a variety

of stimuli. Broncho spasm and increased mucus secretion result in increased airway resistance and wheezing. By convention, asthma is categorized as extrinsic when attacks are precipitated by allergens, and intrinsic when neither specific allergens or elevated IgE can be identified[3].

Chronic Obstructive Pulmonary Disease (COPD) is a trilogy of disorders including chronic bronchitis, peripheral airways disease (bronchiolitis), and emphysema. The first two conditions differ anatomically but are clinically similar in that they are chronic inflammatory processes resulting in excessive mucus production and cough[5].

Asthma

Diagnosis of asthma is from the clinical history and presentation, based on recognizing a characteristic pattern of episodic symptoms in the absence of an alternative explanation. Investigations include a chest radiograph (to exclude other diagnoses, such as a pneumothorax), spirometry (serial PEFR), skin tests and blood examination (usually eosinophilia, raised total IgE and specific IgE antibody concentrations, which may help identify allergens)[7].

Management includes patient education, smoking cessation advice, avoidance of identifiable irritants and allergens, and use of drugs. Home use of peak flow meters allows patients to monitor progress and detect any deterioration that may require urgent modification of treatment. Treatment should be based on the amount by which peak flow is reduced (a PEFR diary should be kept). Drugs used for asthma management (TABLE 1) include oxygen, short-acting β2 agonists (SABAs; such as salbutamol), corticosteroids, leukotriene receptor antagonists and omalizumab (a recombinant humanized monoclonal anti-IgE antibody that reduces the antigen specific IgE).

Table 1: Medical management of asthma

Drug group	Examples	Comments
Beta-agonists	Selective β2-agonists or stimulants (e. g. salbutamol). Others include: Terbutaline, fenoterol, rimiterol Pirbuterol, reproterol, tulobuterol Bambuterol, salmeterol, formoterol	Safest and most effective bronchodilators for routine control of asthma, Relax bronchial smooth muscle with little cardiac effect Actfor3–6h Actforatleast12h
Antimuscarinic bronchodilators	Ipratropium or ox itr opium bromide	Useful particularly for those with asthma associated with bronchitis, Act for upto8h
Methylxanthines	Theophylline preparations (oral sustained release)	Prolonged action and useful for controlling nocturnal asthma
Corticosteroids	Corticosteroid (beclomethasone, betamethasone valerate, budesonide or fluticasone) aerosol inhalations	Effective in haled a long with a bronchodilat or but must be taken regularly High-dose corticosteroid in halant scan cause some adrenal suppression
Mast cell stabilizers	Sodium cromoglicateornedocromil	Occasional lyusedas in halant for pro phylaxis, mainly in children, but some Fail ores pond
Leukotriene recep antagonists tor	Montelukast, zafirlukast	May impair liver function and increase INR

5-Lipoxygenaseinhibitor (impairs Leuko triene release)	Zileuton	Given orally Effective when used alone or with in haled steroids but may precipitate, and Should not be used in Churg–Strausssyn drome, where deterioration and Cardia complications may be seen

Chronic oobstructive ppulmonary disease

Diagnosis of respiratory disorders is from the clinical features supported by imaging (especially chest radio graphy). Investigations include a chest radio graph (which may show hyper inflated lung fields with loss of vascular markings); arterial blood gases (which should be measured if pulse oximetry shows oxygen saturation less than 92%); spirometry; and lung function tests.FEV1 is reduced in all cases (FEV1 of less than 40% signifies severe COPD) and the flow–volume curve shows a typical pattern, with reduced flow rates at mid- and lower-lung volumes. A ratio of FEV1: FVC of less than 70% confirms airways obstruction[8].

Spiral computed tomography (CT) can now scan the lungs in a quick 20–30-second breath-hold and therefore, instead of producing a stack of individual CT slices, which may be misaligned due to patient movement or breathing in between slices, provides high-resolution three-dimensional images[8].

Arterial blood gas analysis yields considerable information about gas exchange efficiency. Arterial hypoxaemia in adults is defined as *Pa*O2 below 10.7kPa breathing room air, although it is not usually treated as clinically important unless below 8kPa, when oxygen saturation will be 90% or less.

Drug therapy includes short-acting bronchodilators (anticholinergic drugs [ipratropium bromide]) and *β*2 agonists (salbutamol) to treat the reversible component of airway disease; corticosteroids (inhaled or systemic); and antibiotics (amoxicillin, trimethoprim or tetracyc line). Mucolytics, such as carbocation, reduce acute exacerbations by almost one-third[9].

Inhaled therapy

Bronchodilators (short-acting β2 agonists [SABA] and short-acting muscarinic antagonists [SAMA]) should be the initial empirical treatment for the relief of breathlessness and exercise elimination. ICS have potential adverse effects (including non-fatal pneumonia) in people with COPD. Most patients – whatever their age – are able to acquire and maintain an adequate inhaler technique. Bronchodilators are usually best administered using a hand-held inhaler device (including a spacer device if appropriate)[10].

Oral therapy

some patients with advanced COPD may require maintenance oral corticosteroids when these cannot be withdrawn following an exacerbation. These individuals should be monitored for the development of osteoporosis and given appropriate prophylaxis. Theophylline should only be used after a trial of SABA and LABA, and only to those who are unable to use inhaled therapy, as there is a need to monitor plasma levels and interactions. The dose of theophylline prescribed should be reduced at the time of an exacerbation if macrolide or fluoroquinolone antibiotics (or other drugs known to interact) are given. There is insufficient evidence to recommend prophylactic antibiotic therapy in the management of stable COPD. Mucolytic drug therapy should be considered in patients with a chronic cough productive of sputum[10].

Oxygen Therapy

Long-term oxygen therapy (LTOT).

Inappropriate oxygen therapy in people with COPD may depress respiration. LTOT is indicated in patients with COPD who have aPaO2 of less than 7.3 kPa when stable, or a PaO2 greater than 7.3 kPa and less than 8

kPa when stable, and one of: secondary polycythaemia, nocturnal hypoxaemia (oxygen saturation of arterial blood [SaO2] of less than 90% for more than 30% of the time), peripheral oedema or pulmonary hypertension. To reap the benefits of LTOT, patients should breathe supplemental oxygen for at least 15 hours per day. To ensure that all those eligible for LTOT are identified, pulse oximetry should be available in all health care settings[11].

Ambulatory oxygen therapy

Ambulatory oxygen therapy should be considered in patients on LTOT who wish to continue oxygen therapy outside the home, and who have exercised saturation, are shown to have an improvement in exercise capacity and/or dyspnoea with oxygen, and are motivated to use oxygen[11].

Non-invasive ventilation (NIV).

Adequately treated patients with chronic hyper capnic respiratory failure who have required assisted ventilation during an exacerbation, or who are hyper capnic or acidotic on LTOT, should be referred to a specialist centre for consideration of long-term NIV. Advanced emphysema is occasionally treated with surgery– excision of large acquired bullae or, rarely, lung transplantation[11].

Tuberculosis

The diagnosis of TB is suggested by the history and confirmed by physical examination, a massively raised erythrocyte sedimentation rate (ESR), positive tuberculin skin tests (TSTs; Mantoux or Heaf test for a delayed hypersensitivity reaction to protein from M. tuberculosis [purified protein derivative; PPD]) and chest imaging. A positive Mantoux reaction indicates previous immunization (BCG; bacilli Calmette–Guerin–live attenuated M. bovis) or current infection– not necessarily disease. Computed tomography (CT) may show areas of calcification or high light a tuberculous abscess. Smears and culture of sputum, blood, laryngeal swabs, broncho alveolar lavage, gastric aspirates or pleural fluid may be tested for mycobacteria[12].

Active TB is diagnosed by sputum microscopy and culture in liquid medium with subsequent drug-susceptibility testing. Nucleic acid amplification tests, imaging, and his to pathological examination of biopsy samples help. IGRA and TSTs have no role in the diagnosis of active disease. A molecular diagnostic test now available in some high-income countries (Expert MTB/ RIF assay) detects M. tuberculosis complex within 2hours, with a higher assay sensitivity than that of smear micro scopy[12].

Treatment for ‘symptomatic sputum-positive’ patients, which should be instituted as soon as possible, is combination chemotherapy, usually isoniazid plus rifampicin plus pyrazinamide or ethambutol for 2 months, with continuation of daily isoniazid and rifampicin for a further 4 months[13]. Treatment for ‘asymptomatic’ patients who are believed to have been infected by contacts, but are not unwell, includes isoniazid for 6 months or isoniazid and rifampicin for 3 months. Rifapentine is a long-acting rifampicin used once weekly. Fluoroquinolones (moxifloxacin) may also act against TB. There may be resistance to one or more than one antibiotic[13] (Table 2)

Table 2: Antitubercular therapy

Drug	Use	Main adverse effects
Ethambutol	Initial therapy	Ocular damage
Isoniazid	Initial therapy, Continuation therapy	Peripheral neuropathy Hepatotoxicity
Pyrazinamide	Initial therapy	Hepatotoxicity
Rifampicin	Initial therapy Continuation therapy	EnhancedliverP450drugmetabolizingenzymesRed urine and saliva Bullous lesions Hepatotoxicity Nephrotoxicity
Streptomycin	Initial therapy	Vestibular nerve damage Circumoral paraesthesia

Prevention

Chronic obstructive ppulmonary disease

Discouraging individuals from starting to smoke tobacco and encouraging smokers to reduce and quit smoking are the first and most important priorities in preventing COPD. Chimney cook stoves and other devices that decrease indoor smoke exposure lessen the risk of respiratory infections in children and potentially the incidence of COPD in non-smokers, particularly in women. Childhood vaccines and prompt recognition and treatment of lower respiratory tract infections will minimize the airway injury that predisposes to COPD in adulthood. COPD may begin in childhood[14]. Management of childhood asthma, controlling occupational exposure to dust and fumes, and other environmental controls could have sub stantial benefits in reducing the burden of COPD[14].

Asthma

The cause of most asthma is unknown and there is no effective strategy for primary prevention. However, potentially modifiable risk factors for development of asthma include smoking during pregnancy and use of broad-spectrum antibiotics in the first year of life. Asthmatics who smoke have a more rapid decline in lung function than lifelong non-smokers. Avoiding smoking during pregnancy and avoidance of passive smoke exposure after birth can reduce asthma severity in children. There is little evidence for effective single-strategy indoor allergen avoidance interventions in adults outside the occupational context, except for remediation of dampness and mould[15].

Tuberculosis

The factors promoting the spread of infection relate to the chance that an uninfected individual is exposed to a person with infectious TB: the more cases in the community, the more likely it is that an individual will become infected. Inhaling only a few tuberculous bacteria can result in infection. However, only about one in 10 people infected with Mycobacterium tuberculosis will develop active disease, although the rate is much higher in young children and people with immuno deficiency conditions. TB lies dormant because the infection is contained by the body's immune system, but it can become active at any point in a person's lifetime[16]. This two-phase sequence by which the disease develops provides an opportunity for prevention. By identifying persons who are proven or are highly likely to have latent infection and treating those who have conditions or circumstances that increase the risk of disease, the likelihood of developing active TB can be substantially reduced. Several drug regimens have been documented to be effective for treating latent tuberculosis. The current vaccine, Bacille–Calmette–Guerin (BCG), offers only partial protection against TB but does reduce the risk of disseminated TB and tuberculous meningitis in children. Research centres around the world are working on developing a better vaccine for TB[16].

Conclusion

Respiratory diseases are an enormous challenge to life, health and productive human activity. Prevention, control and cure of these diseases and promotion of respiratory health must be a top priority in global decision making in the health sector. The control, prevention and cure of respiratory diseases are among the most cost-effective health interventions available–a "best-buy" in the view of the WHO.

Many dental and Maxillofacial patients have obstructive pulmonary diseases, such as chronic bronchitis, emphysema, and bronchial asthma. These diseases have different etio logies but may have overlapping signs and symptoms. Diagnosis and treatment are becoming more sophisticated so that morbidity and mortality are improving. The dental and Maxillofacial profession almost know these diseases in order to be able to offer effective and safe treatment, and must be able to recognize the oral and/or dental manifestations that might arise. Cigarette smoking is a common risk factor that dental health care providers can address with a simple cessation program called the fiveA's. Perioperative management of the patient with pulmonary disease requires an appropriate preoperative assessment. This allows the patient to be optimized for both aesthetic and surgical intervention. Patients must be diligently monitored and observed throughout the perioperative period to identify and provide early intervention to optimize outcome and avoid potential adverse events[17].

Finally, research in respiratory diseases is the hope for today and the promise for tomorrow. Research must answer many questions: how do lung diseases arise, how do they are spread, who is vulnerable, and what actions can be used control or cure them, to name a few. It also helps us understand what keeps people healthy.

References

1. Becker DE. Management of respiratory complications in clinical dental practice. Patho physio logical and technical considerations. Anesth Prog. 1990; 37 (4) :169-175.
2. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD) and asthma. Am Rev Respir Dis.1987;136 (1):225-244.
3. Christensen PJ, Kutty K, Adlam RT, Taft TA, Kamp's chroer BH. Septic pulmonary embolism due to periodontal disease. Chest.1993;104(6):1927-1929.
4. Morris JF, Sewell DL. Necrotizing pneumonia caused by mixed infection with Actino bacillus actino my cetecomitans and Actinomyces is raelii: case report and review. Clin Infect Dis. 1994;18(3):450-452.
5. Huxley EJ, Viroslav J, Gray WR, Pierce AK. Pharyngeal aspiration in normal adult sand patients with depressed consciousness. Am JMed.1978;64(4):564-568.
6. Centers for Disease Control. MMWR. 2012; 61 (46): 938.
7. Wagena EJ, van der Meer RM, Ostelo RJ, et al. The efficacy of smoking cessation strategies in people with chronic obstructive pulmonary disease: results Roma systematic review. Respir Med. 2004; 98:805-815.
8. Diagnosing chronic obstructive pulmonary disease. BMJ 2015; 351:h6171
9. Russell SL, Boylan RJ, Ka slick RS, Scan napieco FA, Katz RV. Respiratory pathogen colonization of the dental plaque of institutionalize delders. Spec Care Dentist. 1999; 19 (3):128-134.
10. Ter penning M, Bretz W, Lopatin D, Lang more S, Dominguez B, Loesche W. Bacterial colonization of saliva and plaque in the elderly. CLIN In fact Dis.1993; 16 Suppl4: S314-S316.
11. Theilade E, Budtz-Jørgensen E. Predominant cultivable micro flora of plaque on removable dentures

in patients with denture-induced stomatitis. Oral Micro bio Immunol. 1988; 3 (1):8-13.

12. Scannapieco FA, Stewart EM, My lotte JM. Colonization of dental plaque by respiratory pathogens in medical intensive care patients. Crit Care Med. 1992; 20 (6):740-745.

13. Buhl R, Farmer SG. Current and future pharmacologic therapy of exacerbations in chronic obstructive pulmonary disease and asthma. Proc Am Thorac Soc.2004; 1:136-142.

14. Tomar SL. Dentistry's role in to bacco control. J Am Dent Assoc.2001; 132Suppl:30S-35S

15. Stewart RD, Kaplan R, Pennock B, Thompson F. Influence of mask design on bag-mask ventilation. Ann Emerg Med. 1985;14(5):403-406.

16. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD) and asthma. This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, November 1986. Am Rev Respir Dis.1987;136(1):225-244.

17. Bryant E. Chronic disease management for patients with respiratory disease. Nurs Times. 2005; 101 (25):46-48.