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Colinet–Caplan Syndrome: History of an Outbreak of Autoimmune Disease in Scouring Powder Workers

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The first modern description linking rheumatoid arthritis to occupational dust exposure is generally attributed to the British physician Anthony Caplan. In 1953, Caplan reported on a "peculiar" nodular pattern on chest radiographs of Welsh coal miners with rheumatoid arthritis that differed from the typical coal workers' pneumoconiosis. However, as early as 1950, the Belgian rheumatologist Émile Colinet described a similar case of rheumatoid arthritis and concomitant pulmonary opacities in a 30-year-old woman with silica exposure. Soon after, he published a second case. Although this condition initially was called Colinet–Caplan syndrome in the Francophone biomedical literature, Colinet's name was later dropped from the eponym. Because Colinet never clearly described the specific occupational context of his cases, Caplan syndrome has been misconstrued as uniquely a disease of coal miners.

We attempted to reconstruct the working conditions of Colinet's patients and found that they were packing Vim, a silica-based scouring powder, at the Savonneries Lever Frères factory in Brussels, Belgium. Colinet's cases were only the first 2 in a series of reports of rheumatoid arthritis and other autoimmune diseases, mainly among young women, in those who worked in the production of silica-based scouring powder between the 1930s and 1980s across Europe. The largest outbreak involved 32 cases of autoimmune disease among 50 former workers of a Spanish scouring powder manufacturing facility. After silica in scouring powders was replaced with less hazardous materials later in the 20th century, no further cases have been reported.

Although scouring powder disease is a historical phenomenon, autoimmune disorders linked to occupational exposure to silica and coal dust have not disappeared but instead are reemerging among those who work with silica-based artificial stone and in other dusty trades.

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Exposure to respirable crystalline silica has been linked to a range of autoimmune disorders with or without lung involvement. The strongest available evidence associates silica with systemic sclerosis (1), rheumatoid arthritis, systemic lupus erythematosus, and antineutrophil cytoplasmic antibody–positive vasculitis (2, 3).

Recognizing that occupational exposure might be related to autoimmune disorders is not new. In his 1775 textbook *Précis d'opérations de chirurgie* (*Essentials of Surgical Procedures*) (4), the French surgeon Louis Le Blanc wrote the following:

> . . . the sandstone dust penetrates . . . the body of the workers who work daily in these workshops filled with dust. They are struck by a cruel disease called 'sandstone disease' [*maladie du grès*] or 'Saint Roch's disease' [*maladie de Saint-Roch*] which appears to be caused by the presence of the sandstone particles. They are most often affected by fatigue and spontaneous or rheumatic pains in all their limbs, and particularly in their joints.

Of note, Saint Roch is the patron saint of stone quarries.

In 1914, the Scottish physician Byrom Bramwell reported that 5 of 9 patients he had cared for with systemic sclerosis (which he referred to as "diffuse scleroderma") were stonemasons. He did not suspect that silica dust caused the disease but instead attributed it to "holding cold chisels" (5). In 1933, Collis and Yule, who were pioneers in epidemiologic research on dust-related diseases, observed a 4-fold increase in the mortality rate of "chronic rheumatic diseases" among workers exposed to silica (6, 7).

Beyond the epidemiologic inferences made by Collis and Yule, the first clinical description of an association between rheumatoid arthritis and occupational dust exposure in the modern era generally has been attributed to the British physician Anthony Caplan. In 1953, Caplan reported on a "peculiar" nodular pattern on chest radiographs among Welsh coal miners with concomitant rheumatoid arthritis that he concluded differed from the typical coal workers' pneumoconiosis (8). However, in 1950, the Belgian rheumatologist Émile Colinet had published an early report of concomitant rheumatoid arthritis and pulmonary opacities in a 30-year-old woman working "in a factory where large quantities of silica flour were handled" without any coal dust exposure (9). The term *silica flour* refers to finely ground crystalline silica that includes particles in respirable size range (10). Because Colinet's report provided relatively limited descriptions of the factory's working conditions, the nature and extent of the occupational exposure has never been well understood.

In this article, we aim to describe the nearly synchronous, independent descriptions by Caplan and Colinet of the association between rheumatoid arthritis with lung abnormalities and occupational exposure to inorganic dust. Although Caplan's observations among coal miners continue to have currency, Colinet's documentation of the initial case reports of scouring powder workers has fallen into relative obscurity despite the later emergence of a range of autoimmune diseases among silica-exposed workers generally. We intend to explore and reconstruct the poorly recognized early history of the epidemic among scouring powder workers. These workers, mainly young women, developed autoimmune diseases and other conditions caused by silica after relatively short periods of heavy exposure to high airborne concentrations of finely milled

crystalline silica. We also intend to contextualize how the scouring powder disease outbreak has contributed to our general understanding of the risk for autoimmune diseases arising from dusty occupations.

METHODS

We performed a standard biomedical literature search using PubMed with the keywords *Caplan, pneumoconiosis, silicosis,* or *silica* and *rheumatoid, scleroderma, systemic sclerosis, lupus, Sjögren, Raynaud, connective tissue,* or *autoimmune*. We selected for further review publications that included cases with scouring powder exposure. Beyond English-language publications, we considered eligible citations published in French, German, Italian, Spanish, and Dutch. The reference citations of pertinent publications were also reviewed. In addition, the Royal Library of Belgium (Brussels), the archives of the KU Leuven University Library and the Belgian labor inspection (Federal Public Service Employment, Labour and Social Dialogue; Brussels), and Unilever's corporate archives (Port Sunlight, United Kingdom) were consulted. Other sources included the records of the 1930 International Labour Office conference on silicosis, such Belgian government publications as *Arbeidsblad/Revue du Travail*, newspapers, and publicly available material from court cases. Although Unilever's archives did not hold past medical records of workers, this source did provide documentation of the work process of scouring powder manufacturing and its exposure potential.

RESULTS AND DISCUSSION

Caplan and the Peculiar Chest Radiographs of Welsh Coal Miners

As noted, the first modern description of an association between rheumatoid arthritis and dust exposure is generally attributed to Anthony Caplan, a medical officer and member of the U.K. governmental Cardiff Pneumoconiosis Medical Panel. This association was initially acknowledged briefly in a 1952 report of a meeting of the Heberden Society but only fully reported in a 1953 publication in *Thorax* (8, 11). That article, authored solely by Caplan, reported 51 cases with concomitant rheumatoid arthritis and pneumoconiosis among 14 000 coal miners who had applied for pneumoconiosis disability benefits between June 1950 and April 1952 (8). In 13 of these 51 cases, he identified a "peculiar" pattern on the chest radiographs–that is, multiple well-defined, round opacities 0.5 to 5 cm in diameter distributed throughout both lung fields but particularly at the periphery.

Caplan called this pattern "rheumatoid opacities"; it did not resemble the typical coal workers' pneumoconiosis (**Appendix Figure 1**, available at Annals.org) (8). Caplan suggested that these radiographic changes (that is, the rheumatoid opacities) might represent "yet another manifestation of tuberculosis modified by dust with an added factor related to the rheumatoid arthritis" but noted that little evidence supported this speculation (8). In a subsequent study led by Jethro Gough–who pioneered the Gough–Wentworth large lung section technique–Caplan's colleagues provided detailed pathologic descriptions of these "rheumatoid" pneumoconiotic nodules (**Appendix Figure 2**, available at Annals.org) (12).

Caplan's work inarguably was pivotal in drawing attention to the potential relationship between rheumatoid arthritis and pneumoconiosis. Although the term "Caplan syndrome" initially referred to the co-occurrence of the typical large opacities on chest radiography and rheumatoid arthritis in coal miners, Caplan later reported a similar case in a silica-exposed sandblaster without coal mining exposure (13). Important to the history of this disease, Caplan subsequently reversed his statement that the syndrome of dust-associated rheumatoid arthritis occurred outside of coal mining and especially in relation to silicosis, stating, "We have been unable to find any … evidence that the prevalence of rheumatoid arthritis is increased in this disease [silicosis]" (14).

Colinet–Caplan syndrome

In July 1950, Émile Colinet, a Belgian rheumatologist at the Saint-Pierre Hospital in Brussels, published a case report describing a 30-year-old woman with a 10-year history of diffuse rheumatic arthritis. Her symptoms had started 2 years after she began work "in a factory where large quantities of silica flour were handled" (9). Colinet did not include an image of the patient's chest radiograph in this initial report but described it as showing "silico-tuberculosis," a term that predates Colinet that was commonly used to describe tuberculosis complicating silicosis. He noted that several of his patient's female coworkers had died of "silico-tuberculosis." In this report, Colinet hypothesized that there could be an association between silicotuberculosis and rheumatoid arthritis (9).

In March 1953, Colinet published a second case with a similar exposure history, namely, a 34-year-old woman with clinical manifestations of both rheumatoid arthritis and systemic sclerosis. She began working at 15 years of age in the same factory as the first patient (15). Of note, Colinet mentioned in this second report that he had learned about the (as-yet unpublished) findings of Caplan's research conducted between 1950 and 1952. Because neither Colinet nor Caplan had been able to detect mycobacteria in the sputum of their patients, Colinet adjusted his initial hypothesis and posited that silicosis could be responsible for inducing the rheumatoid arthritis, "even without tuberculosis" (15).

Reconstructing the Working Conditions of Colinet's Cases

Colinet's publications in 1950 and 1953 did not specify what exactly was produced at the factory where the patients in the cases he reported had worked or what the final product being made was (15). Some publications on Caplan syndrome cite Colinet's initial reports; however, the precise nature of his patients' employment is not surprisingly never explicit. Nonetheless, there is one important but rarely cited outlier to this pattern: In December 1953, Dr. Joseph Clerens–a colleague of Colinet who had been trained in pulmonology–recapitulated Colinet's 2 case histories, referring to their condition as "Colinet-Caplan syndrome." Clerens' report appeared

Figure 1. Savonneries Lever Frères factory in Forest, Brussels, Vim packing department (circa 1936).

The silica flour was mixed with powdered soda ash, soap, and sometimes other substances with a shovel or in a mixing machine. Cartons were filled by hand or mechanical fillers; the lids were then placed and fixed by a machine (17). Reproduced with permission from Unilever plc and group companies.

in the French–Flemish–language publication *Archives Belges de Médecine Sociale, Hygiène, Médecine du Travail et Médecine Légale/Belgisch Archief van Sociale Geneeskunde, Hygiëne, Arbeidsgeneeskunde en Gerechtelijke Geneeskunde* (16).

In his report, Clerens provided key information on the patients' jobs and described their workplace, documenting that they had worked at "*une usine de produits d'entretien renfermant outre le savon, une quantité importante de silice très finement broyée*" ("a factory producing cleaning and laundry products that included not only soap, but also finely milled silica") (**Figure** 1). The first patient started working in the factory at 18 years of age in 1938 and developed her first symptoms 2 years later. She worked at the end of the packing line and had to retrieve boxes with powder that had broken apart. She subsequently had to pour the salvaged powder into a large container. The second patient had worked at the factory for 11 years from 1935 to 1946. Her first symptoms appeared 4 years after she left this job. She had worked at the same production line as the first patient but according to Clerens was "less exposed"; however, he did not elaborate on this in regard to precise job tasks.

Moreover, Clerens published the radiographic image of Colinet's first patient, which Colinet had initially described as showing silicotuberculosis. In fact, this radiograph seemed to show a typical "Caplan" pattern–that is, multiple well-defined, round opacities, particularly at the periphery (**Appendix Figure 3**, available at Annals.org). Of further interest, Clerens initially believed that the association between the patients' jobs and their clinical syndrome was merely coincidental and decided not to support compensation for the patients through the Fonds de Prévoyance (the precursor of the later Belgian Fund for Occupational Diseases). However, he stated that his view later changed because he learned of the work of Caplan that had been presented (by Fletcher) at the Heberden Society in London in October 1952 (11).

Production of Vim at the Savonneries Lever Frères in Forest, Brussels

Neither Colinet nor Clerens ever identified by name the cleaning product manufacturer in question; however, it was most likely the Vim scouring powder factory Savonneries Lever Frères in Forest, Brussels, located less than 5 km from the Saint-Pierre Hospital where these clinicians were active. To further support this theory, a report from the hospital's internal medicine department from January 1953 explicitly described a 41-year-old woman with fatal "acute" silicosis as a Vim scouring powder worker (18). The authors of this report found that the Vim powder looked "the same" under polarized light as the crystals present in the alveoli in their patient's biopsy specimen. Moreover, as early as 1935, the Belgian labor inspection had reported the case of a 28-year-old worker with silicosis who had been unloading, mixing, and cutting the raw materials to make Vim powder for 5 years (19).

In 1889, Lever Brothers–founded by William Hesketh Lever and James Darcy Lever–started producing Sunlight soap at the newly constructed Port Sunlight factory in Wirral near Liverpool, United Kingdom. By the beginning of the 20th century, the company dominated the English

market with this soap. In 1904, Lever Brothers also started producing Vim scouring powder in Port Sunlight. Vim consisted of soap, soda, and finely milled silica that came from quarries in North Wales. It was marketed as "the housewife's handy helper" for cleaning and polishing: quick and efficient, purportedly without leaving scratches on glass, metals, or ceramics.

In 1905, the Société Anonyme Savonneries Lever Frères, a subsidiary of Lever Brothers Limited, opened a factory in Forest, a suburb of Brussels. There, Sunlight soap initially was produced from palm oil from the Belgian Congo. More types of household cleaning and laundry products gradually were introduced. The production of Vim scouring powder in Brussels had started in 1929 (only 6 years before the first report of silicosis noted earlier) (19). Unilever was formed in 1930 after the merger of Margarine Union and Lever Brothers Limited.

Scouring Powder and Autoimmune Diseases

In the years after the cases described by Colinet, publications appeared on a range of autoimmune diseases in workers producing silica-containing scouring powder, as well as in household consumers of such products. In 1968, Titscher (Vienna, Austria) (20) described a 63-year-old woman with Caplan syndrome who had worked as a packer of Silax scouring powder between 1941 and 1961. Koeger and colleagues (Paris, France) (21) described 2 cases of silica-associated autoimmune disease in patients who had been exposed to Ajax scouring powder (Colgate–Palmolive). One case was a 43-year-old man with systemic lupus erythematosus who had worked between 1970 and 1989 in an Ajax manufacturing plant. The second was a 37-year-old woman with Sjögren syndrome, Raynaud phenomenon, and inflammatory polyarthritis who had been using Ajax powder 6 hours per day for 12 years (1968 to 1980) to scour ceramic plumbing fixtures (21). Of note, the exposure of the latter patient was revealed only when "Caplan" nodules (containing silica-laden macrophages) discovered on a lung biopsy led the treating clinicians to obtain a thorough occupational history (21). Mehlhorn and associates (22) described a professional cleaner in Germany with systemic sclerosis who had been using 1 to 2 packages (250 to 500 g) of ATA scouring powder daily for 14 years.

The largest reported outbreak of autoimmune disease occurred in a Spanish scouring powder manufacturing facility (23). When clinicians at the University Hospital of Seville realized in the early 1990s that 4 of their patients with prominent autoimmune disease–related symptoms had worked at the same factory, they encouraged the patients to contact their former colleagues. By then, the production of the scouring powder had already ceased 10 years earlier. Fifty former workers (44 women and 6 men) were recruited out of a workforce of approximately 300, and 32 had a definite autoimmune disorder. Among these, 5 had systemic sclerosis, 3 had systemic lupus erythematosus, 5 had combined systemic sclerosis and lupus, 6 had Sjögren syndrome, and 19 had undifferentiated systemic autoimmune disease (23). They had worked at the factory an average of 6.1 years. Of the 50 workers, 18 had silicosis (of whom 14 had a concomitant autoimmune disorder). Through many court cases, we know that these workers were employed at the Persan factory in the outskirts of Seville producing San scouring powder in very dusty working conditions; one worker stated, "I left working with my clothes and body 'literally' white" (24).

In addition, case reports have been published of patients who had intentionally inhaled silica-based scouring powder (25–27). For example, Dumontet and coworkers (27) described a patient who developed acute silicosis after intentionally inhaling Ajax scouring powder over a 6-month period 3 times daily because it "had a nice smell." Five years later, this patient had developed an autoimmune mixed connective tissue disease (Sharp syndrome) (27).

Of note, before the outbreak of autoimmune disease, the production of silica-based scouring powder was one of the first industries that had been struck by cases of "acute" silicosis. In contrast to classic (chronic) silicosis, acute silicosis occurred in workers after relatively brief exposures to very high levels of silica, resulting in rapidly progressive disease that most often was fatal. The history of acute silicosis in scouring powder workers has been described previously in detail (28). The first reports on acute silicosis in the scouring powder industry started to appear between 1928 and 1930 in the United Kingdom (29–32) and then in the rest of Europe (33–35) and the United States (36, 37), many after persons worked for merely 2 to 5 years in the factories in question.

Conclusion

Throughout the 20th century, a largely invisible and forgotten outbreak of autoimmune diseases affected scouring powder workers, especially young women packing the powder. Published cases probably represent merely the tip of the iceberg. Not only has the extent and interrelatedness of these cases gone underappreciated, but the very eponym linking silica to autoimmune disease, Colinet syndrome, has been erased. The construct that Caplan self-promoted as being specific to coal miners and that dismissed the role of silica in causing rheumatoid arthritis in other settings has led to confusion and underappreciation of this important silica hazard (38, 39).

It is not possible to conclusively determine causality solely on the basis of early case reports of associations among silica exposure, autoimmune disease, and lung abnormalities. Nonetheless, the presence of multiple consistent reports of autoimmune disorders in groups of workers with similar exposures strongly supports a causal relationship. Moreover, the presence of exposure even without lung findings also has been strongly implicated in autoimmune disease caused by silica. Indeed, the accumulating body of epidemiologic evidence of an association between exposure to respirable crystalline silica and a range of autoimmune disorders (without or with frank lung disease) reinforces Colinet's original observations (1–3, 40).

Recent reviews and reports on Caplan syndrome and rheumatoid arthritis have focused almost exclusively on such male-dominated occupations as mining and foundry work, whereas female scouring powder workers thus far have largely been ignored (38, 39, 41). The eponymous Caplan syndrome has been considered a disease uniquely relevant to coal miners, a misguided conclusion. In this article, we show that Colinet's near synchronously reported cases attributable to pure silica exposure without any component of coal dust provide the context for a better grasp of the epidemiology of occupational autoimmune disease. The modified term Colinet–Caplan syndrome has occasionally been applied (especially outside of Great Britain and North America) (16, 42, 43). However, far more important than eponyms is a broadened appreciation of the potential links between a range of occupations (one of which was female-dominated) and autoimmune disease. To achieve a better understanding of silica-associated, occupationally related autoimmune disease, it is critical to take more fully into account the emerging medical recognition of this phenomenon—including Colinet's largely overlooked role.

It was not until the 1980s that scouring powder producers stopped using silica and replaced it with less hazardous materials, such as calcium carbonate. Why this protective substitution finally occurred is unclear. There is no record of scouring powder industry warnings about the potential hazards of silica-based powders to their own workers, let alone to consumers. Analogous histories of hazardous compounds in consumer products, such as vinyl chloride in hairspray, have shown that industries may prefer a "silent" substitution to avoid consumer lawsuits (44). No further cases of autoimmune disorders related to scouring powder exposure that took place after 1989 have been reported, although publications continued to appear in the aftermath of previous exposure (21, 27).

The cautionary tale of silica disease and related autoimmune complications among scouring powder manufacturing workers serves us well in more fully appreciating new outbreaks of silica-caused disease that have emerged in the 21st century. Chief among these is the epidemic of severe illnesses among synthetic stone countertop fabricators, also linked to autoimmune disease (45–47). Further, exposure to respirable crystalline silica seems to be causal in the unexpected surge of severe pneumoconiosis in contemporary miners (48). From these cases, we have learned—or, better put, relearned—that such exposures are associated with a wide range of autoimmune conditions as well as alveolar proteinosis, the pathologic correlate of what often had been referred to as "acute silicosis" (46, 47).

The continual introduction of novel silica-based applications and the reappearance of high-exposure conditions that were believed to have been well controlled warrant persistent vigilance to prevent epidemics of disease in workers (48). Clinicians have a major role to play in the recognition of the adverse health effects of occupational dust exposure. However, fulfilling that role requires attention to the occupational histories of patients, including those with such autoimmune disorders as rheumatoid arthritis and systemic sclerosis. Cross-disciplinary collaboration between generalists and occupational health specialists can be pivotal in that regard.

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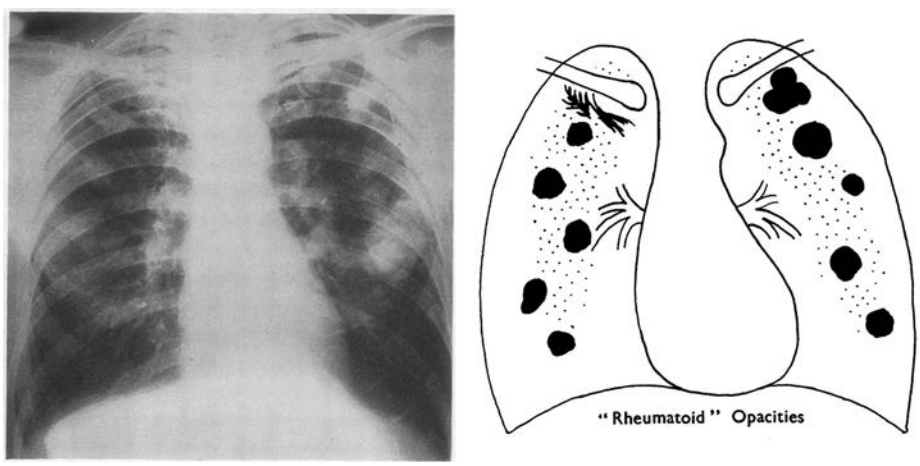
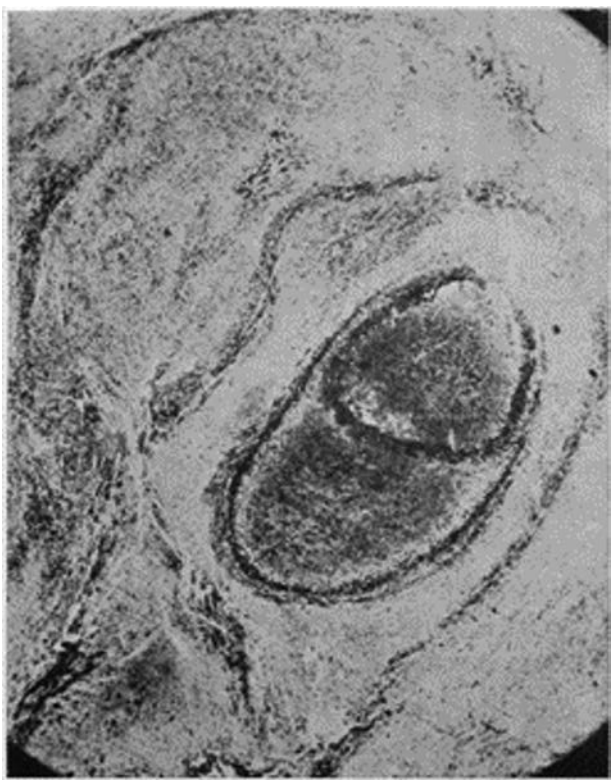
Final approval of the article: P.D. Blanc, S. Ronsmans.
Collection and assembly of data: S. Ronsmans, P.D. Blanc.

Appendix Figure 1.



Left. Chest radiograph from Caplan's 1953 article (8) showing the characteristic "rheumatoid" opacities. The shape, peripheral distribution, and multiplicity of the opacities combined with the slight degree of simple pneumoconiosis distinguish this type of lesion from the ordinary progressive massive fibrosis. ***Right.*** Illustration of the "rheumatoid" opacities (8). Reproduced from Caplan A. Certain unusual radiological appearances in the chest of coal-miners suffering from rheumatoid arthritis. Thorax. 1953;8:29-37. [PMID: 13038735] with permission from BMJ Publishing Group Ltd.

Appendix Figure 2. "Rheumatoid" pneumoconiotic nodule with lines of stranded dust from autopsy lung tissue from a coal miner with rheumatoid arthritis (12).



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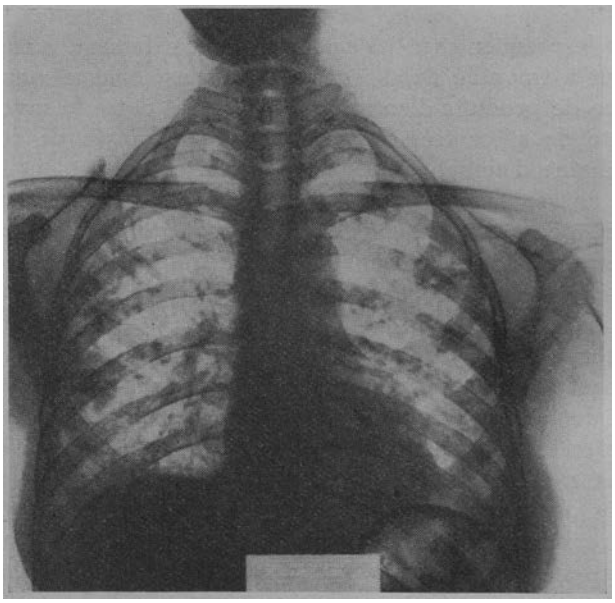
Appendix Figure 3. Chest radiograph (positive image) of the first case described by Colinet.



Colinet did not publish the radiograph in his 1950 case report; in 1953, his colleague Clerens recapitulated Colinet's cases in a subsequent publication. Clerens, who had been trained in pulmonology, noted, "*L'exploration radiographique des poumons mérite une mention spéciale: le cliché montre en effet une infiltration macronodulaire bilatérale, périphérique répondant à la description signalée comme caractéristique de l'association silicose et rhumatisme par le Dr Caplan.*" ("The radiographic exploration of the lungs deserves special mention: the radiography indeed shows a bilateral, peripheral macronodular infiltration responding to the description reported as characteristic of the association of silicosis and rheumatism by Dr. Caplan.") (16). Reproduced from Clerens J. [Pulmonary silicosis and rheumatism or Colinet-Caplan syndrome]. Arch Belg Med Soc. 1953;11:336-42. [PMID: 13139664]. The article is licensed under CC BY 4.0.