Negative pressure pulmonary edema (Review)

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Abstract. Negative pressure pulmonary edema (NPPE) is a complication resulting from acute or chronic upper airway obstruction, often posing challenges in recognition and diagnosis for clinicians. If left untreated, NPPE can lead to hypoxemia, heart failure and even shock. Furthermore, the drug treatment of NPPE remains a subject of controversy. The primary pathophysiological mechanism of NPPE involves the need for high inspiratory pressure to counteract upper airway obstruction, subsequently causing a progressive rise in negative pressure within the pleural cavity. Consequently, this results in increased pulmonary microvascular pressure, leading to the infiltration of pulmonary capillary fluid into the alveoli. NPPE exhibits numerous risk factors and causes, with laryngospasm following anesthesia and extubation being the most prevalent. The diagnosis of NPPE often presents challenges due to confusion with conditions such as gastroesophageal reflux or cardiogenic pulmonary edema, given the similarity in initial factors triggering both diseases. Upper airway patency, positive pressure non-invasive ventilation, supplemental oxygen and re-intubation mechanical ventilation are the foundation of the treatment of NPPE. The present review aims to discuss the etiology, clinical presentation, pathophysiology and management of NPPE.

Contents

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1. Introduction

Negative pressure pulmonary edema (NPPE) is a noncardiogenic pulmonary edema caused by a rapid increase in negative intrathoracic pressure. It can occur due to acute or chronic upper airway obstruction, which can lead to life-threatening hypoxemia (1); upper airway obstruction may be caused by a number of potential factors, including laryngeal spasm, foreign bodies and tracheal intubation, resulting in breathing difficulties. Negative pressure in the capillaries occurs when the patient breathes, causing transudative fluid from the pulmonary capillaries to seep into the alveolar and interstitial tissues and leading to alveolar and interstitial edema (1-6). The relationship between upper airway obstruction and pulmonary edema was first described in 1927 by Moore and Binger (7). However, the first clinical case was not reported until 1973 (2). Since then, NPPE has been reported multiple times with varying incidence statistics. In a study of 176 children with severe upper airway obstruction, the incidence rate of NPPE was 9.6% (8). In another study, the incidence rate of NPPE in patients with acute upper airway obstruction was reported to be as high as 12% (9). From the statistics of the Australian Incident Monitoring Study (AIMS), the reported incidence rate of NPPE in patients with laryngospasm was 3% (10). The incidence rate of NPPE has also been reported to be as low as 0.1% in laryngospasm cases (11). Thus, the estimated incidence rate of NPPE is between 0.1 and 12% (9,12-14). However, considering the frequent occurrence of upper airway obstruction during the peri-anesthesia period, it was speculated that the actual incidence rate may be much higher than that reported so far, since several cases are misdiagnosed or overlooked (1). Reports on mortality also vary. The mortality rate of NPPE has previously been described as 11 to 40%, with a more recent literature review showing a mortality rate of only 2% (15-25). A recent systematic review of NPPE in adult ear, nose and throat (ENT) surgery reported a mortality rate of 5% and identified age and ICU admission as the main risks for increased mortality (3). Due to the high incidence and misdiagnosis rate of NPPE, further improvements in the level

of diagnosis, treatment and management of NPPE are expected in clinical practice. The present review aims to summarize the latest advances in the epidemiology, pathophysiology, clinical manifestations and treatment of NPPE.

2. Etiology

Several acute or chronic diseases can cause upper airway obstruction and each of them could lead to NPPE (26). Upper airway trauma and, more commonly, laryngospasm account for ~50% of NPPE after obstruction (4). In addition, upper airway infections and vocal cord dysfunction are also potential causes of NPPE (1,4).

Acute airway obstruction. In adult patients, airway obstruction leading to NPPE occurs most commonly in the event of a post-extubation laryngospasm (4,27). Most cases in children are subglottic obstructions caused by the glottis, acute infectious croup or epiglottitis (28,29). In these pediatric cases, patients who present with ventilatory failure due to glottic or supraglottic obstruction with prolonged stridor (30) and pulmonary edema are usually diagnosed after the initiation of mechanical ventilation.

Chronic airway obstruction. Chronic upper airway obstruction is a prevalent condition observed in patients with various underlying factors, including obesity, obstructive sleep apnea, tonsil or gland hypertrophy, upper airway tumor, mediastinal tumor, nasopharyngeal mass, goiter and acromegaly (1,31-34). In particular, adult patients with obesity and obstructive sleep apnea, characterized by episodes of hypopnea and hypoxemia, have demonstrated a propensity for intermittent pulmonary edema stemming from recurrent upper airway obstruction (3).

Other risk factors. Several studies have reviewed cases that included risk factors for perianaesthesia-induced NPPE, such as obesity with obstructive sleep apnea and anatomical intubation difficulties, as well as nasal, pharyngeal and laryngeal surgery or disease (35-37). Patients with the aforementioned risk factors should preferably undergo awake intubation, as they may be at increased risk of airway complications following extubation performed after general anesthesia; therefore, optimal upper airway muscle tone is ensured in these patients (36). These patients should be closely observed in the post-anesthesia care unit after surgery. The duration of the observation varies depending on the specific individual (36).

3. Pathogenesis and pathophysiological features

The occurrence of NPPE is the result of a combination of factors. There are two main pathogenic mechanisms in NPPE: Hydrostatic and increased permeability pulmonary edema. The mechanism behind hydrostatic pulmonary edema suggests that NPPE is caused by marked fluid shifts triggered by changes in the intrathoracic pressure (Fig. 1) (1,4,38). The mechanism behind increased permeability pulmonary edema suggests that the destruction of alveolar epithelium and pulmonary microvascular membranes caused by severe mechanical stress results in increased pulmonary capillary permeability and protein-rich pulmonary edema (26,38). Pulmonary edema fluid/plasma protein ratio measurement is routinely used to distinguish hydrostatic pulmonary edema from increased permeability pulmonary edema, since the ability of the alveolar epithelial barrier to filter out the alveolar edema fluid is usually impaired in patients with acute lung injury but not in patients with hydrostatic pulmonary edema (26). For example, Fremont *et al* (26) collected edema fluid and plasma samples from 10 patients with NPPE during the first 8 h after intubation. The mean pulmonary edema fluid/plasma protein ratio in these patients was 0.54±0.15, and the mean rate of alveolar fluid clearance within 8 h was 14.0±17.4%/h (26). The pulmonary edema fluid/plasma protein ratio and the presence of net alveolar fluid clearance support the mechanism of hydrostatic pulmonary edema.

Starling principle. Starling's principle is an important theory that describes the behavior of the fluid passing through the pulmonary capillary endothelial-alveolar wall barrier (1). The Starling equation includes factors that determine the formation of NPPE: $Q_f=[Kx(P_{mv}-P_i)]-[\sigma x(\pi_{mv}-\pi_i)]$, where Q_f is the net fluid flux between capillary membranes, K is the coefficient of capillary permeability, P_{mv} is the hydrostatic pressure of the capillary membrane, P_i is the hydrostatic pressure of the alveolar interstitium, σ is the reflection coefficient (that is, the ability of a membrane to block protein passage), π_{mv} is the osmotic pressure of microvascular proteins, and π_i is the interstitial protein osmotic pressure. In physiologic conditions, when the hydrostatic pressure is at equilibrium, the primary plasma fluid flow from the capillaries to the lung interstitium is minimal and the fluid is then reabsorbed by the lymphatic vessels. However, when the rate of interstitial fluid formation is higher than the reabsorption capacity, edema forms (1,4,39).

Müller's maneuver. Müller's maneuver is a forced attempt to inhale after the glottis has been closed. This action can markedly increase the negative pressure in the pleural cavity (1). The pressure around the pulmonary blood vessels and in the pulmonary interstitium can also be markedly reduced through pressure transmission to the surrounding structures (1). In healthy individuals, the range of intrathoracic pressure is between -8 and 4 cmH_2O at the end of calm inhalation and between -4 and 2 cmH_2O at the end of exhalation. However, when the airway is obstructed, a strong inspiration can generate negative intrathoracic pressure as low as -50 to -100 cmH_2O (1,23). Strong inspiratory efforts may generate even more negative pressures (-140 cmH_2O) (23). Although the exact threshold of negative intrathoracic pressure to induce pulmonary edema remains unclear, it is hypothesized that there may be individual differences, since the parameters of each individual in the Starling equation are different (1).

Ventricular preload and afterload. The decrease in intrathoracic pressure is transmitted to the pulmonary interstitium, eventually causing an increase in venous blood flow back to the right atrium (40). The increased workload in the right side of the heart results in an increase in pulmonary blood volume, pulmonary capillary hydrostatic pressure and hydrostatic pressure gradient across the capillaries (17). This promotes the formation of pulmonary edema. By contrast, a decrease in intrathoracic pressure can increase left ventricular (LV)

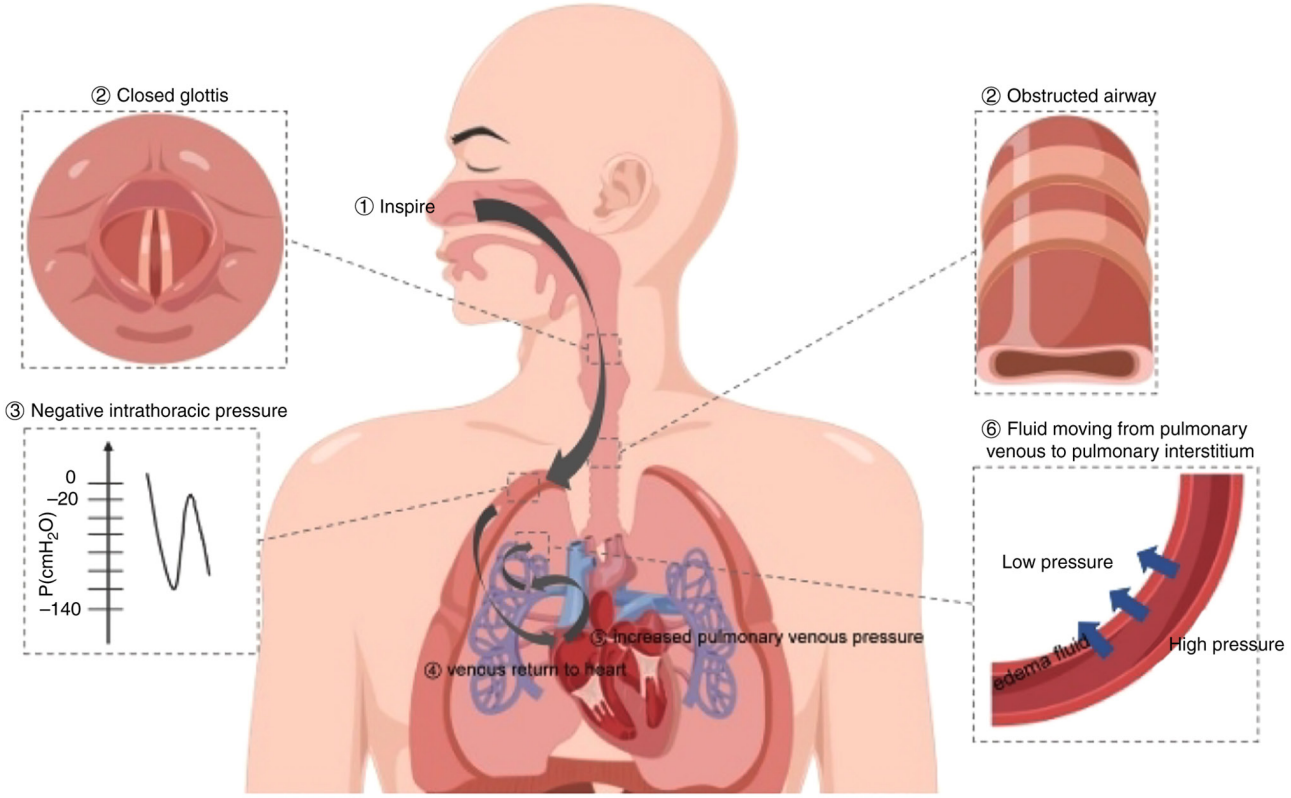

Figure 1. Pathogenesis of NPPE. NPPE occurs when there is an obstruction or partial obstruction in the upper airway. Forceful inspiratory efforts against the obstruction create negative pressure within the thoracic cavity, increasing venous return and left ventricular preload. The elevated pressure in the pulmonary veins causes fluid to leak from the capillaries into the lungs, leading to pulmonary edema. Numbers denote the order of events. NPPE, negative pressure pulmonary edema.

afterload, increasing both LV end-diastolic volume and pressure (17). This reduces the LV ejection volume and ejection fraction, ultimately leading to increased pulmonary microvascular pressure (17).

Interventricular association. Increased venous return leads to increased filling of the right ventricle, compressing the interventricular septum toward the left ventricle. This results in a decrease in LV volume and compliance (an 'interventricular correlation'), which ultimately leads to an increase in LV end-diastolic pressure. Negative intrathoracic pressure increases the pressure gradient between the left ventricle and the aorta, thereby increasing LV afterload (4).

Pulmonary capillary permeability. It is commonly considered that the permeability of pulmonary capillaries increases under the condition of negative thoracic pressure (41). In general, when pulmonary vascular compliance is low, an increase in pulmonary blood volume can alter membrane permeability and even disrupt membrane integrity, which ultimately leads to leakage of body fluids and blood cells into the alveoli (20,41). This damaging effect has been demonstrated both experimentally and clinically (1,42,43).

Hypoxemia and hyperadrenergic states. Hypoxemic and hyperadrenergic states that accompany upper airway obstruction can also contribute to the development of pulmonary edema (44,45). The hyperadrenergic state is induced by upper airway obstruction alone and is enhanced by hypoxemia. Hypoxia and metabolic acidosis (occurring during hypoxia due to anaerobic metabolism, impaired circulation or impaired oxygen utilization) lead to vasoconstriction in the pulmonary microcirculation, which increases pulmonary circulatory resistance, thus altering pulmonary capillary membrane permeability and inhibiting myocardial function (46). Increased excitability of the sympathetic nervous system results in the centralization of the circulation and the movement of systemic blood toward the pulmonary circulation, thereby increasing pulmonary microvascular pressure (46).

The hyperadrenergic state can also alter the integrity of the pulmonary capillary membranes (1). In addition, autonomic hyperexcitability promotes the development of neurogenic pulmonary edema, a condition characterized by the accumulation of fluid in the lungs due to dysfunction or injury of the central nervous system, occurring as a result of various neurological conditions or insults, such as traumatic brain injury, intracranial hemorrhage, seizures, brain tumors, or infections affecting the central nervous system (1). Increased pulmonary capillary membrane permeability and pulmonary microvascular pressure, which result from the strong release of catecholamines from the adrenal medulla, are currently considered to be the main pathogenic mechanisms of pulmonary edema (19).

4. Clinical manifestations

Symptoms. Notably, patients may present with predisposing factors for developing upper airway obstruction. Symptoms

of upper airway obstruction include stridor, respiratory distress, paradoxical chest movements and the involvement of accessory muscles in breathing (36,41). Clinical manifestations of acute pulmonary edema include dyspnea, tachypnea, cyanosis, asthma and the production of a profuse pink foamy sputum (1). Consequently, medical complications, including hypoxemia, hypercapnia and metabolic acidosis, may occur as a consequence of the underlying respiratory dysfunction and disturbed gas exchange in acute pulmonary edema (1).

Disease progression. Cases of delayed onset are rare, but pulmonary edema has been reported to occur 1-6 h after upper airway obstruction (44,47,48). Since the severity of clinical manifestations is variable, some mild cases may not be recognized. The severity of the condition is related to the duration of the obstruction and the degree of pulmonary capillary damage (3,46).

It has been suggested that the observation period following extubation should be extended in patients with pre-existing or predisposing factors for upper airway obstruction, even if clinical manifestations of pulmonary edema have not been observed (44,46). For several patients, pulse oximetry monitoring in the operating room and post-anesthesia care can discern any risk factors (1). The symptoms and signs of NPPE are atypical. Occasionally, the clinical manifestation may only cause a slight decrease in pulse oximetry. This may be more evident when the patient is breathing room air and, in some cases, only chest imaging can identify the risk for NPPE after the examination (1).

Symptoms subside. Once the airway is protected, the negative airway pressure is relieved by positive-pressure mechanical ventilation (1,26). Imaging analysis of alveolar or interstitial edema has indicated that NPPE usually resolves within 12-24 h (1). Occasionally, some cases take longer for the NPPE to resolve, which is related to the degree of pulmonary microvascular damage. In most cases, NPPE resolves within 12-24 h (26).

Consequences of NPPE. Some patients with NPPE may suffer from associated long-term complications, such as myocardial infarction, transient ischemic attack, non-ST-elevation myocardial infarction, hypoxic brain injury and pulmonary hemorrhage. A small number of patients may die from causes such as septic shock and cardiac arrest (3).

5. Differential diagnosis

Clinically, it is difficult to diagnose NPPE in patients with mild symptoms such as mild respiratory distress, a mild decrease in oxygen saturation, a mild cough or cough with frothy sputum or mild chest discomfort. The diagnosis can often be made when the upper airway obstruction is relieved, but the condition is not yet completely resolved (1,46). NPPE should be differentiated from the diseases described below, as their treatment differs from that of NPPE.

Aspiration of gastric contents. If NPPE is excluded, aspiration of gastric contents and subsequent pneumonia should be considered first. Aspiration pneumonia is occasionally self-limiting with only the use of supportive care, such as oxygen therapy, fluid and nutritional support, symptom management, respiratory support, and monitoring and observation, depending on the quantity and quality of the inhaled material, such as pH and type of particulate matter inhaled. Direct laryngoscopy or fiberoptic bronchoscopy can help confirm the diagnosis of particulate aspiration (5,49). Chest X-ray imaging does not help to distinguish between pulmonary edema and aspiration pneumonia (49).

Acute respiratory distress syndrome (ARDS). Based on the risk factors of the patient, ARDS must be excluded. ARDS is a severe lung condition characterized by sudden and widespread inflammation in the lungs, leading to impaired oxygenation and difficulty in breathing. While both ARDS and NPPE involve impaired oxygenation and respiratory distress, their underlying mechanisms differ. ARDS is primarily caused by inflammation and injury to the alveoli (air sacs) and lung tissue, resulting in increased permeability of the lung's blood vessels and leakage of fluid into the airspaces. By contrast, NPPE is caused by negative pressure in the lungs due to upper airway obstruction, leading to fluid accumulation and pulmonary edema. Rapid clinical improvement also helps to confirm the diagnosis of post-obstructive NPPE (1).

Hypervolemia. A measurement of the central venous pressure and pulmonary capillary wedge pressure can be conducted to account for the possibility of circulating hypervolemia. Treatments include limiting fluid volume (ensuring that fluid intake is balanced or reduced compared to fluid output), dilating blood vessels and using diuretics.

Abnormal cardiac function. A diagnosis of cardiac insufficiency must be excluded, especially in patients with a prior history of cardiac disease or significant cardiac risk factors, such as hypertension, diabetes, obesity and age. These cardiac insufficiencies include myocardial ischemia, arrhythmia and cardiac decompensation caused by valvular heart disease. Imaging aids can be used, with NPPE often showing prominent bilateral perihilar alveolar infiltrates (5). Cardiogenic pulmonary edema infiltrates follow a more interstitial pattern and typically show marked shunting of blood flow to the lungs (5,11).

Coronavirus disease 2019 (COVID-19). Sudden respiratory failure and bilateral pulmonary patchy infiltrates are the most common manifestations of NPPE. The clinical diagnosis of NPPE may be confused with that of COVID-19. Both NPPE and COVID-19 cause ground-glass opacities on chest CT scans (50). The characteristic changes of NPPE mainly appear in the central area, whereas COVID-19 mainly manifests in the peripheral area of the lungs (50). In addition, NPPE results in decreased vascular clarity, whereas COVID-19 causes vasodilation in the lesion area (1,51). These differences, along with the medical history of the patient, are critical to distinguish between these two similar imaging findings. Clinicians must understand the differences between COVID-19 and NPPE to ensure proper diagnosis and treatment (50).

6. Treatments

Treatment of NPPE depends on its severity and cause. In mild cases, oxygen therapy alone may be sufficient. With proper

diagnosis and treatment, patients can be expected to recover from NPPE within 24 h.

Ventilatory support. The primary method for treating NPPE is to maintain airway patency and provide oxygen support. Invasive tests are generally not required when considering the diagnosis of NPPE, but close observation in the intensive care unit is required until the condition is stable. Endotracheal intubation is usually required to open the airway and support mechanical ventilation. Among the cases reported by Lang *et al* (44), 85% of adult patients and children required intubation, 50% required mechanical ventilation and 50% required continuous positive end-expiratory pressure (PEEP) ventilation. Initial attempts were made to maintain airway patency and oxygen without intubation. Some patients required only oxygen support, only continuous PEEP with a mask or both (1). Patients with severe pulmonary edema often required reintubation in the operating room or post-anesthesia care unit. Most patients treated with mechanical ventilation were able to resolve pulmonary edema and extubation within 24 h. For those patients treated with mechanical ventilation, PEEP could improve oxygenation and reduce the required oxygen concentration (1).

It can be challenging to perform PEEP if the upper airway obstruction makes endotracheal intubation difficult (52,53). Difficulties with endotracheal intubation can also be encountered in patients with acute epiglottitis. In these cases, clinicians need to be prepared to obtain adequate rapid airway access via cricothyroidotomy or tracheostomy (52,53).

Fluid management. Diuretics and steroids are often used as adjunctive agents in the treatment of cardiogenic pulmonary edema to promote fluid clearance (54,55). However, the use of some diuretics, such as furosemide, is controversial (56). Some studies suggested that diuretics are only useful in patients with ARDS and their effect in other patients is limited (1,4,45). In the past, fluid intake restriction and the use of diuretics were not recommended for patients who were diagnosed with NPPE (4,45,57). Since these patients are deficient in effective circulating blood volume owing to the transfer of large amounts of fluid to the lungs, this may aggravate their present state (57).

Other treatment measures. Invasive hemodynamic monitoring is usually not required but it can be helpful in differential diagnosis (58). Adrenocorticoid therapy was used in the past but this approach is not currently supported or recommended, as it does not directly address the underlying mechanism of NPPE (14,42). The use of bronchodilators has also been studied and, although the obstruction may not be caused by bronchospasm, these studies suggested that bronchodilators, such as β-agonists, could increase alveolar fluid clearance to reduce symptoms of pulmonary edema (1,59). As aforementioned, patients who have developed upper airway obstruction or have predisposing factors, but have not yet exhibited pulmonary edema, require close observation in the post-anesthesia care unit, although the duration of observation remains controversial (1). Previously, Grant *et al* (60) reported the case of a 26-year-old female patient with laryngeal papillomatosis who developed laryngospasm after direct laryngoscopy. The patient developed severe NPPE, for which mechanical ventilator support was ineffective. The patient was successfully treated with venovenous extracorporeal membrane oxygenation.

Prevention. To reduce or eliminate NPPE, several prevention strategies have been investigated. Oropharyngeal secretions of patients should be thoroughly aspirated before extubation, as bloody secretions may induce laryngospasm (61,62). An increased number of laryngoscopy attempts is associated with an increased incidence of laryngospasm (63). A dose of 5 mg of dexamethasone may be used before extubation to reduce laryngeal edema caused by multiple intubation attempts (64,65). This effect is associated with a reduction in the frequency of laryngospasms by deepening anesthesia and enhancing muscle relaxation (66). A dose of 1-2 mg/kg of lidocaine 5 min before tracheal extubation has been reported to reduce laryngospasm in children (67,68). Propofol at a dose of 0.5 mg/kg administered 60 sec before extubation was also effective in reducing the incidence of laryngospasm (66,69). In addition, the cuff leak test could help prevent the risk of post-extubation edema (62). The cuff leak test is based on the principle that air leaks around the tracheal tube where the cuff is deflated will be inversely proportional to the degree of laryngeal obstruction resulting from laryngeal edema (70). Extubation may be successful if air leaks can be heard when the patient coughs during PEEP (70).

7. Conclusion

NPPE is a rare but potentially fatal complication of upper airway obstruction; patients with atypical symptoms are often misdiagnosed. NPPE should be considered in patients presenting with symptoms of pulmonary edema following upper airway obstruction, after excluding other contributing factors. The treatment of NPPE includes close monitoring, prompt relief of airway obstruction, administration of supplemental oxygen and, if necessary, assisted ventilation. Due to interindividual variations, the epidemiology, etiology and pathophysiological processes of NPPE have been subjects of controversy and pose ongoing challenges.

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Authors' contributions

JM, TL, QW and HY conceived, designed and coordinated the study. TL, QW, JM, HY, ZG, QF, YZ and XX drafted this manuscript. All authors substantially contributed to the conception, writing and revision of the work. All authors

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Ethics approval and consent to participate

Not applicable.

Patient consent for publication

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Competing interests

The authors declare that they have no competing interests.

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