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A CASE OF ALUMINIUM DUST LUNG

A Necropsy Report

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Much has been reported on the importance of silicosis as a form of pneumoconiosis, yet, the report on the condition of aluminium dust lung another entity in the consideration of pneumoconiosis has been attracting attention which is of relatively recent trend. The papers on the subject of aluminium dust lung was first reported by Baader followed by Doese,[2] Goralewski,[3] Kahlau [8] and the others[10,11]. An original necropsy report on this particular disease considered to be most raliable one was by Goralewski and Jäger [4] and later numbers of reports have appeared in Germany [9,10,16] and also in Anglo-American Countries.[14,15,18] These reports, however, were inclusive of not only of the findings of aluminium dust origin but also a pneumoconiosis developed as the result of inhalation of bauxite fume containing some silicate known as bauxite fume pneumoconiosis[15,18]. The report of true aluminium dust lung attributed to aluminium dust basing on the study of necropsy cases was heretofore unknown in this country. The present case which we have encountered recently is a typical aluminium pneumoconiosis which seemed to have occurred mainly due to pure aluminium metallic dust.

A Case Report

A male patient, Mr. [redacted] Age 32

Clinical History

The patient was a worker in a factory where metallic powder for the decorative purpose was manufactured and he worked for about three years and half when he began to complain respiratory distress with persistent cough and difficult breathing. With a diagnosis of pneumoconiosis, the patient was hospitalized for the treatment. The patient got worse gradually because of advanced pulmonary change and died 3 years and 3 months after the onset of disease on May 31, 1955.

Pathological and Anatomical Findings

A. Pathological and anatomical diagnosis.

1. Aluminium dust lung.

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2. Hypertrophy of the right heart.
3. Congestion of the lungs and the spleen.
4. Old pleural fibrotic adhesion.
5. Fatty degeneration of the liver.

B. Macroscopic and microscopic findings.

Lungs and pleura: Pleura pulmonalis showed marked thickening throughout the pleural surface on both sides and densely adherent to the parietal pleura particularly noticeable in the interlobular area of the middle and upper lungs of which the thickness was 0.3 cm. The pulmonary parenchyma was hardened to a fair degree throughout the lungs and the cut surface revealed a marked change of reddish brown color and presented the minute granules of uneven and irregular appearance. In the right apex of lung there were three cavities each of which had a finger tip size and also several cavities as large as a thumb's head were found directly under the preural surface in the middle of right upper lobe (Fig. 1). There were numerous smaller cavities of a grain to small bean size distributed over lobes of the right and left lungs (Fig. 2). These cavities were empty and had made up of smooth membranous wall composed of minute connective tissue. A few small bean to a soy bean sized hilus glands were found at the both hilus presenting dark gray color and black medullary appearence when sectioned.

Histologically, common findings were far advanced proliferative changes of collagen fibers of the lung parenchyma by which alveolar space was pressed (Fig. 3, 4), having invaded with numerous mononuclear cells or giant cells or replaced alveolar exudate by fibroblastic cells and also with partial atelectatic induration. In some parts there were mononuclear and giant cells in which fatty granules were included, and there were at times the substances which appeared like opaque columnar crystals (Fig. 4). The remaining alveolar walls were in general infiltrated with small round cells and with marked hyperplasia. Small blood vessels and capillaries found in the proliferative connective tissue have presented stenotic appearence due probably to the pressure. Some vascular walls have become so hyperplastic to a degree that their caliber have been practically obstructed. There were circumscribed areas where infiltration of small round cells around these vessels and capillaries found in the proliferative connective tissues. The lung parenchyma of which the alveolar structure remained normal presented a picture of marked congestion.

The interstitial tissue showed either granules or lumps of dark to brownish pigments and most of them have been phagocytized by histiocytes and partly deposited in the intracellular spaces. These changes were also seen with the lymph glands of the hilus. Black pigments found in the tissue resembled more like carbon deposit but there were some pigment granules containing aluminium besides hemosiderin or carbon pigment (Fig. 4).

Heart: The heart was definitely enlarged grossly, it was twice the size of the fist of the dead. The anterior middle wall of the left ventricle had a thickness of 1.3 cm., while the right ventricular wall was hypertrophied to 0.5 cm. and with dilated

cavity. Hypertrophy of the right ventricle was most noticeable in general, and histologically, the hypertrophy of the myocardial fibers have been observed. This case seemed to present a picture of the chronic cor pulmonale considering congestive changes of the lungs, spleen and kidneys.

Liver : Although there were no marked changes noted grossly but histological characteristics seen were dark brownish granules or needles like to columnar pigments being phagocytized in moderately swollen stellate cells throughout the tissue, and also pigment deposits could be seen in the hepatic cells (Fig. 6). Hepatic cells in the periphery of liver lobules have undergone moderate degree of fatty degeneration and some of these cells were possessed of double nuclei. But none in the other areas.

Kidneys : Smaller blood vessels and capillaries of the interstitial cells and glomeruli were engorged to some extent but no other changes of note found.

Spleen : Splenic sinuses were dilated somewhat and filled with red blood cells but the boundary of the medullary cords were indistinct. Medullary cord showed certain degree of cellular proliferation and filled fair numbers of red blood cells. Medullary cells presented dark brownish granular pigments and lymphoid follicles appeared atrophic. The capsule had undergone some thickening.

Thyroid gland : A part of follicles dilated were filled with colloid and follicular epithelium have become squamous but no marked changes recognized.

Adrenals : There were congestion limited to the cortex.

Testicles : Basal membrane of the seminiferous tubules were hyperplastic and spermatozoon formation appeared to be reduced. All other organs examined showed no notable changes.

Aorta : Only change noted was moderate connective tissue hyperplasia of the intima but partial.

C. Histochemical Tests.

1. Test for aluminium
   Lungs, liver and kidneys were examined by Okamoto's method [13] but no positive granules could be seen.
2. Bleaching test with chlorine gas
   The tissue sections of lungs, liver and spleen were bleached by chlorine gas[13]. Bleaching was demonstrated in dark pigments of the stellate cells of the liver, and pulpa cells of spleen, and in a part of dark brownish granules of the interstitial pulmonary tissue.
3. Test for inorganic and organic irons
   For inorganic iron Perls-Stieda's method and for organic iron Okamoto's method [13] were used respectively of the tissues of the lungs, liver, and spleen. Dark pigments showing peculiar bleaching quality were found to be negative for iron.
4. Test for copper
   With Okamoto and Utamura's test of p-dimethylamino-benzyliden rhodanin for copper in tissues of the lungs, liver and kidneys were found to be negative.

Results of Analyses of Tissues

A. Spectroscopic analysis of metals: The present case and a control of male, age 19, who died of coronary disease were examined. One gram each of lungs and livers of these two cases were removed and put in quartz pots and heated at 500°C and the tissue ash obtained used for test. Shimazu's quartz Spectrograph QF-60 was used and the photographs taken of the emission spectra of the examined material with carbon arc electrodes. The elements found quantitatively based on line density of spectra lines were as shown in Table 1.

TABLE 1 Result of Spectroscopic Analysis of Metals in Various Orgsans

	Present case		Control case	
	Lung	Liver	Lung	Liver
Na	++	+++	++	++
K	++	+++	++	++
Ca	+++	+++	++	++
Mg	+++	+++	±	+
Al	++	++	±	±
Fe	++	++	±	+
Si	++	+	−	−
Cu	+	+	+	+
Zn	+	+	+	+
P	+	+	+	+

B. Quantitative determination of metals: Because we have found sufficient amount of metals, i. e., aluminium, iron, magnesium, and silicon, among them analyses were made of particularly with aluminium and iron. Quantitative test for copper was also tried in order to ascertain the character of dark granules found in the liver by the histochemical method.

a. Determination of aluminium in organs: For control cases, specimens of the lung, liver, and spleen (formaline fixation) of three cases of male, age 28, died of hemorrhage, a male, age 20, died of cerebral injury and a male, age 28, died of hemorrhage. These have been analysed by Winter, Thrun and Bird's method [17] after having pulverized by heating 500°C. As shown in the Table 2, in lungs the quantity of aluminium was increased up to 4.5 times that of control's. In the liver it was increased 2.1 times but in kidneys and spleen, it was only 1.1 to 1.2 times.

TABLE 2 Aluminium in Organs (γ/10g)

	Present case	Control case				Present case / Control
	Age 32, male	Age 28, male	Age 20, male	Age 28, male	Average	
Lungs	228	45	l. 52 r. 48	l. 57.6 r. 53.6	51.24	4.5
Liver	170	72	81.6	89.6	81.07	2.1
Kidneys	50	45	l. 40 r. 43	45	45	1.1
Spleen	56	41	47.2	47.2	45.93	1.2

b. Determination of iron in organs: One control case was a man, age 28, died of hemorrhage. The specimens of the lung, kidney and spleen of the control case as well as the case in question were made ash at 500°C heat and iron estimated by means of Hummel and Willard's[5]. It was found that iron content in the lungs, kidneys and spleen of the present case were reduced rather than increase from the control, however, in the liver, the increase was

TABLE 3 Iron in Organs (mg %)

	Present case	Control case	Present case / Control case
	Age 32, male	Age 32, male	
Lungs	l. 11.0 r. 11.1	20.7	0.53-0.54
Liver	6.6	4.0	1.7
Kidneys	4.1	4.0	1.0
Spleen	4.6	7.2	0.64

only up to 1.5 times though this did not have a direct bearing on the case (Table 3).

c. Determination of copper in organs: For control case, a male, age 28, died of hemorrhage was used. The specimens of liver, kidneys and spleen were taken and applied 500°C and ashes were examined quantitatively by Numata and Matsukawa's method[12]. The results were variable, some showed increase while others have decreased attaching little pathological significance. Liver alone in this case showed reduction of which the reason was unknown (Table 4).

TABLE 4 Copper in Organs (γ/10 g)

	Present case	Contral case	Present case
	Age 32, male	Age 28, male	Control case
Lungs	l. 26 r. 51	l. 71 r. 35	0.4 1.5
Liver	14	104	0.13
Kidneys	28	27	1.0
Spleen	–	33	

Results of Examination of Inhaled Metallic Powder

Examination of inhaled metallic powder manufactured in the factory where the patient had worked revealed that this aluminium powder contained of 96% aluminium dust which was found to be 99.3% metallic aluminium and as an impurity stearic acid 4% which contained traces of silicon, iron and copper.

Discussion

It has been demonstrated that the pneumoconiosis of aluminium origin could cause advanced fibrosis without fail in the pulmonary parenchyma[1,9] and the present case is no exception, for we have found disseminating parenchymatous fibrosis of the same degree throughout the lung lobules unlike the finding which Kahlau had described that such a change would be limited to the upper lobe. So-called "Aluminium Körperchen", an entity which Kahlau[9] has confirmed was not demonstrated in our case. The other changes accompanied were the right ventricular hypertrophy with congested lungs, spleen and kidneys presenting a picture of chronic cor pulmonale due to pulmonary fibrosis.

According to Baader[1] aluminium powder inhaled by way of respiratory tract combines with sodium chloride of body fluid, converting aluminium ion which in turn produces coagulation of the tissue protein resulting cellular necrosis while aluminium firmly combines with protein and undergoes pathologic process and origin of disturbance depends entirely on inhalation of the metallic powder. Present investigation leads us to reach a similar conclusion from what we have examined of these organs chemically.

Considering further on the nature of dark brownish granules found in lungs, liver, and speen which could be bleached by chlorine gas, Jäger and Jäger[6] by using Goppelsröder's method has demonstrated a metallic element directly from the lung tissues but applying Okamoto's method which seems to be superior than morin method of Goppelsröder for this purpose, we had failed to detect this metal. This

fact signifies that it will not be demonstrable histochemically by the reason of the metal combined closely with protein as stated above. But following four facts lead us to believe that the granular substance must be aluminium origin ; 1. granules were insoluble even after treated with 10% hydrochloric acid for 24 hours, 2. fine granules of aluminium powder could be bleached just as these granules were, 3. these granules present negative test for either iron or copper, their sources are not hemosiderin or carbon powder which is never bleached, and 4. aluminium content is much higher in the lungs and liver which contain more granules than those of controls.

In considering the result of the analysis of inhaled metallic powder, it has been known that in Canada and United States much has been studied in the use of aluminium for prevention of pulmonary silicosis that toxity of aluminium powder for local tissue is far less than silicates, yet Kahlau[9], Goralewski and Jäger[4], and we have clarified that aluminium powder alone is capable of producing serious metallic intoxication which may invite a fatal outcome. Reports of Wyatt and Riddell[18] on bauxite fume pneumoconiosis have similarly brought out a significance of aluminium toxicosis. Therefore application of the method for inhaling aluminium powder seems to require caution.

As yet an aluminium dust lung found in necropsy case due to straight aluminium powder is hardly known in our country, and reported cases from other countries are also usually small, hence, this case may have contributed valuable information in elucidating etiological aspect of the aluminium dust lung.

Conclusion

Report of a case of disseminated chronic interstitial pneumonia (aluminium dust lung) which had been induced by pure aluminium powder and presented evidences that the case was of pneumoconiosis of aluminium origin. This, also, is a first report of the case in Japan of an typical aluminium dust lung. The case has threw a light on the nature of characteristic black granules herefore unknown thought to be contained of aluminium in the lungs, liver and spleen, and has a peculiar bleaching quality when attacked by chlorine gas unlike carbon powder or hemosiderin.

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Fig. 1. A cavity as large as a finger tip size found in the right apex of lung.

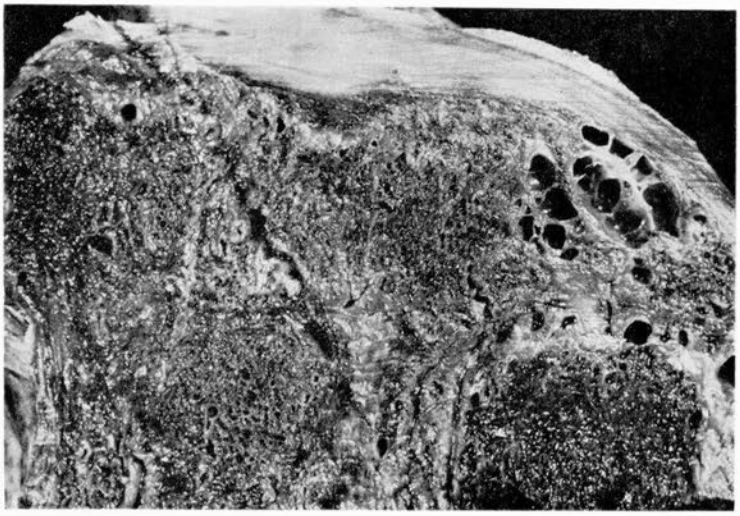

Fig. 2. Apex of left lung. Multiple small emphysematous bullae were found under the dense pleural scar.

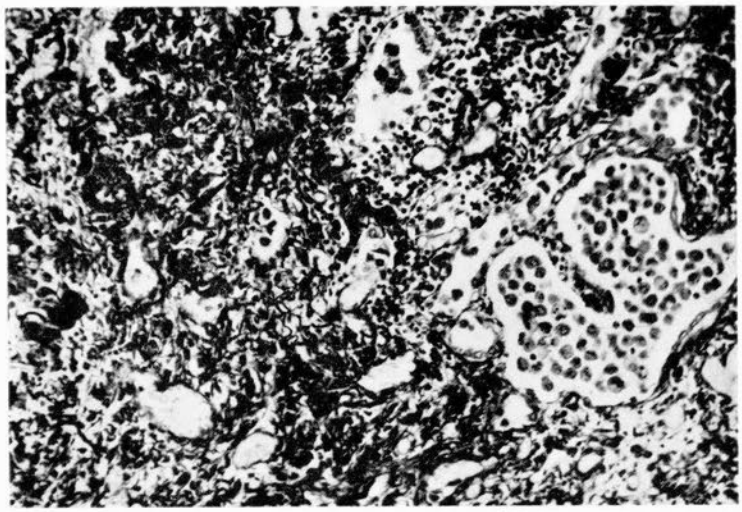

Fig. 3. Diffuse proliferation of connective fibers.

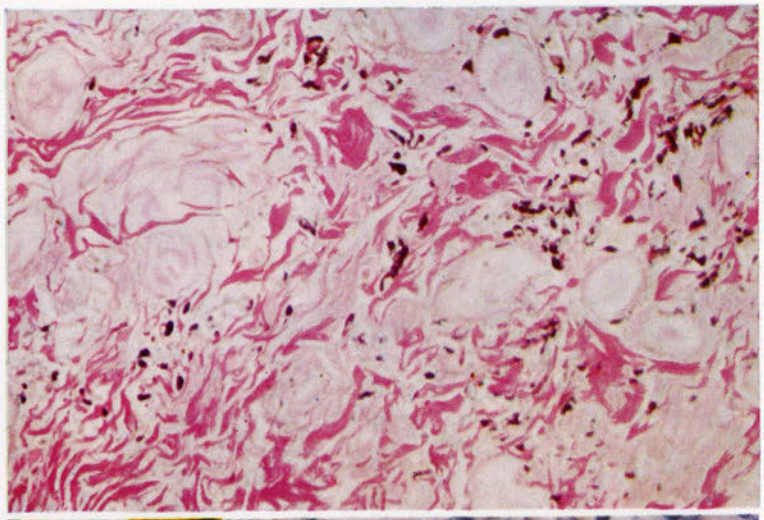

Fig. 4. Lung showing severe diffuse interstitial fibrosis and marked deposit of dark pigment which involved aluminium or carbon dust.

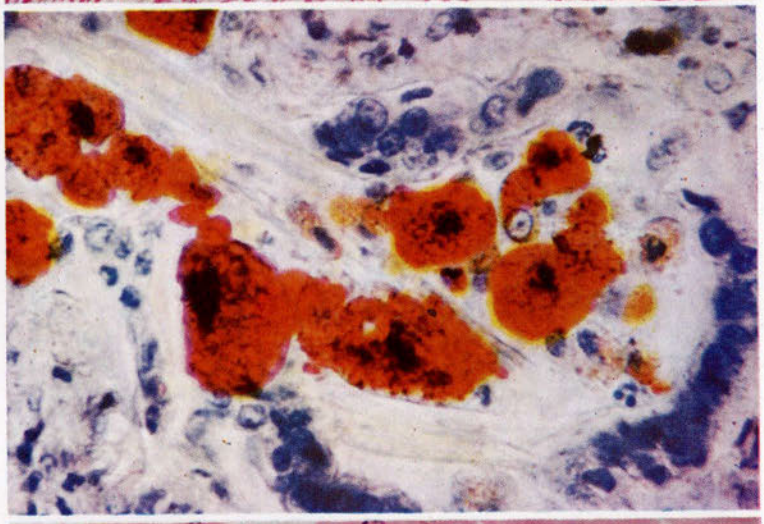

Fig. 5. In pressed alveolar spaces were found columnar crystals and epithelial cells with fatty degeneration.

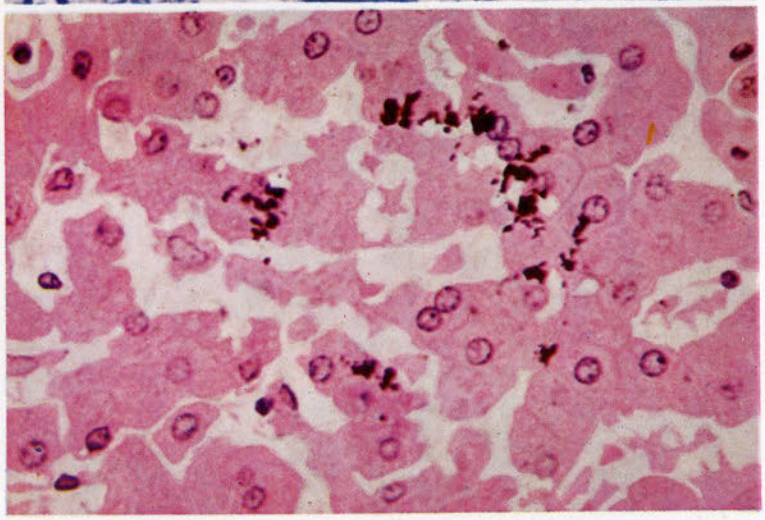

Fig. 6. Dark pigments deposited in hepatic cells and swollen stellate cells.