Chemical pneumonia due to air pollution after New Year’s fireworks in 2016 in Stuttgart, Germany: A case report

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Abstract
This case report describes chemical pneumonia experienced by myself (male, 34 years old at the time) caused by inhalation of polluted air after New Year’s fireworks in 2016 in Stuttgart, Germany. The toxic exposure resulted in dyspnea, chest pain, and coughing for several days as well as changes in blood parameters indicating inflammatory reactions associated with chemical pneumonia. This case highlights the detrimental effect to human health of acute exposure to air polluted by exploded fireworks.

Introduction
The explosion of fireworks causes intense air pollution due to the release of particulate matter (PM) consisting of toxic substances. Toxins include trace metals such as chromium [1] [2], titanium [2] [3], iron [2] [4], copper [2] [3], aluminium [2] [4] [5] and nickel [6], as well as lead [2] [3] [6] [7] [8] [9], manganese [2] [3] [6] [9], manganese dioxide [10], barium [2] [3] [5] [7] [11], strontium [2] [3] [5] [7] [8] [9], magnesium [3] [4] [5] [7] [8], calcium [2] [6], zinc [2] [3] [6] [9], sodium [2], vanadium [9], lithium [12], bismuth [3], arsenic [3] [9] and cobalt [3] [6]. In addition, sulphur dioxide and nitrogen dioxide [13], dioxins [14] [15] [16], fluorine [12], sulphate [3] [12], chlorobenzenes and chlorophenols [15] have been detected in air polluted by fireworks alongside extractable organic compounds such as alkanes, hopanes and polycyclic aromatic hydrocarbons [2]. The size of the PM is generally in the micrometer range, but novel fireworks also use nano-powders, resulting in PM in the nanometer range [11] [17]. This can increase the toxicity of PM further due to the ability of nanoparticles to reach the deepest alveolar area of the lungs, penetrate cellular membranes and bypass detoxification mechanisms of the human body [18] [19] [20] [21] [22].

Air pollution due to PM from fireworks is not only a problem in terms of environmental pollution but also with regard to its impact on human health. The high concentration of PM can cause acute and/or chronic negative health effects in humans. This is especially true given that the PM is composed of substances which are toxic even at low doses individually, let alone in combination with others (e.g. Pb [23] [24], Al [25] [26] or Ba [27]).

The negative effects of PM pollution on human health is well known [28] [29] [30], and the impact of air pollution on the population’s health as a result of PM due specifically to firework explosions is increasingly recognized [31] [32]. Unfortunately, there are currently no large toxicological studies about the effects (especially the long-term effects) of firework combustion products on human health. Legal frameworks regulating the use of fireworks are also insufficiently implemented or enforced to date, despite calls for action from the scientific community [11] [32] [33].

The intensity of air pollution in a city due to fireworks depends on the volume of fireworks, the local meteorological conditions and the geographical topology of the area. Some cities have a geographical topology that hinders the sufficient exchange of air between the city and the suburbs. Stuttgart in southern Germany is one such place. In case of a specific meteorological condition termed “inversion” (i.e. a temperature inversion with cold air at the ground and an increased air temperature with altitude), convection is suppressed in the city, which, in combination with the specific geographical topology, causes severe air pollution. The problem of air pollution in Stuttgart is well known [34] [35]. Stuttgart has the highest air pollution (as of 2015, according to the German-Federal-Parliament [36] [36]) in Germany with respect to PM with a diameter of ≤10 µm (PM10) and nitrogen dioxide.
Objective
Here I present a case of chemical pneumonia experienced by myself due to air pollution caused by New Year’s fireworks in 2016 in Stuttgart.
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Particulate matter PM10 concentration (Stuttgart)

Lead-214 air concentration (Stuttgart)

Dyspnea

Cough

Chest pain

PM10 (Stuttgart)

PM10 (Zurich)

Lead-214 [Bq/l]

Dyspnea severity [0, 1]

Cholesterol

Chest pain severity [0, 1]

Cough severity [0, 1]

Lymphocytes [%]

Monocytes [%]

Eosinophils [%]

Basophils [%]

Neutrophils [%]

Leukocytes [10^9/l]

Creatinine [µmol/l]

Creatinine clearance [%]

Urea [mmol/l]

Hb [g/l]

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Figure Legend

Figure 1. Maps of the air pollution by particulate matter in Germany, development of the air pollution around New Year in Stuttgart, development of the symptoms and results of the blood analysis.

(A-D) Maps of the air pollution by particulate matter (PM10) in Germany from December 31, 2016, to January 2, 2017. Images created by the Umweltbundesamt (UBA), the central environmental agency of Germany. The green circle and the black arrow indicate the location of Stuttgart. Figure (D) is similar to (B) but with an increased scale (0–150 µg/m³) to highlight the intensity of air exposure in Stuttgart and Munich.

(E) Development of the PM10 concentration around New Year in Stuttgart (at 2 measurement locations: Neckartor and Bad Cannstatt). The blue horizontal line indicates the current EU safety limit. The grey vertical bars indicate the time I spent outdoors.

(F) Lead-214 concentration in the air of Stuttgart around New Year (measurement location: Stuttgart TV tower).

(H) Daily PM10 average values for Stuttgart (measurement location: Bad Cannstatt).

(I) Daily PM10 average values for Zurich (measurement location: Kaserne). The symptom strengths of dyspnea, chest pain and cough are shown in (G), (J), and (K) respectively.

(L-W) Results of the blood analysis performed about 96 h after the initial toxic air exposure (January 5, 2016) in comparison to the individual reference ranges (i.e. the mean ± standard deviation of the values obtained by blood tests performed during the previous two years, n = 6). Abnormally high or low values with respect to the individual reference ranges are marked with a red circle.

Results & Discussion

The author of this publication was motivated to write a case report about himself after experiencing the symptoms described. The subject is a male (34 years old at the time) with a body mass of 67 kg, height of 179 cm, body mass index of 20.9 kg⋅m⁻²; and – prior to the chemical pneumonia – blood parameters corresponding to the general health state [values of 2016 (n = 2), mean ± standard deviation]: homocysteine: 11 ± 1 mol/l, ferritin: 268 ± 17 µg/l, transferrin saturation: 82.2 ± 10.2%, atherogenic index of plasma: -0.28 ± 0.1, high-sensitive C-reactive protein, hs-CRP: 0.2 ± 0 mg/l]. In this case report, the subject provides a first-person account of events and symptoms.

In the past, I did not suffer from any lung disease and my health condition is generally good (despite having hereditary hemochromatosis [diagnosed 2015] and experiencing the Flammer syndrome [37]).

After traveling from Zurich (Switzerland; 47.3769° N, 8.5417° E) to Stuttgart (Germany; 48.7758° N, 9.1829° E) on December 31, 2016, and spending the rest of the day (including the night) indoors, I went on a 3 h walk on January 1, 2017 (from approx. 9:30 am to approx. 1:30 pm). I began my route in the west of Stuttgart, before walking up a local hill (Hasenberg, height: approx. 450 m) and returning back to my starting point. It should be noted that I was carrying my then 6-month-old son (weight: approx. 8 kg) in a baby carrier at the time, which increased the physical exertion. During this time, Stuttgart was affected by severe air pollution due to the New Year’s fireworks. The air pollution was especially strong in the streets between tall buildings preventing air exchange. The air pollution in Germany due to New Year fireworks was particularly bad in Stuttgart and in the area around Munich (Fig. 1A-D). The strong air pollution in Stuttgart after New Year was due to a combination of the combustion and explosion gases from the fireworks, and the special weather conditions, i.e. an atmospheric inversion which prevented the exchange of air in the city situated in a basin. Air pollution in Stuttgart over the New Year period peaked on January 1, (Fig. 1E and 1H), daily average of PM10 from January 1: 174 µg/m³; as well as the lead-214 concentration values in the air (Fig. 1F).

After about 15 min into my walk, I felt chest pain which increased in intensity; in parallel I increasingly experienced dyspnea. Also, I needed to cough multiple times. After returning at home, the symptoms persisted. Over the next 2 days, I spent only 2 short periods outdoors and finally returned to Zurich on the second day after the initial exposure (i.e. on January 3, 2017). Compared to Stuttgart, the air pollution levels were lower in Zurich on New Year’s Day and afterwards (Fig. 1I-1H).
Figures 1(G), (J) and (K) visualize how the strength of the symptoms (dyspnea, chest pain, and cough) developed during the next 7 days. Dyspnea was strongest on the first day of the exposure to the toxic air and the symptoms disappeared almost completely after 7 days. Chest pain was strongest on the day of exposure and declined rapidly. In general, the chest pain was strongest every time I took a deeper breath than normal. The urge to cough was also strongest on the first day of exposure and got weaker in the following days. The cough was dry.

On January 5, (i.e. 4 days (96 h) after the initial toxic air exposure) a blood analysis was performed to assess my health state. To see if there was a change in my overall health state I compared the new blood parameters values to my individual reference ranges, i.e. the mean ± standard deviation of the values obtained by blood tests performed during the previous 2 years (n = 6). Deviations of the new values from these individual reference ranges (i.e. a value below or above the standard deviation) was considered as abnormally low or high, respectively. With this approach, individual-specific changes of the blood parameters could be assessed, independent of the general laboratory normal ranges.

The complete blood count showed the following picture of the white blood cell parameters (Fig. 1L-W, Fig. 1I) no abnormal deviation regarding the absolute values of the leukocytes (5.3 G/l, individual reference range [IRR] = 5.5 ± 0.3 G/l), neutrophils (2.6 G/L, IRR = 2.6 ± 0.2 G/l), lymphocytes (2.1 G/l, IRR = 41.7 ± 2.4 G/l), monocytes (0.3 G/l, IRR = 0.3 ± 0.04 G/l), or eosinophils (0.1 G/l, IRR = 0.1 ± 0.05 G/l), but (ii) abnormally high values of the absolute basophil counts (0.1 G/l, IRR = 0.02 ± 0.04 G/l) and differential counts (1.7%, IRR = 0.8 ± 0.2%), as well as a slight elevation of the differential counts of monocytes (5.4%, mean = 4.9 ± 0.4%).

In addition, hs-CRP was increased (0.4 mg/l, IRR = 0.28 ± 0.07 mg/l), as well the international normalized ratio (INR) (1.11; 1.07 ± 0.02) and ceruloplasmin (0.2 G/l; 0.18 ± 0.01 G/l). Interestingly, the iron-related parameters also show a statistically significant deviation from the individual reference ranges: a decrease in the unsaturated iron-binding capacity (2.69 μmol/l, IRR = 9.98 ± 5.32 μmol/l), an increase in the transferrin saturation (94.4 %, IRR = 78.88 ± 11.51) and a slight increase in the serum iron concentration (45 μmol/l, IRR = 38.13 ± 7.47 μmol/l).

The dyspnea symptom became weaker in the following days. 1 week after the intoxication event, the dyspnea symptoms had almost vanished (but were still significantly recognizable during physical activity which induced deeper breathing). Coughing decreased to just a few times per day, and the chest pain vanished completely. There was a relapse of the symptoms 11 days after the incident (January 12): While traveling by airplane from Zurich to London, the cabin air began to smell like exhaust gas after landing. About 30 s after recognizing the chemical smell, the dyspnea and chest pain symptoms started and peaked in the time span about 3–10 min afterwards. The chest pain then faded away but the dyspnea remained for about 1 h. Similar symptoms were experienced again 15 days after the initial exposure (January 16): Dyspnea symptoms started after spending about 15 min in a research laboratory, where the air had a “chemical” smell (possibly due to some previous soldering, the running of technical machines and/or some painting work that was going on outside). After leaving the room, the symptoms declined over the following 2 h.

In the following months, several instances where noticed with difficulties of breathing or shortness of breath, i.e. dyspnea, during exercise. In addition, a higher sensitivity to odors, especially cigarette-smoke and perfume, was noticed.

2–3 months after the toxic air exposure it was noticed that the number of hemangioma (mainly on the chest and back) increased. One of the hemangioma appeared on the inner part of the upper leg (over the biceps femoris muscle) and increased in size very rapidly. As the hemangioma was bleeding and causing pain it was removed surgically (May 2017) but returned, increased in size (diameter: 5 mm) and had to be removed with a deeper removal of the skin and tissue a few months later (August 2017). The histological analysis revealed that it was a benign tumor.

The symptoms (dyspnea, chest pain, and cough) experienced after the inhalation of polluted air due to fireworks, as well as the changes in blood parameters, indicate chemical pneumonia. The blood parameters revealed inflammatory reactions, as evidenced by
slightly elevated hsCRP, differential counts of monocytes, basophile counts, INR and ceruloplasmin. A leukocytosis, however, was not prevalent in the blood results—possibly due to the relatively long delay between exposure and blood analysis (4 days), preventing the detection of the increased leukocyte concentrations in the acute phase. Also, the moderate changes of the other blood parameters might be due to the 4 day delay between exposure and analysis.

Unexpectedly, there is only one report available about chemical pneumonia associated with inhaled polluted air from fireworks. Hirai et al. [38] reported the case of a man who inhaled smoke from fireworks for 3 consecutive nights and developed cough, fever, dyspnea, and acute eosinophilic pneumonia (AEP) associated with leukocytosis, hypoxia and increased eosinophils in bronchoalveolar lavage (BAL) fluid. Compared to my case, leukocytosis was also evident in my blood analysis (4 days after the exposure) while eosinophils in BAL fluid was not assessed, meaning that no conclusion can be drawn as to whether AEP was initially also present in my case.

Interestingly, my increased levels of basophils have also been reported in humans exposed to metal fumes [39].

The observed increase of ceruloplasmin in my case also shows that the event caused an inflammatory reaction (ceruloplasmin being an acute-phase reactant and endogenous antioxidant [40] [41]) and may also indicate a detoxification reaction of the body due to the increased presence of copper in the blood caused by the inhaled air polluted also with copper [2]. An increase in ceruloplasmin is also documented in cases of asthma and chronic obstructive pulmonary disease [42], supporting the conclusion that the observed increase of ceruloplasmin in my case is indeed associated with an anti-inflammatory response to the inflammation of the airway.

The changes in the iron panel, i.e. increase in plasma iron and transferrin saturation alongside decrease in unsaturated iron-binding capacity, indicate also an iron poisoning. That the polluted air from fireworks also contain PM with iron has been reported [2] [4].

My observed increased sensitivity to air pollution weeks and months after the actual intoxication event is in line with reports of increased bronchial reactivity after exposure to polluted air, i.e. the dust of the World Trade Center explosion at the terrorist attacks of 9/11 in 2001 [43] [44] [45] [46] [47]; in particular, a persistent hyperreactivity and reactive airway dysfunction was observed in the people exposed to this toxic dust [48]. The case where the air inside the airplane after landing caused dyspnea and chest pain symptoms is in line with the inhalation of polluted air with toxic fumes in the airline cabin (aerotoxic syndrome) [49] [50] [51].

The appearance of hemangioma in the months after the exposure may not be coincidental since an increased incidence of hemangioma was reported to be associated with exposure to toxic substance, e.g. bromides [52], propylene [53], cadmium chloride [54] or sulfur mustard [55]. The inhalation of the toxins in the polluted air due to the firework explosions may have triggered the occurrences of the hemangiomas in my case.

Conclusions

In conclusion, this report presents a case of chemical pneumonia with subsequent health-related effects due to air pollution caused by New Year’s fireworks in 2016 in Stuttgart.

This case highlights the detrimental effect of an acute exposure to polluted air from exploded fireworks to human health and underlines the necessity for governments to regulate (more strictly) the usage and chemical composition of fireworks.

Limitations

There are 2 limitations regarding the assessment of the possible chemical pneumonia: (i) the blood parameters were determined a few days after the intoxication event missing the strongest changes that possibly happened immediately after the event; (ii) no spirometry of other direct lung function test was performed.

Additional Information
Citations


