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Polymer fume fever (review)

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Abstract. *Despite high chemical inertness of polytetrafluoroethylene, its pyrolysis, or the incomplete combustion reaction (oxidation-free thermal degradation), can lead to a rapid onset of flu-like symptoms when the resulting products are inhaled. This phenomenon is known as polymer fume fever and is most commonly observed in smokers. The hypothesis that ultrafine nanoparticles present in the air facilitate the transport of hydrofluoric acid molecules is the only standard that comes close to explaining this phenomenon. In some cases, when a toxic cause is not suspected, invasive tests are performed unnecessarily. There is a risk that the patient continues to be intoxicated and may develop pulmonary oedema or acute respiratory distress syndrome. There are no specific antidotes for Teflon pyrolysis products. The pervasive utilisation of polymeric products in industrial and domestic settings, in case of accidents or misuse, has the potential to exert a considerable negative influence on public health. Polymer fume fever is a rare, flu-like, occupational disease that is not always diagnosed promptly. A better understanding of the pathological process provides clinicians with the ability to make the correct diagnosis, properly educate patients, and ultimately achieve better treatment outcomes.*

Keywords: *polytetrafluoroethylene; pyrolysis products; polymer fume fever; diagnosis; treatment*

If you have a fever and body aches, you hardly think it's because you inhaled something.
M. Amirshahi, 2024

Polymer fume fever (PFF) is a disease that results from the inhalation of degradation products of polytetrafluoroethylene (PTFE; Teflon©). To a much lesser extent, inhalation of vapours from other plastics (polyvinyl chloride, chlorinated polymers, polyurethane, etc.) can also result in the development of the disease [1–9]. It is the least common of the inhalation fevers, which also includes metal fume fever and organic dust toxic syndrome. In the United States, PFF accounts for a mere 0.06 % of all inhalation pathologies [10].

Despite the fact that PTFE, otherwise known as a tetrafluoroethylene polymer, was first identified in 1938, further research resulted in the description of a new compound that would subsequently have a profound and irreversible impact on the world. PTFE is a reactive, hydrophobic material with a low coefficient of friction at room temperature. These features are widely used in a variety of industries, ranging from coating pans to create a non-stick surface, making breathable, highly resistant fabrics for outerwear and tactical clothing, lubricants, sprays, medical devices, including coating stents and implants, catheters, hernia mesh, etc. [1, 2, 10, 11]. Due to its exceptional hydrophobic and oleophobic properties, PTFE is widely used in the manufacture of packaging materials and cooking utensils. However, the use of PTFE products has led to a significant environmental impact, with the contamination of the air, water, and soil [12].

Polytetrafluoroethylene and its precursors are called forever chemicals, due to their high stability and extremely slow degradation in the environment [13, 14]. Despite extreme chemical inertness of PTFE, its pyrolysis, or the incomplete combustion reaction (oxidation-free thermal degradation), can result in the rapid onset of flu-like symptoms when resulting products are inhaled [1, 15]. The condition has become popularly known as Teflon flu, in reference to the trade name of a widely used non-stick coating [14].

Adverse health effects are associated with the formation of nanoscale particles consisting of degradation products, including polymers in vapour in the form of aerosol, which can penetrate deep into the bronchioles and pulmonary interstitium [7]. Polytetrafluoroethylene aerosol, especially freshly formed one, on which degradation products are sorbed, has a pyrogenic effect. When inhaling cold PTFE dust after 2 to 5 hours, all workers showed symptoms called Teflon flu. The presence of polytetrafluoroethylene aerosol in the air at a concentration of 0.2–5.5 mg/m³ was found to result in re-

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current fever attacks among most workers. The formation of PTFE microparticles is observed at temperatures of > 120 °C. The diameter of these particles is less than 2.5 microns, which allows them to deeply enter the respiratory tract. In the ambient atmosphere, they can reach concentrations of 500,000/cm^3. As the pyrolysis temperature is increased, the nanoparticles become finer. At a temperature of approximately 600 °C, they disappear completely, transforming into gaseous decomposition products in an aggregate state. A certain amount of ultrafine particles coagulate and self-organise into conglomerates that are too large to reach the lungs. PTFE microparticles themselves are not capable of causing polymer fume fever, but serve as carriers of toxic vapour components of toxic substances, such as hydrofluoric acid, which cannot independently reach the lower respiratory tract [10]. Inflammation can be triggered by an adverse biological reaction to chemicals and metals that are also present on the surface of the particles (the Trojan horse phenomenon) [7].

Typical Teflon fever is most commonly experienced when working with polytetrafluoroethylene heated to > 350 °C. PTFE thermally degradable substances have been identified as specific products of pyrolysis or degradation and are likely to be the main xenobiotics responsible for the clinical effects of polymer fume fever. Although PTFE releases toxic vapours at relatively low temperatures (up to 250 °C), people usually do not experience symptoms until the material is heated to 350 °C. Pyrolysis starts at a temperature of approximately 400 °C [1, 2].

Teflon pyrolysis products are formed depending on the temperature:

— from 250 °C: hydrogen fluoride (hydrofluoric acid), carbonyl fluoride, octafluorobutylene;

— at 300–380 °C: hexafluoroethane, octafluorocyclobutane, octafluorobutylene;

— at 450 °C: tetrafluoroethylene;

— at 460 °C: hexafluoropropylene;

— at 475 °C: perfluorooctylene [2, 10, 16, 17].

Perfluoroisobutylene (PFIB) is a highly toxic, ten times more poisonous than phosgene, colourless, odourless gas, very dangerous even in case of short-term inhalation. Medial lethal dose (LD_{50}) is 4 mg/m^3. PFIB irritates the respiratory tract and can be used as a potential chemical warfare agent [17]. Based on animal models, the experiment proved that a PFIB concentration of 150–180 ppm-min (parts per million-minutes) is sufficient to kill half the population, while a comparable dose of phosgene was 750 ppm-min [10].

Due to its inertness and relative thermal stability, the acute toxicity of fully polymerised PTFE was initially considered low. However, this assertion was challenged in 1951 with the first reported series of cases of polymer vapour fever due to occupational exposure [2–4, 6, 8, 18]. Subsequent epidemics over the years have further confirmed the findings of adverse health effects associated with overheating of fluorocarbon polymers, including Teflon [2]. 90 % of all clinical cases of PFF were men, most patients were smokers, and 80 % had no known comorbidities [19].

Polymer fume fever is primarily an occupational pathology resulting from exposure to PTFE thermal degradation products and ultrafine particles that can be generated during hot moulding. But it is often not recognised as an occupational disease [7, 10]. High-temperature production operations are carried out in technically isolated spaces and do not cause harm to human personnel in non-emergency conditions [10].

The temperature increase required for the formation of polytetrafluoroethylene pyrolysis products is rare in industry, but it cannot be ruled out in the event of technical malfunctions, accidents and inadequate ventilation, such as critical overheating of the electrical insulation coating of cables, pyrolysis of fluorine-containing grease during welding of stainless steel pipes, ignition of technical devices with Teflon structural elements, etc. [20]. Fires during accidents at chemical plants pose a particular danger to humans [17].

PTFE pyrolysis can be particularly dangerous when it is impossible to leave the contaminated area. Such an accident can occur, for example, on a submarine or spacecraft where a fire has broken out, when sailors or astronauts are forced to stay in the toxic gas-contaminated atmosphere [17].

However, both in emergency situations and during traditional work with polymers, episodes of Teflon fever occur mainly in smokers. First of all, mechanical contamination of tobacco products or smoking accessories with substances containing polytetrafluoroethylene (for example, after using mould removing spray and dry lubricants) is possible [2, 6, 7, 21, 22]. After several cases of flu-like outbreaks among technicians, epidemiologists determined that these symptoms were most often the result of poor industrial hygiene. People who worked with untreated PTFE and then touched their cigarettes without proper hand hygiene showed symptoms of PFF overwhelmingly [2, 6, 7, 11, 23].

In industrial operations with granular moulds or PTFE substrate, fluorinated microparticles can form an aerosol in a confined atmosphere and are deposited on cigarettes. They have a diameter of up to 1 μm, which allows them to contaminate tobacco products, even if they are contained in a closed package or blister [10]. During combustion, the hot end of a cigarette reaches temperatures of 850–900 °C, while temperatures above 250 °C are sufficient for the pyrolysis of fluorocarbon polymers. It is the products of this decomposition that are toxic and cause polymer fume fever, as they are inhaled with cigarette smoke [1, 2, 10, 22]. A smoker is likely to receive a toxic dose in a single breath. Subsequent inhalation of tobacco smoke provides sufficient exposure to PTFE to trigger an episode of polymer fume fever, which can be caused by exposure to particles of 0.1 to 1 micron in size, which makes them able to penetrate lung tissue [10]. Breathing in air when PTFE-coated material has been burnt or melted can be sufficient to cause symptoms of Teflon fever. Workers who do not smoke are practically unaffected by PFF [2, 15, 20].

Teflon spray is used to lubricate technical components subjected to prolonged high mechanical loads in a wide temperature range from –50 to +250 °C to minimise friction, as well as for the treatment of leather goods. A case of pulmonary polymer fever with pulmonary fibrosis in a young man has been reported as a result of inhalation of Teflon by-products for waterproofing horse tack. Domestic cases of polymer fume fever have been described mainly in relation to waterproofing agents [10]. Cases of Teflon fever with respiratory

distress have been reported after using waterproofing spray on mountain equipment in a closed, non-ventilated room with a subsequent rise in temperature to 39 °C [10, 21, 24].

Waterproofing spray-associated pneumonitis can progress to acute respiratory failure [25–27]. PTFE-induced fever can occur when using mould remover agents indoors, in the ink pad industry or in the manufacture of thermoformed products [10].

More unusual routes of exposure to particulate matter include inhalation of pyrolysed fluorocarbon-based hairspray, which can lead to fever and pneumonia shortly afterwards [2, 5].

Ski, snowboard and surfing enthusiasts can use fluorinated waxes to reduce friction as much as possible. The wax is applied to the base of skis or boards using the heat of an iron. When the wax is processed with a soldering iron or torch, the temperature can easily exceed 360 °C. The mechanical removal of excess wax with brushes and scrapers produces aerosolised particulate matter in the air, leading to direct inhalation of volatile substances, aerosols and dust. PFAS are known to pollute the environment near ski areas. The exact number of people directly affected by PFAS fluorinated ski waxes is unknown; however, it is estimated that > 10,000 people in both Sweden and Finland are professionally involved in the processing of skis and snowboards. In the United States, the ski industry employs approximately 80,000 people and has about 7,500 registered coaches. At international competitions, waxing booths are set up, and waxers are at risk of Teflon fever, especially if they smoke while working [28]. Recently, fluorocarbons contained in some ski waxes have been found to be responsible for pulmonary symptoms [10]. Preparation of 3 pairs of skis with 30 g of powdered fluorinated wax pollutes 20 km^3 of air [14, 19, 28].

In general, non-stick cookware is quite safe when used properly. "I assume that we all absorb some fluoroplastic with our food, which has peeled off our frying pan", says I. Cousins, a professor at Stockholm University, an environmental chemist. "I wouldn't really worry about it, it will just pass through our bodies in transit".

Over the past two decades, poison control centres in the United States have received more than 3,600 reports of suspected cases of polymer fume fever, a flu-like illness linked to a chemical coating found on some non-stick pans [14]. In 2023, there was an unprecedented surge in such cases. J. Weber, director of the Missouri Poison Control Centre, attributes this increase to the misuse of non-stick cookware. "People don't follow the instructions and don't use things properly", she said [19].

Most modern PTFE exposures occur in the home and result from inhalation of vapours released by overheated PTFE-coated pots and pans [1, 3, 4, 6, 7, 14, 17, 29]. The risk of inhalation of vapours increases if the heated cookware is dry, for example when no oil has been added, or all the water has evaporated. Exposure to PTFE particles after cooking oil has been applied to the non-stick coating appears to be less intense, as the relative vapour points of common cooking oils are lower than the temperature at which superheated PTFE provokes symptoms of PFF in humans [2–4, 15]. German researchers proved that a Teflon-coated pan had the highest toxic emissions at 370 °C [14]. If water is added to hot cookware with a melted/charred non-stick coating without allowing it to cool, it will instantly produce highly toxic "explosive steam" [1].

If a faulty microwave melts the Teflon inside it, it becomes not only vapour toxic but also explosive [3, 4].

During the COVID-19 crisis, there was a significant increase in the recycling of plastic waste [30]. Studies are being conducted on the possible harmfulness of constant wearing of medical masks, but no reliable results have been obtained [31]. Teflon becomes a serious environmental problem if it is incinerated at a high enough temperature to melt, and its pyrolysis products are released into the atmosphere as gas [7]. As early as 1977, Japanese scientists warned of the dangers of burning fluorocarbon products, such as PTFE, emphasising the fact that this produces gases that are many times more toxic than phosgene [28].

While it is common knowledge that Teflon and similar fluorinated polymers are used in many cooking accessories, it is less known that they are also found in the interior of many military vehicles, particularly armoured ones. As perfluoroisobutanol is produced when Teflon burns, it can be fatal to the crew and firefighters are at risk of toxic exposure [17].

Teflon balls for airguns are coated with PTFE, which allows them to be easily pushed through the barrel, which does not get very hot during firing, but the use of Teflon-coated balls in firearms is prohibited in most European and American countries. Dry lubricants made from fine fluoroplastic particles for firearms maintenance are not intended to lubricate the barrel of a firearm from the inside, which can get very hot during firing. Such products are not used to lubricate highly loaded parts with high operating temperatures.

The warheads of incendiary (thermite) projectiles made from active materials create a high-temperature combustion and explosion effect when they hit the target at high speed, releasing a large amount of thermal energy instantly, causing greater damage to the target. The calorific value of the combustion of PTFE-containing composites, as well as the thermal decomposition of fluorine-containing thermite reaches an energy of almost 20,000 J/g, causing a four-stage pyrolysis with the formation of a large number of chemical products of varying toxicity [32–34]. Although there is virtually no publicly available literature on the subject, it can be assumed that there is a high probability of inhalation exposure of military and civilian populations to Teflon pyrolysis products if the aggressor uses this type of munition. This also applies to aircraft fires and explosions of missiles with Teflon components.

Toxicology centres in the United States and Japan have reported only 3,600 suspected cases of polymer fever over the past two decades [14, 19]. In mild cases, the spontaneous episode resolves relatively quickly (in 14–72 hours) without significant consequences, so the patient believes they have acute respiratory viral infection and does not report the event to the doctor [10]. This is a significant under-reporting. Only 10 % of workers with symptoms consistent with PFF seek medical attention [2, 10, 14].

Pathophysiology. The pathophysiology of polymer fever is not fully understood, and researchers have proposed various theories, none of which has reached consensus [10]. Studies investigating the pathophysiological mechanisms of PFF,

toxicokinetics and toxicodynamics of PTFE degradation and pyrolysis products are very limited [2, 35]. One theory is that fluoridated microparticles break down and release hydrofluoric acid, which reacts with water in the body and causes chemical damage. This theory was put forward in 1951 when the first reports of polymer fever were published, but has not been confirmed by subsequent studies. The hypothesis of the transport of hydrofluoric acid molecules by ultrafine nanoparticles present in the air is the only reference that comes close to explaining this phenomenon [2, 8, 10].

A direct link between the cytokine storm and PFIB-induced acute lung injury has been identified in animal models. Levels of 10 pro-inflammatory and one anti-inflammatory cytokine were significantly increased in the lung tissue of mice exposed to PFIB [35]. An inflammatory syndrome with hyperleukocytosis of predominantly polynuclear neutrophils, with a peak in concentration at 9–12 hours and normalisation at 24–48 hours after poisoning; an increase in C-reactive protein to 2,640 mg/l, hypoxia ($PaO_2 \leq 60$ mm Hg) and a decrease in maximum lung capacity were noted [2, 10, 36]. Inflammatory hyperpermeability of the pulmonary vessels is thought to be the main aetiological factor [15]. The vapour products of PTFE pyrolysis are potent irritants and contribute to inflammatory processes. One of the earliest theories, put forward in 1972, was the release of endogenous pyrogens by leukocytes in response to the attack of alveolar tissue by PTFE particles, explaining both the respiratory symptoms and the development of a febrile state. The hypothesis of a non-specific inflammatory reaction is based on allergic manifestations caused by the activation of alveolar macrophages and the release of inflammatory proteins (tumour necrosis factor α, interleukins 6, 8) upon contact with PTFE pyrolysis products. Type II pneumocytes can metabolise xenobiotics and produce surfactant. They proliferate in response to aggression and are activated during recovery, playing a role in the development of tolerance. The phenomenon of resistance after repeated toxic exposure has been identified in laboratory animals, but this theory has not been confirmed in clinical trials [10]. Polymer fever in humans occurs independently of previous contact: it does not cause tachyphylaxis and therefore, unlike metal fume fever, there is no particular susceptibility on Mondays [2–4, 37].

The action of free radicals is primarily caused by PTFE pyrolysis products like perfluoroisobutylene, which leads to oxidative stress and cellular damage in the lung with the development of microscopic exudative oedema 27 minutes after exposure. The formation of free radicals, such as hydrogen peroxide, as a result of chemical reactions explains the damage to the alveolar-capillary barrier [3, 4, 10, 35, 38].

Clinical manifestations. Polymer fume fevers are usually benign with spontaneous resolution of symptoms, although they may be associated with dangerous manifestations, especially in patients with preexisting severe cardiorespiratory disease [3, 4, 6].

Polymer fume fever usually presents as a mild flu-like illness characterised by fever (39–40 °C), sweating, dyspnoea, chills, sore throat, myalgia, tachycardia (up to 120 beats per minute), shortness of breath, chest tightness, non-productive cough at rest and headache as the main symptoms. All smokers reported mild inspiratory discomfort behind the sternum, cough and bad taste in the mouth during toxic exposure. Shortly after the cough subsided, they experienced back pain. The shivering started after 30 minutes and lasted for 2 to 3 hours [20]. Some publications highlight the onset of symptoms within the first few seconds after ingestion of Teflon-contaminated cigarettes; other authors report a delay in clinical manifestations of 1 to 24 hours [1, 2, 10, 15, 36].

Since its first report in 1951, polymer fume fever has been presented with a variety of clinical manifestations, ranging from flu-like symptoms to severe toxic effects, such as pulmonary oedema, pneumonitis and death, with the severity of the disease depending on the specific conditions and duration of exposure to the toxins. With a sufficiently high pyrolysis temperature and/or prolonged toxic exposure, severe lung damage, including radiological consolidation, is a potential complication [1, 5, 6, 8].

PFIB is more irritating to the skin, eyes, nose, throat and lungs than other Teflon pyrolysis products and is ten times more toxic than phosgene. It is an extremely toxic gas that affects the lungs when inhaled. The latency period of PFIB damage is one to four hours before symptoms of pulmonary oedema appear [17]. Inhalation of small amounts of the gas can cause acute lung damage and is a major cause of morbidity and mortality in critically ill patients. Higher doses cause pulmonary oedema with wheezing, dyspnoea, sputum production and cyanotic skin discolouration. The toxicity of PFIB is similar to that of other fluorolefins. It is directly proportional to the reactivity of this olefin with nucleophiles, resulting in pulmonary oedema, pneumonitis and death [17, 19, 36]. In the experiment, PFIB induces pulmonary oedema, including translocation of blood proteins into the alveoli. High-performance capillary electrophoresis of lung proteins showed that albumin, transferrin and IgG are the three main proteins that diffuse into the alveolar space. At an early stage, inflammatory hyperpermeability of the pulmonary vessels after PFIB exposure leads to damage to the tight junctions of cells, which in combination with subsequent changes in actin leads to an increase in the permeability of the vascular-alveolar barrier. Transbronchial lung biopsy in a patient with PTFE smoke-induced pulmonary oedema showed marked neutrophil migration into the oedematous alveoli. Significant neutrophil infiltration and elevated levels of inflammatory cytokines have been found in lung lavage from laboratory animals exposed to PTFE vapour. Both cases are consistent with pathological data on the exudative phase of acute respiratory distress syndrome (ARDS) [2, 15, 17, 19]. In severe polymer fume fever, ARDS was present in 56 % of cases, accompanied by multiple dry rales and sonorous rhonchi and a corresponding radiological picture [3, 4, 15].

Exposure to Teflon products by inhalation can cause severe symptoms of non-cardiogenic pulmonary oedema, manifested by wheezing, dyspnoea and sputum production. A cyanotic skin colour may also be observed. Initially, there may be a cough and chest pain. However, the dangerous symptoms of pulmonary oedema can persist for several hours before a sharp deterioration in health occurs [2, 17]. Chest radiographs sometimes show diffuse infiltration of the lung fields in the form of bilateral reticulonodular patterns [10]. Radiological signs of pulmonary oedema caused by PTFE vapours may be bilateral opacities or patchy consolidation

with clear preservation of the peripheral area. This is because it is harder for toxic fumes to reach the peripheral alveoli, thus avoiding inflammation in these areas. Another explanation is related to the peculiarities of pulmonary lymph flow. Small particles in PTFE vapour can be removed by the lymphatic drainage system directly or by phagocytosis and macrophage migration. Lymph moves in two opposite directions: centripetally in the centre of the lung and centrifugally in the periphery. Centrifugal lymphatic drainage in the periphery of the lung may effectively remove PTFE particles to the pleural lymphatic pathways rather than centrally to the pulmonary gates.

Infections complicating polymer fever due to perfluoroisobutylene exposure are common and require blood cultures, but the WHO does not recommend antibiotic prophylaxis for PFF [10].

The content of fluoride anion in the pyrolysis products, which binds tightly to Ca^{++} ions to form fluorapatite, causes the development of hypocalcaemia and hyperkalaemia. With prolonged toxic exposure, hypocalcaemia can cause cardiac arrhythmias up to and including cardiac arrest in the diastolic phase [15].

Repeated poisoning with PTFE pyrolysis products can lead to chronic obstructive pulmonary disease, occupational asthma or pulmonary fibrosis [3, 4]. A growing body of evidence also suggests that chronic occupational exposure to unheated PTFE aerosols may put workers at risk of granulomatous lung disease [2, 7, 10, 26, 27].

Although the diagnosis of polymer fever is clinical, it is advisable to obtain a complete blood count and chest X-ray. The diagnosis of PFF requires a clear history that specifically identifies the relevant exposure to fluoropolymer degradants and rules out other potential causes [2, 10].

Doctors in emergency departments are obliged to consider cases of polymer fume fever according to severity of clinical manifestations. Because of the lack of knowledge about its existence the diagnosis is not verified. Sometimes, when a toxic cause remains unsuspected, unnecessary invasive tests are performed. There is a risk that the patient remains intoxicated and may develop pulmonary oedema or ARDS [1, 10].

Treatment. The decision to hospitalise should be based on the timing and severity of clinical manifestations and toxic effects. It is advisable to closely monitor patients with significant and prolonged toxic exposure, as it may take some time for maximum symptoms to develop [2]. In view of this, the risk of acute pulmonary oedema and ARDS in Teflon fever requires in-patient observation of victims [10].

The typical course of polymer fume fever is mild and self-limiting. Treatment is supportive and should aim to relieve symptoms. Fever can be effectively controlled with antihistamines, antipyretics, such as paracetamol, aspirin or non-steroidal anti-inflammatory drugs. Bronchospasm is effectively treated with inhaled beta-agonists or glucocorticoids. Patients with hypoxia should receive oxygen therapy [2–5, 10, 15, 18]. Cardiac glycosides should be prescribed according to indication [18].

There are currently no specific antidotes for PFIB. In the experiment, prophylaxis with N-acetylcysteine (N-ACC) significantly improved the survival of animals and significantly reduced the wet lung/body weight ratio, protein and phospholipid content in alveolar exudate. N-acetylcysteine can regulate the redox system in lung tissue and effectively protect the target organs of treated animals. Protection against the lethal effects of inhaled PFIB was demonstrated when N-ACC was administered orally 4, 6 or 8 hours prior to exposure, and the duration of protection was related to the timing of an increase in plasma cysteine, glutathione and N-ACC levels [17, 38]. A promising therapeutic approach in the early stages of acute respiratory distress caused by PFIB is the combined use of N-ACC and the natural surfactant curosurf intratracheally. Treatment of PFIB poisoning is based on reducing pulmonary oedema by administering diuretics. Furosemide and torasemide have limited the oedema, type and severity of pathological changes associated with PFIB inhalation and delayed the fatal outcome. Cholinolytics may play a preventive and therapeutic role in Teflon fever. Hyoscyamine 0.5 mg orally has been used as a model cholinolytic. There are some reports of a beneficial effect of octreotide (sandostatin) subcutaneously 0.1 mg every 8 hours [17].

A common mistake made by doctors is antibiotic prophylaxis for PFF or beta-agonist treatment in suspected pneumonia or decompensated exacerbation of chronic obstructive pulmonary disease [10]. The use of antibiotics is indicated only in cases of confirmed bacterial pneumonia [19].

In case of ARDS or severe non-cardiogenic pulmonary oedema, non-invasive ventilation with two-stage positive airway pressure or endotracheal intubation with mechanical ventilation may be required [2, 15, 39].

Japanese researchers have demonstrated the expediency of intravenous use of a reversible competitive inhibitor of neutrophil elastase that does not affect the function of other human proteases, sivelestat (elaspol) at a dose of 4.8 mg/kg/day, administered under conditions of positive end-expiratory pressure of 8 cm H_2O, in cases of poisoning with Teflon pyrolysis products [15].

A thorough analysis of the workplace exposure should be carried out by an occupational physician or clinical toxicologist in conjunction with a qualified occupational hygienist [3, 4, 17].

Prognosis. Polymere fume fever is usually benign, and most patients make a full and rapid recovery. Symptoms generally peak within 24 hours of toxic exposure and gradually resolve within 24 to 48 hours. The severity of the condition is directly related to the pyrolysis temperature and exposure time. Even if patients experience more severe symptoms like pulmonary oedema, full recovery is expected within 5 to 10 days. Permanent lung damage, such as pulmonary fibrosis, is rarely described and appears to be limited to those individuals who are repeatedly exposed to toxicity. Death from polymer fever and permanent disability are quite rare [1, 2, 5, 6, 8, 10, 15].

Conclusions

1. The widespread use of polymeric products in the workplace and at home can have a significant negative impact on public health in case of accidents or misuse.

2. Polymer fume fever is a rare, flu-like, mostly occupational disease that is not always diagnosed in time.

3. A better understanding of the pathological process enables clinicians to make the correct diagnosis, educate patients and ultimately achieve better treatment outcomes.

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Полімерна лихоманка (огляд)

Резюме. Політетрафторетилен є надзвичайно хімічно інертним, але його розкладання шляхом піролізу, тобто реакції неповного згоряння (безокиснювальної термічної деградації), може призвести до швидкого виникнення грипоподібних симптомів при вдиханні кінцевих продуктів. Цей стан зветься полімерною лихоманкою і найбільш часто зустрічається в курців. Гіпотеза про транспорт молекул фтористоводневої кислоти наддрібними наночастинками, присутніми в повітрі, є єдиною, що наближається до пояснення цього явища. Іноді, коли токсичну причину не підозрюють, інвазивні тести проводяться без необхідності. Ризик полягає в тому, що пацієнт продовжує перебувати в стані інтоксикації і можуть розвинутися набряк легенів або гострий респіраторний дистрес-синдром. Специфічних антидотів проти продуктів піролізу тефлону не існує. Поширене використання полімерних виробів у промисловій та побутовій сферах може мати значний негативний вплив на здоров'я населення в разі аварій або неналежного використання. Полімерна лихоманка є рідкісним грипоподібним, здебільшого професійним захворюванням, яке не завжди діагностують вчасно. Краще розуміння патологічного процесу дозволяє клініцистам ставити правильний діагноз, належним чином навчати пацієнтів і, зрештою, досягати кращих результатів лікування.

Ключові слова: політетрафторетилен; продукти піролізу; полімерна лихоманка; діагностика; лікування