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Case reports

Multiple occupational hazards, multiple occupational diseases - a study case

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Abstract

Industrial workers frequently have exposure to multiple hazards. Depending on the specific activity the intensity, duration and type of exposure may differ during the entire professional life, even in the same working place. These workers are at risk of developing several work-related disorders or even multiple occupational diseases. This study presents a case of a welder who performed heavy work (lifting and handling masselottes and metal casts) and was exposed to gases, fumes and vapors. He was diagnosed 8 years before with occupational lumbar discopathy and occupational pulmonary fibrosis (welders' lung) based on symptoms and X-ray examination. He continued to work in the same workplace. At the current admission the respiratory symptoms (dyspnea on low exertion, wheezing, cough with seromucous sputum) were more severe and a large opacity of 5.3/4.3 cm slightly heterogeneous structure, with spiculated, iodophilic contour in lateral area of the subpleural segment of the upper right pulmonary lobe was found on the CT scan. This opacity was interpreted as probable lung cancer and the patient was sent for surgery.

The case illustrates the multiple health consequences of the working activities and the necessity to evaluate in a proper manner all possible hazards present in a certain working place.

Keywords: *welding, fibrosis, lumbar discopathy, pulmonary cancer*

Introduction

Depending on the working conditions, a person can be exposed to multiple occupational hazards and each of these hazards should be assessed for their possible health consequences. Recognition of one occupational disease does not exclude the diagnosis of another occupational disease, especially when the exposure refers to hazards with very long latency periods, such as carcinogens.

Case study

The present study reports an occupational medicine case of a worker exposed to multiple occupational hazards.

A 55-years old welder was admitted in the Occupational Clinic with dyspnea on low exertion, wheezing, cough with seromucous sputum for about 2 years, low back pain with irradiation to the left lower limb, up to the level of the fingers, accompanied by paresthesia, thoracic pain, nocturia, polyuria and interrupted, two-stage urinary stream. His clinical picture was dominated by the respiratory symptoms, which were worse than the previous year.

He had a total exposure to welding hazards for 20 years; from 1986-1987, 1988-1992 and continuously (15 years) from 2008 to present. Currently, he was carrying out his activity in the cast iron and steel foundry, where he performs the cutting of the masselottes into smaller pieces and the iron and steel castings by autogenous welding. He worked in a very large industrial hall with poor general ventilation, dusty, with known exposure to silica. Previous cases of silicosis were recorded from this workplace, in founders. In the last three years he worked outdoors, performing the same activities as when he worked indoor. To protect locomotor system overload the masselottes are transported by crane, and the smaller parts with the help of the magnetic plate.

During his professional activity was exposed and still is to the following occupational hazards: welding gases and fumes; crystalline silica dust; noise; manual weight handling; prolonged orthostatism and unfavorable microclimate conditions. The patient used the protective gloves without the inner lining because it bothered him. By removing this layer of material his hands came into direct contact with the rigid structure of the protective glove and he developed contact dermatitis over time.

The medical history of the patient showed that the first respiratory signs started 24 years after the first exposure, in 2010, with dyspnea on exertion. The patient was a smoker of 20 PA and was also diagnosed with work-related chronic bronchitis. Three years later, in 2013, the X-ray confirmed occupational pulmonary fibrosis.

In the 2013 hospitalization, he was also diagnosed with occupational lumbar discopathy L5-S1. The onset of osteoarticular pain was in 2009, and at the present admittance the X-ray did not show any progression.

In 2021 he was diagnosed with allergic contact dermatitis to cobalt and chromium salts and allergy to perfumes and methyl dibromo glutaronitrile (from textile dyes). There were also other comorbidities identified in his medical record such as recurrent maxillary sinusitis, hypercholesterolemia, hypotonic biliary dyskinesia, right renal microlithiasis, cervical spondylarthrosis, cervical discopathy C2-C3, C4-C5 and C5-C6.

At the current clinical examination the followings were noticed: punctiform scars on both calves post-welding, abdominal scar of about 10 cm after a home accident, bilateral peri malleoli venectasias, bilateral halluces with onychomycosis, a normal BMI (24.16), thoracolumbar kyphoscoliosis, lumbar spine straightness, pain on percussion of the lower thoracic and lumbar spinous processes, cracking on active mobilization of the bilateral shoulder, pain on internal rotation of the left coxo-femoral joint, bilateral trapezius muscle contracture and dorso-lumbar paravertebral muscle contracture. At the time of the examination the respiratory system presents bilaterally symmetrically transmitted vocal vibrations, diminished bilateral vesicular sounds and wheezing. The cardiovascular system showed a regular rhythm (72 beats per minute), no murmurs, no turgescent jugular, normal pulse in peripheral arteries and blood pressure in normal limits (132/89 mmHg). Neurologic examination showed a positive sensitized Romberg maneuver, positive 45-degree Lasègue test on the lower left limb with hypoesthesia on the left external popliteal sciatic nerve route, left upper limb paresthesia. The patient has optical correction at near and at distance.

The lab tests were positive for inflammation (the erythrocyte sedimentation rate = 43 mm/1h; C-reactive protein = 18.19 mg/L; Fibrinogen = 576 mg/dl 200-400 mg/dl). Bacteriological examination of sputum was negative and other investigations were in normal limits.

Posteroanterior and lateral radiography (Figure 1) of the chest revealed oval, ill-defined opacity of medium intensity with a diameter of 4/3 cm in the left upper lobe, hilio-basal linear opacities of bilateral basal interstitial fibrosis, hilums with increased projection and intensity, thickened horizontal straight split, free costo-diaphragmatic sinuses and normal cord.

Due to the allergic risk, CT scan with administration of intravenous contrast material could not be performed. A native chest CT (Figure 2) showed a diffuse, bilateral, fibrotic-looking moderately accentuated pulmonary interstitium associated with diffuse reduction in transparency. Several emphysematous bullas in the lateral segment of right upper lobe, apical left upper lobe, right and left lower lobe and Fowler segment

were described. These findings were interpreted as changes associated with the chronic interstitial lung disease. There was also several tubular bronchiectasis in the right and left lower lobe, but with no pleural dressings, tracheobronchial tree of normal caliber and without wall thickening.

A significant radiological finding was the large opacity of 5.3/4.3 cm located in lateral area of the upper right pulmonary lobe, in the subpleural segment. The opacity had a slightly heterogeneous structure, with spiculated, iodophilic contour, suggestive of an expansive process.

Figure 1. Chest X-ray

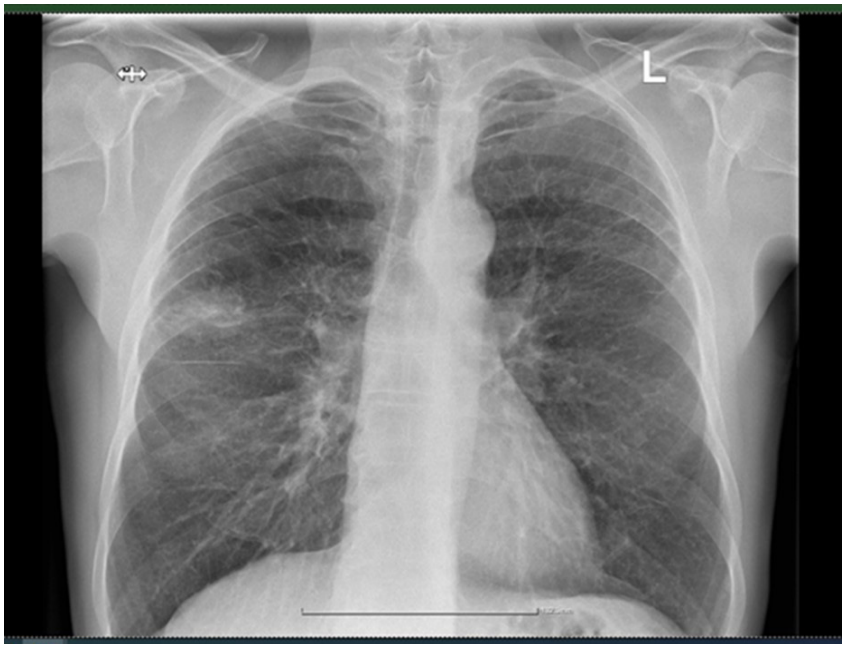
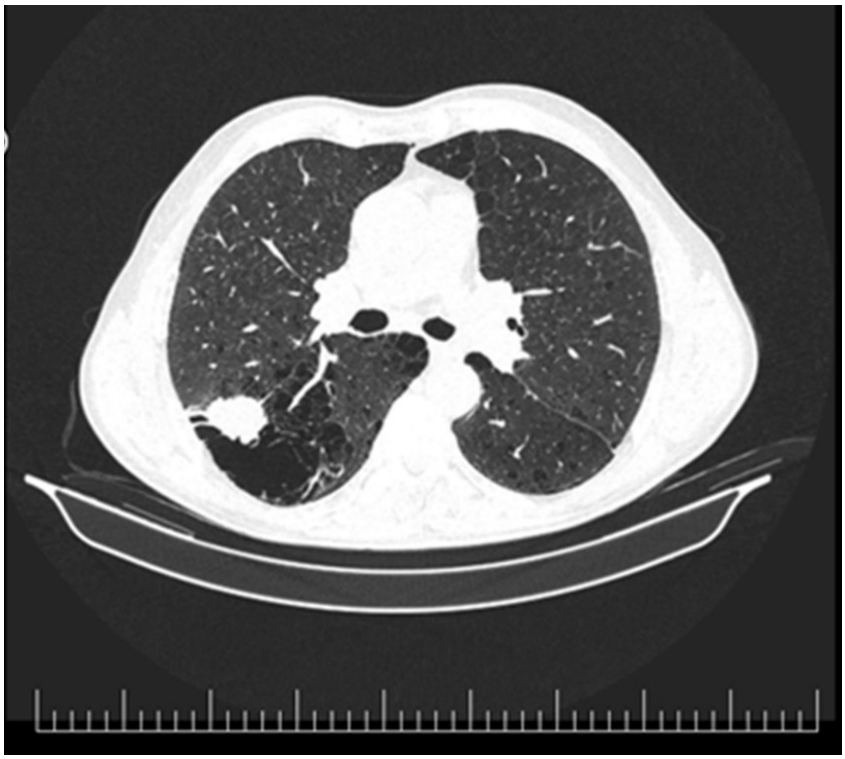
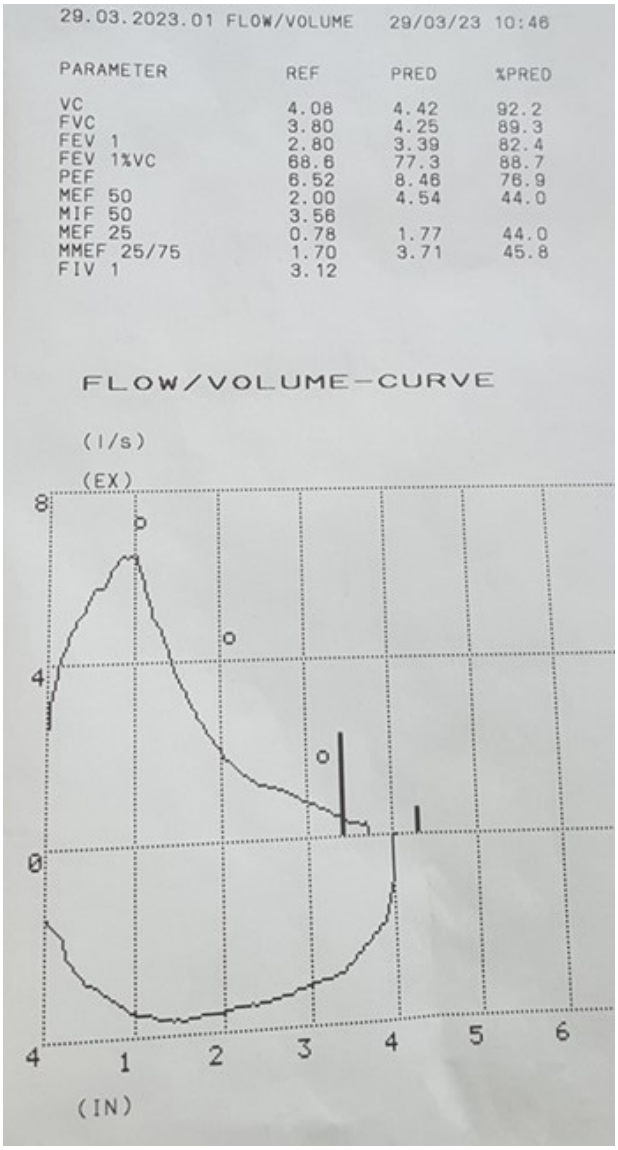


Figure 2. Image of the CT scan



Pulse oximetry at rest showed SaO2=98%. In spirometry (Figure 3), a distal obstructive syndrome was revealed. The vital capacity (VC) was 92,2% predictive, the expiratory volume in one second FEV1=82,4% predictive and the FEV1/FVC ratio FEV1%VC=68,6%, MEF50=44,0% predictive. The results were comparable to the ones recorded 6 years ago, when the diagnosis of chronic bronchitis was established and for which he was treated with long-acting bronchodilator and respiratory physiotherapy.

Figure 3. Spirometry



Other investigations were within normal limits, with a few changes highlighted by abdominopelvic ultrasonography: an enlarged liver (140/67 mm) with hyperechoic structure and high posterior attenuation and also the prostate was slightly enlarged, nonhomogeneous with calcifications.

The subsequent urological consultation established a benign prostate hypertrophy and chronic prostatitis for which treatment was recommended for 1 month and with subsequent control.

Discussion

Considering all the occupational hazards, this welder had 2 occupational diseases in his medical history: lumbar discopathy due to heavy lifting, welders' fibrosis (possible mixt dust pneumoconiosis due to the silica exposure). The third one (occupational cancer) was the challenge of the current admission, as large opacities in the lung imaging might be due to lung cancer or complicated silicosis both being possible consequences of the silica exposure and less frequent after exposure to the welding fumes. In this case, the CT scan was highly suspicious for cancer, which was indeed confirmed by lung biopsy.

Crystalline silica dust is classified as human carcinogen, group 1, from 1997 [1]. In 1989, welding fumes were classified by International Agency for Research on Cancer (IARC) in group 2B as "possibly carcinogenic to humans", but from 2017, welding fumes and ultraviolet radiation from welding are classified as group 1 carcinogens [2]. Crystalline silica dust and welding fumes are among the top 8 occupational carcinogens, according to 2019 statistics from the UK [3]. An extensive meta-analysis of 85 studies confirms the carcinogenic role of crystalline silica dust on the lung. Although a decrease in this risk is observed in newer studies, it remains high especially for silicosis patients, the pooled risk estimate was 2.32 (95 % CI 1.91–2.81) for SMR studies and 2.49 (95 % CI 1.87–3.33) for SIR studies [4].

The significant elevated risk was found also in those with low exposure (cumulative exposure <0.4 mg/m3-years), with no apparently threshold, but with a tendency to lower the risk after cessation of exposure. The relation between exposure and lung cancer risk seems to be monotonous [5]. The calculated increased lung cancer risk for every 1 mg/m3-year increase in was 1.06 (95% CI, 1.04–1.08), regardless smoking. Nevertheless, in this pooled case-control studies, the addictive effect of smoking was estimated for lung cancer and, separately, for each of the three major types of lung cancer; the multiplicative effect was noticed only for the overall cancer risk, probably due to the small number of cases in some subgroups. This study confirmed a meta-analysis result from 2014 showing a multiplicative risk by simultaneous exposure to cigarette smoking in patients with silicosis (RR= 4.47; 95% CI, 3.17-6.30) [6]. Thus, for the patient in the case study, a smoker of 20 PA, the probability that the pulmonary opacity was lung cancer was, indeed, very high.

Different processes and metals welded, as well as the extent of exposure and the conditions of the working place may impact the lung cancer risk. Initial studies showed an elevated risk only in smoking welders [7], but a later meta-analysis from 2006, which includes 60 studies from 14 countries, found an estimated lung cancer risk among welders, as compared with non-welders, of 1.26, without any difference according to the tip of welding [8]. Even more, a more recent meta-analysis of 20 case-control and 25 cohort/nested case-control studies, underlined the independent contribution of the welding fumes to the risk of lung cancer, as they estimated a relative risk of 1.17 (1.04 to 1.38; I^2=41.2%) after adjustment for smoking and asbestos exposure [9].

Conclusions

For this study case several conclusions should be taken into consideration: first, that more than one occupational disease has to be considered if multiple hazards are present at work. Second, that the latency for each of these diseases might be different and so might be the time to diagnosis. Third that from the same hazard different diseases might occur. Therefore, any worker exposed to occupational hazards has to be carefully monitored during and after the exposure cessation. Even if they have already been recognized with an occupational disease or have retired, the risk might persist and they should be reassessed at intervals appropriate to the type of exposure.

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