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Review

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Management of spontaneous pneumothorax in children

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ABSTRACT

Primary spontaneous pneumothorax (PSP) is defined as air or gas accumulation in the pleural cavity. It can occur spontaneously or secondary to an underlying pulmonary disease or after a trauma. Spontaneous PSP has been traditionally categorized as primary or secondary. Primary spontaneous PSP affects commonly young healthy people especially tall and thin adolescent males. Secondary cases may occur in patients with underlying pulmonary disease. The main aim in the treatment of PSP is to empty the air in the pleural cavity and allow the lung to re-expand. There are two main approaches for the treatment of spontaneous PSP; non-operative management options include bed rest, oxygen supplementation with watch-and-wait option, needle aspiration and surgical management options ranges from chest tube insertion to more invasive intervention such as lung resection, pleurodesis or bullae excision via thoracoscopy or thoracotomy. There is still a lack of consensus in the diagnostic approach and treatment strategies in PSP in children. Therefore, we aimed to review the clinical findings, radiological imagining, treatment options and surgical strategies in children with PSP.

Keywords: Spontaneous pneumothorax, diagnose, surgery, children

INTRODUCTION

Primary spontaneous pneumothorax (PSP) is collection of air in the pleural cavity, between the lung and the chest wall and it was firstly described in 1891 by Laennec.[1] At that time, most of the cases with PSP were secondary to tuberculosis and some others occur in healthy patients with no underlying disease. The first description of PSP in healthy people was described by Kjaergaard[2] in 1932.

The mechanism of air collection in the pleural space may occur due to communication between the alveolar spaces and the pleura, direct or indirect communication between the atmosphere and the pleural space and presence of gas-producing organisms in the pleural space.[3]

It can be categorized as spontaneous, iatrogenic, traumatic or tension PSP. Spontaneous PSP has been traditionally classified as primer or seconder. Primer spontaneous PSP is defined as PSP occurring in patients without lung disorders, whereas secondary spontaneous PSP occurs in the setting of an underlying pulmonary disease.

The incidence of PSP is reported to be 7 to 18 per 100 000 population per year in males and 1 to 6 per year in females.[4] Spontaneous PSP is a relatively rare condition in children. The peak age of occurrence in this age group is bi-modal and most of the cases occur either in the neonatal period or in late adolescence.[5] It tends to occur in adolescence with a predominant male occurrence.[6]

The most important risk factor for PSP is smoking.[7] Cannabis also increases the risk of PSP and multiple large peripheral bullae at the lung apex are often seen in young patients with a history of cannabis smoking without any significant parenchymal damage.[7]

In addition to smoking, levels of air pollution, pleural porosity, changes in atmospheric pressure or exposure to loud music are also involved in the etiology of PSP.[8,9]

PSP usually occurs at rest; it presents with acute onset of local pleuritic chest pain accompanied by shortness of breath and may also be occur due to increased intrathoracic pressure. Procedures for the treatment of spontaneous PSP include observation, needle aspiration, insertion of intercostal catheter or invasive surgery procedures such as thoracotomy or thoracoscopy. Patients with prolonged chest tube drainage may require surgical intervention to remove the bullae or blebs and to create a pleural adhesion to avoid recurrences. There is no consensus on the timing and type of surgical intervention and best method for pleurodesis to prevent recurrence. Clinical

guidelines and consensus reports have been developed for adult patients but controversy still exists for the diagnosis and treatment of PSP in children. Herein, it is aimed to review the existing multidisciplinary approach to PSP including the clinical evaluation, radiological imagining, treatment options and surgical strategies.

PATHOPHYSIOLOGY

The pathogenesis of PSP is not completely understood. For many years the accepted theory for PSP is that the subpleural bleb or bulla opens spontaneously and begins to leak air.[10] However, this theory has been challenged recently because blebs and bullae do not always the cause of PSP. Bullae are air filled spaces larger than 1 cm in diameter which located distal to terminal bronchioles and are associated with pulmonary tissue damage.[11] Blebs are localized collections of air between the lamina elastica interna and externa of the visceral pleura. Both of these parenchymal abnormalities can be described the term 'emphysema-like changes' (ELCs).[11] There are areas of weakness of the visceral pleura which are prone to rupture allowing air to leak into the pleural space.

PSP occurs in patients with no associated lung disease, which does not mean that there is no underlying pathological process. Abnormal pleura is very common in PSP patients.[3] The macroscopic appearance of the visceral pleura has been shown to be abnormal in patients with PSP. It has been reported that in 77% of patients with PSP having blebs or bullae seen at thoracoscopy at the first episode.[12] Chronic distal airway inflammation with lymphocyte and macrophage infiltration alongside fibrotic changes are shown in the microscopic appearance of areas of lung tissue resected from patients with PSP.[13]

PSP usually occurs in tall, thin males, between 10 to 30 years-of-age. The gradient of negative pleural pressure increases from the lung base to the apex, so that alveoli at the lung apex is significantly greater distending pressure than those at the base of the lung so this theory can explain the development of apical subpleural blebs in tall individuals.[14]

Some inherited disorders may predispose to PSP, such as Marfan's syndrome, Birt-Hogg Dube syndrome, other mutations of the folliculin gene (FLCN), alfa1-antitrypsin deficiency and homocystinuria and approximately 10% of patients with PSP have a positive family history.[15] Female patients with these syndromes, pregnancy and childbirth pose a risk for PSP and women should be particularly informed for the risk of PSP development.[16]

Any patient presenting with first PSP had familial syndrome should have CT scan of the chest and a referral genetic test should be considered.[16]

The accumulation of neutrophils and macrophages leads to an inflammation by disrupting elastin fibers in smoking patients so that smoking is a major risk factor for PSP. Smoking cessation is the only reversible risk factor known to reduce the likelihood of recurrence.[7] The cause of the inflammation seen without a history of smoking is still unexplained in PSP patients.

In addition to enviromental factors, structural alterations may play role in the development of PSP. It has been reported that patients with PSP have emphysematous-like histology and may develop bullae and blebs.[6] Specific microRNAs (miRNA) may play critical role in inflammation and regulation of eosinophil activation. Previous reports showed that up-regulation of miRNA-24 expression may relate with fibrosis and chronic alterations in the airways of children with PSP.[17] The miRNA-24 expression in the exhaled breath condensate (EBC) of children with PSP significantly down-regulated and suggested that patients especially with bullous disease, should be closely monitored in the long-term period.[18]

SSP is commonly associated with chronic obstructive pulmonary disease but also with other underlying important lung disease, including cystic fibrosis, tuberculosis, diffuse parenchymal lung disease.[19]

Tension PSP is more common with traumatic reasons or with ventilated patients and extremely rare in spontaneous PSP. It occurs when the intra-pleural pressure becomes greater than atmospheric pressure and is caused by one-way valve effect whereby air passes into the pleural space on inspiration. When the pressure becomes negative, air in the airway unable to escape on expiration due to closing of the valve. It is an emergency situation when compared to other pneumothoraces because the intrathoracic pressure gradually increases and requires rapid intervention.

CLINICAL EVALUATION AND IMAGING

Sudden onset of chest pain, difficulty breathing and tachycardia are the predominant symptoms of PSP in children as in adults. The typical symptoms of chest pain and dyspnea may be relatively minor or even absent, so that a high index of suspicion is required. In general, the clinical symptoms associated with SSP are more severe than those associated with PSP.

The physical signs of a PSP include reduced lung expansion and breath sounds on the side of the PSP. In addition to these signs, cyanosis, sweating, severe tachypnoea, tachycardia and hypotension can be seen presence of tension PSP.

Posterior-anterior chest radiography is the standard examination in patients with suspected PSP. It has been reported that inspiratory and expiratory radiographs are equally sensitive in detecting PSP and therefore expiratory radiographs do not need to be taken routinely.[20] Besides these techniques, computed tomography (CT) is more sensitive than chest radiographs and it can be regarded as the gold standard in the detection of small pneumothoraces and size estimation (**Figure 1**).[21] CT is also useful in the presence of surgical emphysema and bullous lung disease and for identifying additional lung pathology.[22] However, it is not required in the majority of cases, as chest radiograph is usually sufficient to make a diagnosis, thus young patient population is avoided from excessive radiation dose. However, prolonged chest tube drainage and continuous air leak from the chest tube is considered as clear indication to obtain a CT scan.

There is an artificial intelligence (AI) report on chest ultrasound, which is considered to be more sensitive than chest radiography and this is a promising area of research.[23] AI models are only useful as a screening tool to determine the presence or absence of PSP, but future studies are needed as they do not incorporate the individual patient's circumstances and other medical conditions.[24]

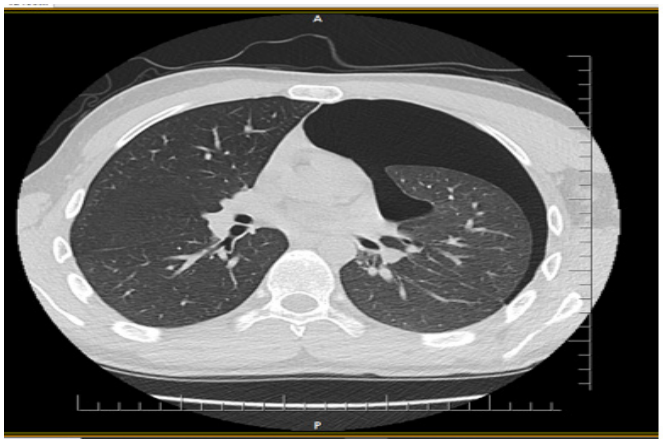


Figure 1. CT image of PSP
CT: Computed tomography, PSP: Primary spontaneous pneumothorax

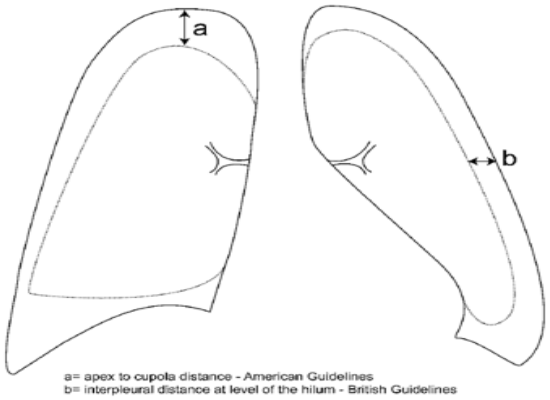
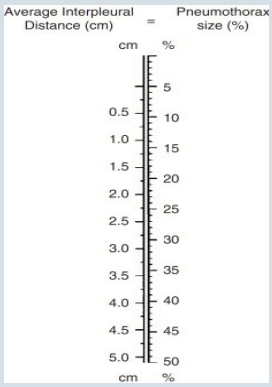


Figure 2. Schematic appearance of the measurement method for depth of the pneumothorax

Moreover, the plain PA chest X-ray has been used to quantify the size of the PSP. However, it tends to estimate the size because it is a two-dimensional image while the pleural cavity is a three-dimensional structure. There are three methods can be used to measure size of the PSP; Light method,[25] Collins method,[26] Rhea method[27] (**Table**). Although treatment and follow-up of recommendations are based on size of the PSP in different guidelines, there is no consensus on the definition of 'large PSP' and best method to estimate the size of PSP.

Table 1. Formulas used for determining of the size of the pneumothorax

Light	% of pneumothorax = 100–(D_L^3/D_H^3×100) D_L is the diameter of the collapsed lung D_H is the diameter of the hemithorax on the collapsed side
Collins	% of pneumothorax=4.2(4.7*(A+B+C)) A: distance between partially collapse lung apex to the apex of thoracic cavity B: mid points of upper half of collapsed lung C: mid points of lower half of collapsed lung
Rhea	Measuring average interpleural distance (AID) and using the normogram AID: A*B*C/3 A: distance between partially collapse lung apex to the apex of thoracic cavity B: mid points of upper half of collapsed lung C: mid points of lower half of collapsed lung 

The British Thoracic Society (BTS) Guideline uses interpleural distance at the level of the hilum, accept that a 2 cm radiographic PSP approximates to a 50% PSP by volume, it is called large PSP.[28] The American College of Chest Physicians (ACCP) uses the distance between apex to cupola for calculation of size of PSP and accept that ≥3 cm radiographic PSP is large PSP (**Figure 2**).[29] The Belgian Guideline uses Light index and pleural gap along the entire length of the lateral chest wall which is definition of large PSP.[30] Comparison between the techniques in different guidelines have shown poor agreement.[31]

In a multivariate analysis, Choi et al.[32] found that the presence of blebs on high resolution CT scans and chest X-rays were independent risk factors for ipsilateral recurrence. The European Respiratory Society (ERS) task force statement indicates that cross sectional imaging can be useful in complicated cases, where underlying disease is suspected and in patients requiring surgery.[7] The ACCP consensus panel did not recommend the routine use of CT imaging for the patients with first episode of PSP.[28] In addition, no consensus has been achieved by the experts, regarding the utility of CT scans for evaluating the patients with recurrent PSP, persistent air leaks and as a preoperative imaging modality. In a survey by the European Pediatric Surgeons' Association (EUPSA), it has been found that the use of CT scans in PSP mostly depends on the surgeon's personal preference instead of evidence-based recommendations.[33]

Compared the use of ultrasound instead of chest X-rays for follow-up of patients evacuated for PSP and found that ultrasound was superior to chest X-rays for follow-up.[34] It requires a trained team and access to ultrasound so that it can be used as an alternative to radiography.

In conclusion, when a child presents with sudden onset of chest pain or difficulty breathing, a detailed anamnesis should be taken and PSP should be suspected especially if there is a difference in breath sounds on physical examination. A chest radiograph should be ordered. A chest X-ray scan may support the diagnosis by showing air in the intrapleural space.

TREATMENT OPTIONS FOR PNEUMOTHORAX

The main aim in the treatment of PSP is to drain the air from the pleural cavity to allow the lung to re-expand and prevent recurrence. The treatment of PSP is lacking a definitive consensus to date and it is still controversial.

There are five main therapeutic approaches for the treatment of spontaneous PSP:[35]

1. Conservative approach
2. Ambulatory device such as a flutter valve
3. Needle aspiration using a small-bore cannula or aspiration kit

4. Intercostal drain attached to a drainage system
5. Surgery for either acute or preventative treatment.

NON-SURGICAL MANAGEMENT OF PSP

The distinction between PSP and SSP is based on data showing differences in treatment outcomes, duration of air leakage and suitability for surgical resection.

The initial management of PSP depends on the size of the PSP and the clinical condition of the patient. Non-operative treatment options include bed rest, oxygen supplementation with watch-and-wait option or needle aspiration. The air in the pleural cavity is absorbed four times faster with oxygen supplementation, therefore, nasal oxygen therapy is used in conservative follow-up.[36] In addition, there is a group that does not recommend the systematic use of oxygen therapy in patients treated for PSP because of poor methodological quality of data from previous studies and the many potential disadvantages, such as additional costs and hospitalization.[37]

While there are no clear recommendations for choosing a treatment option, previous adult guidelines have been predominantly based on clinical symptoms, PSP size and number of episodes. ACCP consensus reports recommend a more invasive approach considering the size of the PSP, while BTS guidelines recommend a conservative approach based on clinical symptoms.[28,29]

According to BTS guidelines, observation is the treatment of choice for small PSP (size <2 cm) without significant breathlessness. Asymptomatic patients with large PSP can also be managed with observation alone. Needle aspiration (NA) is first choice for patients with large PSP (size >2 cm) with breathless. If not successful, chest drain is recommended.[28]

According to ACCP guidelines, clinically stable patients with small PSP (size <3 cm apex to cupola distance) should be observed in the emergency department for 3 to 6 hours. Clinically stable patients with large PSP (size ≥3 cm apex to cupola distance) should undergo a procedure to re-expand the lung and should be hospitalized. The lung should be re-expanded by using a small-bore catheter or chest tube. Clinically unstable patients with large PSP should undergo hospitalization with insertion of a chest catheter.[29]

According to BTS guidelines, breathlessness in an adult with PSP is an indication for active intervention.[28] The BTS guidelines highlight the use of NA as an initial intervention in large and/or symptomatic patients because of NA is as effective as chest drains and is less painful than chest drains.[28] However, Gariepy et al.[38] showed that in contrast to the adult series, there was no advantage of the use of needle aspiration in children. But NA is failure in approximately one-third of patients will require a second procedure[28] so the ACCP recommend chest drain insertion.[29]

Complications are rarely reported and the risk is lower with NA than with chest drain insertion but chest drain insertion shows a higher success rate than NA.[39] Pediatric surgeons responding to a survey of members of the European Pediatric Surgeons' Association showed that they mostly preferred chest tube and pigtail placement over simple aspiration (**Figure 3**).[33]

Suction is another option for re-expansion of the lung. Suction is not recommended routinely, because it may cause

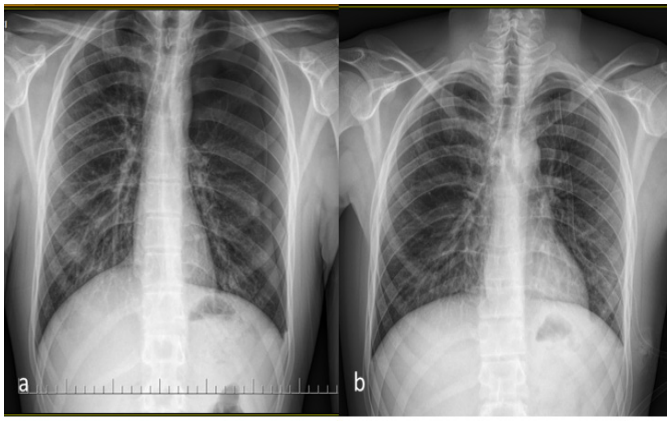


Figure 3. a) Chest radiograph of pneumothorax **b)** Chest radiograph of primary spontaneous pneumothorax after chest tube, left hemithorax is re-expanded

of re-expansion pulmonary oedema (RPO).[28] RPO may occur in some PSP patients followed by chest tube insertion and drainage procedure. It is rare complication but life-threatening, caused by rapid pulmonary re-expansion in especially young patient with large and persistent PSP.

When ensure that the air leak into the pleural space has resolved, chest tube can be removed. Several studies have evaluated attempted clamping before tube removal and one retrospective study found more frequent recurrence after chest tube drainage removal compared with no attempted clamping.[40] In a consensus statement, 53% of panel members reported that they would never clamp a chest tube to detect the presence of an air leak.[29]

In a survey, surgeons performing thoracoscopic surgery preferred shorter duration chest tube drainage compared to those performing open surgery.[33]

The current survey revealed that the majority of The Turkish Association of Pediatric Surgeons (TAPS) members were influenced the size of the PSP for choosing treatment options. They prefer tube thoracostomy as the first treatment, the second preferred option was oxygen administration. Needle aspiration option was rare and none of them preferred surgical management as the initial.[41]

All patients with SSP should be admitted to hospital and most patients will require chest drain. Because SSP is less likely to be tolerated by patients than PSP due to associated lung disease.[28,29] Aspiration is less successful in SSP but can be considered in symptomatic patients with small pneumothoraces to avoid chest drain.[28]

Medical chemical pleurodesis can be used for difficult or recurrent pneumothoraces but surgical options are now more effective. Several agents have been used for chemical pleurodesis including talc, tetracycline, autologous blood, fibrin glue. Large particle talc does not lead to the acute respiratory distress syndrome, therefore it is safe in use and most effective sclerosing agent.[42] So talc is the most widely used agent for chemical pleurodesis in Europe.[7] It can be used if a patient is unwilling or unable to surgery. Inflammation of the pleura causes dense adhesions so that the pleura adheres to the thoracic wall. This method may prevent recurrence but makes it difficult to perform thoracoscopy or thoracotomy in the future. In addition, chemical pleurodesis is very painful compared to thoracotomy.[11]

Conservative treatment was superior to non-conservative treatment, even if eight weeks were required for radiographic

resolution. This strategy reduced the number of procedures, length of hospital stay, adverse event rate and days off work.[43]

SURGICAL MANAGEMENT OF PSP

Seventy percent of patients having their first PSP never relapse, so performing surgery at first episode PSP seems like too invasive.[44] Therefore the other guidelines for the management of patients with PSP recommend air removal from intrapleural space via NA or chest tube insertion firstly in patients with PSP, surgery should not be performed as first line procedure.[37]

Surgical management of PSP ranges from chest tube insertion to more invasive intervention such as lung resection, pleurodesis or bullae excision via thoracoscopy or thoracotomy.

The objectives of surgical treatment are surgical repair of persistent air leaks and prevention of recurrence. The first objective is to identify any visible bullae or blebs on visceral pleura and resect this part, the second objective is to create adhesion between pleural surfaces. There is no evidence on when is the ideal time for thoracic surgery in cases of persistent air leak. Although 5 days was previously considered the cut-off point, some studies have shown that persistent air leakage lasting longer than 7 days can be controlled without surgery.[45]

There is also study showing that prolonged delays more than 5 days reduces the effect of thoracoscopy.[46]

In online survey of EUPSA, they found that presence of bullae >2 cm (77%) and recurrence (76%) were accepted as the most common indications for surgical treatment.[33]

Although there is a controversy still exists about the timing surgery, some indications for surgery is well accepted by many surgeons;[28]

1. Second ipsilateral PSP
2. First contralateral PSP
3. Synchronous bilateral spontaneous PSP
4. Persistent air leak (despite 5-7 days of chest tube drainage) or failure of lung re-expansion.
5. Spontaneous hemothorax
6. Professions at risk (i.e, pilots, divers)
7. Pregnancy

Open thoracotomy with pleural abrasion was the surgical treatment for PSP, described by Tyson and Candall[47] in 1941. Previous experience of surgeons performing open thoracotomy with parietal pleurectomy has shown that the method is highly effective and has low PSP recurrence rates.[48]

The traditional open approach has gradually been replaced by minimal invasive surgery. At the moment, video-assisted thoracoscopic surgery (VATS) has become the "gold standard" of PSP treatment, as evidenced by numerous medical reports from around the world.[49]

Re-operation following VATS is more often required than open thoracotomy with a high rate of both late recurrent pneumothoraces, prolonged early postoperative air-leakage.[50] Some clinical trials including PSP patients showed that thoracoscopy offered no superiority over limited thoracotomy.[51] However, VATS is significantly superior to thoracotomy in terms of less postoperative pain, better wound cosmetics, shorter hospital stay, better functional recovery and patient satisfaction.[49]

During surgery may undergo bullectomy with patients with PSP, electrocoagulation, laser ablation, stapling, Ligasure, endo-loop or manual suturing can be used. In addition to bullectomy, adhesion should be created between the two opposing pleura to prevent recurrence.[29] In the past, pleural abrasion was widely used for surgical pleurodesis. Some studies have shown that partial pleurectomy is more advantageous than pleural abrasion and that the combination of both techniques leads to better results.[52]

In one study the surgical treatment of PSP with mesh coating was found to be significantly advantageous compared to thoracoscopic bullectomy, allowing for a lower recurrence rate, hospital stay and chest tube drainage time.[53]

Medical thoracoscopy with talc poudrage has been shown to be a safe and effective technique for recurrence prevention of both PSP and SSP. Talc poudrage with medical thorascopy has similar recurrence rates to VATS, so it is most commonly used approach in current practice.[19]

In patients where urgent intervention is not required, management options largely depend on the patient's priorities and available local services. For example, a patient may need to fly in the near future. The current advice is that passengers should not fly until 7 days after resolution on the chest radiograph and that there is a high risk of recurrent PSP.[54]

After thoracic surgery, patients should be informed that they should delay their flight 4 weeks for non-essential air travel and 2 weeks for essential air travel.[54]

Our study represents a review of the available literature and is important in demonstrating the differences in approach in various guidelines. However, the weakness of our study is that most of the studies included adult patients and the guidelines were aimed at adults. We would like to emphasize that it is important to conduct more studies on pediatric patients and to follow the current literature with the developing technological developments.

CONCLUSION

The precise incidence of PSP is unknown but it has become clear that PSP is a disease associated with abnormalities within the pleura. The management PSP shows variability not only in conservative management, but also in surgical treatment. Clinical outcome and underlying pathologies variable, it is difficult to recommend a treatment algorithm for PSP cases. Most of current treatment recommendations are based on adult studies, there is no standard care and treatment for PSP in children. With the development of new technologies, more large-scale randomized studies are needed to better classify PSP and to establish new approaches in the diagnosis, treatment, follow-up of patients and the lack of a guideline for pediatric age groups is one of the most important reasons for the need for new studies.

ETHICS COMMITTEE APPROVAL

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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