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Is it possible to standardize the treatment of primary spontaneous pneumothorax? Part 1: etiology, symptoms, diagnostics, minimally invasive treatment

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Abstract

The authors of this report present the history of primary spontaneous pneumothorax (PSP) treatment, its etiology, clinical symptoms, and diagnostic methodology. Further, they discuss minimally invasive methods of treating PSP such as thoracentesis and chemical pleurodesis. They discuss the pros and cons of each method, emphasizing that, according to the international recommendations, they should be used as the first line of treatment for PSP.

Key words: primary spontaneous pneumothorax, thoracentesis, chemical pleurodesis.

Streszczenie

Autorzy niniejszego doniesienia przedstawiają historię leczenia samoistnej pierwotnej odmy opłucnowej (SPOO), etiologię jej powstania, objawy kliniczne oraz metodykę diagnostyki. Ponadto omawiają mało inwazyjne metody jej leczenia, takie jak nakłucie i drenaż jamy opłucnej oraz pleurodezę chemiczną. Przedstawiają wady i zalety każdej z tych metod, podkreślając, że zgodnie z międzynarodowymi zaleceniami powinny stanowić pierwszą linię postępowania lekarskiego w leczeniu SPOO.

Słowa kluczowe: odma opłucnowa, nakłucie, drenaż, pleurodeza chemiczna.

Introduction

Pneumothorax refers to the presence of air or gases in the pleural cavity. The term was introduced into the medical lexicon by Itard in 1803 and Laennec in 1819 [1]. The term spontaneous pneumothorax was proposed by Kjaergaard in 1932 [2].

Primary spontaneous pneumothorax (PSP) is a global problem; based on various data, its incidence is estimated at 18–28 men/100 000 inhabitants and 1.2–6 women/100 000 inhabitants [3]. Other publications estimate this incidence at 24 men/100 000 inhabitants and 9.8 women/100 000 inhabitants [4]. The peak of PSP incidence is observed among young individuals between the ages of 15 and 34 [5].

The most important risk factor for this condition is tobacco addiction. In comparison to non-smokers, the risk is 7 times higher in light smokers (1–12 cig./day), 21 times higher in moderate smokers (13–22 cig./day), and 80 times higher in heavy smokers (> 22 cig./day) [6]. It is perturbing that 80–86% of young patients after PSP treatment continue to smoke tobacco; as a result, during the first 4 postoperative years, the risk of recurrence is very high and is estimated at approximately 54% [4]. The fact that familial predisposition increases the risk of developing PSP to 11.5% has also been underscored [6].

Another factor considered to cause PSP is inflammatory changes in the ultimate segments of the airway, which can cause pulmonary tissue to rupture when intrapulmonary pressure changes during breathing [7].

The influence of atmospheric pressure on the development of PSP was discussed for a long time in various scientific publications; at present, only rapid changes of atmospheric pressure (e.g., associated with diving, traveling by plane, or violent hurricanes) are considered to contribute to the rupture of already existing blebs or bullae, or pulmonary alveoli affected by inflammation [8]. The most common mechanism of PSP development is the rupture of a subpleural air pocket (a so-called bleb, up to 1 cm in diameter), located mainly in the apical part of the lung [9].

Primary spontaneous pneumothorax etiology

The exact cause of PSP development is still largely unknown. During surgical procedures, uni- or bilateral pulmonary blebs and/or emphysematous bullae, located primarily in the apical sections, are found in > 80% of patients. These lesions, known as emphysema-like changes (ELCs), are located under the visceral pleura and are observed in approximately 79% in CT scans of patients undergoing surgical treatment. They are considered to form an independent and specific nosological entity [10, 11].

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In 2012, Belchis *et al.* conducted microscopic studies demonstrating fibroblastic damage of the pulmonary tissue leading to fibrosis of the visceral pleura with islands of fibroblastic foci and myxoid stroma in most PSP patients [5, 12]. Primary spontaneous pneumothorax development is believed to be caused by ELC rupture, which seems to confirm Vanderschueren's hypothesis concerning the mechanism of pneumothorax development. Although investigations of lung segments from PSP patients conducted with electron microscopy did not demonstrate macro- and microscopic sites of "air leakage", they did demonstrate the presence of diffuse areas of disrupted mesothelial cells of the visceral pleura covered with a layer of inflammatory cells with increased fibrosis and pores 1–20 µm in diameter, suggesting the presence of diffuse pleural porosity [13, 14]. Hence the return of Stradling and Pole's theory: in 1966, these researchers put forward visceral pleural damage as a cause of PSP. Based on these premises, Cao *et al.* [15] returned to the previously known method of inducing adhesions by the intrapleural administration of autologous blood. According to these authors, the patient's blood coagulating above the damaged visceral pleura may seal the site of the injury and facilitate its healing.

The view held in recent decades that the presence of air in the pleural cavity is a result of bleb rupture in accordance with the "one-airway-one-bleb-one-leak" rule is overly simplistic as there is a group of PSP patients in whom no blebs were found intraoperatively. Hence the return of an old theory, accepted by most researchers, that the visceral pleura has "pores" through which air may permeate from the lungs into the pleural cavity [16]. It appears that the development of blebs, bullae, and visceral pleural porosity is influenced by a number of incompletely understood factors, the most important of which include: congenital predisposition, peripheral chronic pulmonary parenchymal inflammation (ELCs), anatomic abnormalities of the airway, ischemia of the apical pulmonary segments, malnutrition – low body mass index (BMI), abnormalities in connective tissue structure, cannabis smoking, AIDS, and many other factors that remain unknown [11, 17].

According to Haynes and Baumann, the real cause behind rupturing blebs or emphysematous bullae resulting in pneumothorax development has still not been fully explained [11]. Blebs and emphysematous bullae are not the only causes of PSP. Amjadi *et al.* performed over 250 thoracoscopic sympathectomies due to Raynaud's disease, demonstrating the presence of blebs in approximately 6% of the patients undergoing surgery. In the analyzed patient group, 46% were chronic tobacco smokers. The researchers did not remove the blebs during the sympathectomy procedures. Preoperatively, none of the patients disclosed PSP in their medical history, and not even one case of pneumothorax was observed during the 9-year follow-up after the procedure. The authors proposed a hypothesis that blebs are precursors of PSP appearance [18]. These observations seem to be substantiated by the results of thoracoscopic examinations conducted by Noppen and Dekeukeleire, who believe that the numerous causes of PSP development and recurrence include not only bleb rupture, but also porosity of the visceral pleura [19].

Clinical symptoms and diagnostics

Detailed physical examination combined with radiological imaging form the basis of PSP diagnostics. Asthenic body build, "shortening" of breath, and sudden chest pain observed in approximately 81% of patients are the initial and most frequent symptoms of PSP.

The sudden chest pain is believed to result from bleb rupture. This causes air to reach the pleural cavity as the irritant material (bronchial secretion with bacteria) is released from the lungs, which stimulates the development of an inflammatory process in this area. Another rather characteristic symptom is the feeling of moderate dyspnea noted in approximately 39% of patients. Other notable PSP symptoms include the appearance of persistent cough, necessity of deep breathing, paroxysmal tachycardia, and poor well-being. Therefore, a significant portion of patients is initially examined for cardiovascular diseases [20]. The feeling of dyspnea and breathing difficulties may signify the presence of rare conditions accompanying PSP such as tension pneumothorax (1–5% of patients) or bilateral pneumothorax (0.5–1.9% of all patients) [21].

Arterial oxygen saturation is normal in 75–95% of PSP patients; while breathing indoors, the patients usually exhibit values exceeding 92–94% [22].

The standard of radiological examinations for PSP according to the British Thoracic Society (BTS) guidelines from 2010 is a chest image in a PA projection taken with the patient standing up. A lateral image of the chest from the side affected by the pneumothorax can also be taken, but it is not required in routine medical practice [23]. At present, computed tomography is the "gold standard" for detecting small pneumothoraces, which allows one to precisely calculate their dimensions. Among the many radiological methods of determining pneumothorax size, of note is the Light index, which consists in measuring the mean dimensions of the lung and the pneumothorax at the level of the hilum and substituting them into the following formula: pneumothorax size (%) = 100 – (mean lung dimension in cm/mean pneumothorax dimension in cm) × 100 [24].

Most often, the size is determined in accordance with the guidelines published by the American College of Chest Physicians (ACCP) or the BTS. Americans evaluate the size of pneumothorax based on the distance between the cupula and the apex of the collapsed lung, distinguishing between small (< 3 cm) and large (> 3 cm) pneumothoraces. In turn, British physicians determine the size based on the distance between the chest wall and the side of the collapsed lung measured at the level of the hilum. Like their American counterparts, they classify pneumothorax as small or large. At this point, it should be underscored that in most cases the size of the pneumothorax does not correlate exactly with its clinical symptoms [9] (Fig. 1).

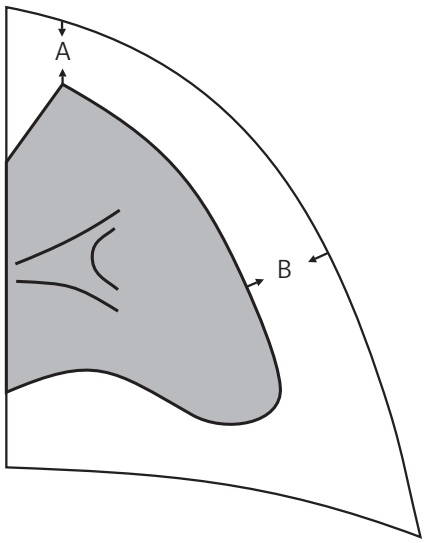

Fig. 1. Assessing pneumothorax size based on chest radiograms. A – The ACCP defines the size of pneumothorax as the distance between the cupula and the apex of the collapsed lung (< 3 cm: small pneumothorax; ≥ 3 cm: large pneumothorax). B – The BTS defines the size of pneumothorax as the distance between the chest wall and the side of the collapsed lung measured at the height of the hilum (< 2 cm: small; ≥ 3 cm: large)

Tab. I. Factors influencing the success of PSP treatment with pleural puncture

Age < 50 years	70–81% success
Age > 50 years	19–31% success
Pneumothorax volume < 3 l	89% success
Pneumothorax volume > 3 l	0% success
Percentage of lung collapse < 50%	77% success
Percentage of lung collapse > 50%	62% success

In the last decade, ultrasonography has been increasingly used to diagnose PSP; various authors stress that not only is it a minimally invasive method that can be easily repeated, but it is also more sensitive than radiological examinations [25].

Treatment

At present, there are two key problems to solve with regard to PSP treatment:
– How to stop the air from leaking?
– How to minimize recurrence?

The methods that are currently most frequently used for treating primary spontaneous pneumothorax include conservative treatment (patient observation), simple puncture of the pleural cavity, intercostal drainage (with a pigtail catheter or a drain), intercostal drainage combined with chemical pleurodesis, thoracoscopy combined with chemical pleurodesis, open thoracotomy with mechanical pleurodesis, video assisted thoracic surgery (VATS) combined with chemical pleurodesis, and open thoracotomy combined with chemical pleurodesis [26, 27].

Tab. II. Comparison of the efficacy of pleural puncture and drainage and the associated rates of recurrence after the 1st year

Puncture	Drainage
67% efficacy	80% efficacy
Recurrence after the 1st year	
16.5%	23%

Conservative treatment

In cases with small pneumothoraces and scant clinical symptoms, conservative treatment is recommended. The patient's mobility should be limited; they should be placed in bed on the affected side and perform respiratory exercises consisting in overcoming resistance during exhalation. The air from the pleural cavity is spontaneously absorbed at the rate of approximately 2%/24 h (50–75 ml/24 h), especially when the patient is offered clean oxygen to breathe. It has been suggested that the patient can be discharged home if no symptoms of pneumothorax aggravation are noted after approximately 6 h of hospital observation (in the Emergency Room), provided that the patient is able to return to the hospital quickly. Approximately 8–15% of patients qualify for this method of treatment. Its efficacy is estimated at 8–100%, while PSP recurrence is noted in 22–50% of patients treated with this method [11, 27].

Pleural puncture

Considering that PSP pathology results from the rupture of subpleural blebs, it appears that this treatment method, enabling the removal of air from the pleural cavity, should be used more often. Frequently, the rupture is so small that the whole lung "contracts" after the puncture, and the air leak is stopped completely. Lung reexpansion (decompression) after puncture is relatively high: 50–83% according to various publications. It depends on the following factors (Tab. I).

After the puncture, the patient must be monitored for at least 6 h in the ER [6, 28]. In randomized controlled trials and a meta-analysis comparing pleural puncture with drainage, statistical analysis revealed no significant differences in the treatment outcomes of these two methods (although the latter was favored) [2, 29]. Another meta-analysis demonstrated that puncture should be the first method of PSP treatment as it significantly shortens the length of hospitalization, thus markedly reducing treatment costs [30]. In turn, randomized trials (5 reports from the years 1994–2012 and 387 analyzed patients) compared the efficacy of puncture vs. drainage with a small drain, reporting the following results (Tab. II).

The intergroup differences presented above were not statistically significant [5, 31].

Based on these data and the guidelines published in the UK (BTS, 2010) and Belgium (Belgian Society of Pneumonology, 2006) recommending pleural puncture as the first step in PSP treatment, it appears that these recom-

mendations are worth considering in standard medical management [23, 32].

However, not everyone agrees with these guidelines, as a French prospective study from 2014 and American guidelines (ACCP, 2008) showed that treating the first episode of PSP using a small-bore drain with a valve is a good alternative to pleural puncture and/or drainage, reducing the treatment costs significantly [20, 33].

Pleural drainage

This is the next step after puncture if the lung does not expand. Most often, drains with diameters of 10–40 F are used. In the Delphi consensus statement from 2001, researchers from the ACCP recommend performing intercostal drainage in any patient with PSP exceeding 20% of the pleural cavity volume [34]. From the patient's perspective, intercostal drainage of the pleural cavity is a very painful procedure. Approximately 50% of patients treated with this method score the pain as 9–10 points on the VAS scale. Additionally, randomized trials demonstrated a number of dangerous complications occurring during chest drainage (internal organ injuries, hemorrhages, infections, and even death) [35].

For several years now, drains of low diameter have been recommended for intercostal drainage of the pleural cavity. It has been demonstrated that these drains are as effective as their larger counterparts, but are associated with a significant reduction in postoperative pain. Moreover, smaller drains are associated with a significant reduction in the length of hospitalization (by 3–4 days on average). These findings have been confirmed by other authors: their retrospective studies demonstrated that 11–13 F drains are not only better tolerated by the patients, but offer better treatment outcomes than 20–28 F drains [36, 37].

However, there is no clear answer whether suction should be performed in the patients undergoing drainage. The optimal values for the suction force range from 10 to 20 cm H_2O. Other values are not recommended. It has been demonstrated that the lung expands on its own within 3 days after drainage without suction in 70% of patients [20]. An expert opinion from the BTS published in 2010 suggests that suction may be beneficial for a small number of patients. So-called "bubbling" combined with no lung expansion persisting for more than 48 h after the procedure is believed to be an indication for the application of suction.

These observations are confirmed by a Polish report from 2014, which demonstrated a marked benefit of using suction in PSP patients with so-called persistent air-leaks [38].

A retrospective study encompassing 55 patients treated with small-bore drainage connected with a Heimlich valve showed that this method of treating PSP was highly effective [39]. It is believed that drainage with a small-caliber catheter (pigtail catheter, cavafix, neo pneumocath) with a Heimlich valve can be provided as an out-patient treatment even to patients with a large pneumothorax, provided that the patients live near the clinic. These observations are confirmed by a report by Ayed *et al.*; when a puncture is unsuccessful in a PSP patient, the authors perform drainage with an 8 F drain with a Heimlich valve and discharge the patient from the ER after 6 h of observation [36, 39].

Researchers from Taiwan published a report in 2014 stating that the first-line gold standard for PSP treatment should be intercostal drainage with a very thin 8 Fr catheter with a Heimlich valve; in the case of recurrence, the second line of treatment should consist in drainage combined with minocycline pleurodesis [40, 41]. In most patients, drainage is used for 2–7 days. In some centers, an air leak persisting for more than 7 days is an indication for surgical treatment [27].

Pleural drainage with chemical pleurodesis

Chemical pleurodesis is performed to induce adhesions between the pleurae, thus preventing PSP recurrence. Administering an irritant into the pleural cavity results in aseptic pleuritis, causing the pleurae to adhere to each other. The year 1935 saw the first use of talc (hydrated magnesium silicate) as the substance for obliterating the pleural cavity. Since then, it has been used in many centers around the world, and its popularity can be attested by the fact that it is one of the most frequently used substances for obliteration both in Europe and in English-speaking countries around the world (USA, UK, Canada, Australia, New Zealand) [42]. The highest efficacy is associated with talc molecules smaller than 15 µm.

In clinical practice, the agent is most often provided as 2–4 g of talc with 20 ml of lidocaine in the form of a talc slurry administered through a drain into the pleural cavity [43]. In contrast to previously held views, it has been demonstrated that the use of talc is safe and does not cause acute respiratory distress syndrome [44]. Other substances used intrapleurally to achieve obliteration include bleomycin, dextrose, 50% glucose, iodine tincture, tetracycline (in the form of a slurry), povidone-iodine, picibanil, silver nitrate, quinacrine, and autologous blood [41]. Over the last few years, most publications have reported tetracyclines, minocycline in particular, to be the agents most frequently used for obliteration. At present, they are recommended as the first line of agents for chemical pleurodesis in PSP patients. Tetracyclines (dosed at 500 mg doxycycline and 7 mg/kg b.w. minocycline) should be administered intrapleurally in the form of a slurry in 200 mg (20 ml) of lidocaine, which markedly reduces postoperative pain in 10–70% of patients [23].

A different opinion was presented by Chen *et al.*, who demonstrated that approximately 68% of patients treated with minocycline experienced pain for up to 6 months after the procedure and required intramuscular injections with pethidine [16]. It was demonstrated that treating PSP with drainage alone is associated with a 23–27% rate of recurrence, while combining drainage with chemical pleurodesis reduces this rate to approximately 5%. In a randomized trial announced in 2013, Chen *et al.* proposed the first line of PSP treatment to consist in puncture and/or drainage com-

bined with minocycline pleurodesis, which significantly reduces recurrence. Concurrently, they demonstrated that, in contrast to talc, intrapleural minocycline does not change the ventilatory parameters of the lungs (VC or FEV_1) in PSP patients after the conclusion of treatment [16, 41].

The facts presented in this report prove that despite the 200 years that have passed since the first diagnosis of pneumothorax, the precise cause of the condition remains unknown. According to many researchers, the treatment methods described above should constitute the first line of treatment for PSP.

Disclosure

Authors report no conflict of interest.

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