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Effects of Aluminium Chloride Exposure on the Histology of lungs of Wistar Rats

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ABSTRACT

Aluminium is presents in many manufactured foods and medicines and is also added to drinking water for purification purposes. The lungs consist essentially of; the spongy respiratory tissue in which gaseous exchange occurs between blood and air, and a branching system of air tubes called bronchioles and bronchi which “pipe” air into and from the pockets and passageways of the spongy respiratory tissue. The purpose of this study was to evaluate the possible effects that aluminium chloride exposure could have on the histology of lungs of wistar rats. Twenty wistar rats were used for this study. They were well fed with grower mash, provided with adequate water and kept under good ventilation. The wistar rats were divided into five groups as follows: Group I was the control, group II was given 475mg/Kg, group III received 950mg/kg, group IV received 1,425mg/kg and group V received 1,900mg/kg of aluminium chloride through oral intubation for period of eight weeks. The wistar rats were humanely sacrificed, the lungs removed, fixed, processed and stained with Haematoxylin and eosin. Photomicrographs of the lungs showed congested blood vessels in the aluminium treated groups. Based on our observations, we therefore conclude that aluminium chloride exposure was detrimental to the histology of lungs.

INTRODUCTION

There are right lung and left lung that reside in the chest cavity and surround the heart. A thin membrane called the pleura covers the outer surface of the lung. The air we breathe gets into the lung through an airway (path for air). The right lung has three separate sections (upper, middle, and lower lobes), while the left lung has just an upper and a lower lobe. Each lobe has its own bronchi and blood supply. Further along in the airway, within the lung, the bronchi continue to divide into ever-smaller (narrower) tubes, much like the branches of a tree. Hence, the term trachea-bronchial tree. The walls of the bronchi contain muscles that can cause the airway to expand (widen) or contract (narrow). Alveoli are lung air sacs made of simple squamous epithelial cells for diffusion of gases. Capillaries plus alveoli form the respiratory membrane for the exchange of gases between the blood and the lungs. The oxygen exchange in the lungs takes place across the membranes of small balloon-like structures called alveoli attached to the branches of the bronchial passages. These alveoli inflate and deflate with inhalation and exhalation (Shier *et al.,* 2007; Gary and Kevin, 1996). The lungs are contained in the thorax. The thorax has a cage-like framework composed of the vertebral column, the ribs, the costal cartilages and the sternum. The bottom of the cage is a dome-shaped musculotendinous sheet, the diaphragm. The two lungs fill two large compartments in the thoracic cavity. Each compartment is lined with a fibroelastic membrane, parietal pleura, which are provided with an internal layer of squamous mesothelial cells. Likewise each lung is covered with a similar membrane, the visceral pleura, the outermost layer of which consists of squamous mesothlial cells. A film of fluid is present between the parietal pleura that lines each cavity and the visceral pleura that covers each lung; this fluid has lubricating value and allows the visceral pleura covering the lungs-hence, the lungs themselves- to slide during respiratory movements along the parietal pleura that lines the cavities (Arthur, 1974). The lungs consist essentially of; (1) the spongy respiratory tissue in which gaseous exchange occurs between blood and air, and (2) a branching system of air tubes called bronchioles and bronchi which “pipe” air into and from the pockets and passageways of the spongy respiratory tissue. The main bronchus from each lung connects with trachea and this, in turn, by means of the larynx, nasopharynx and

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the nose (or the mouth if need be), connects with the outside air. Hence on respiratory act, air is drawn through the nose, down to the trachea, into the bronchial tree to its end branches and from there into the passageways and the pockets of the spongy, capillary-rich respiratory tissue, where, and only where, gaseous exchange occurs (alveoli duct and alveoli) (Arthur, 1974). Aluminium has the potential to be neurotoxic in human and animals. It presents in many manufactured foods and medicines and is also added to drinking water for purification purposes (Newairy *et al.*, 2009). Aluminium is widely used in antacid drugs, as well as, in food additives and tooth paste (Abbasali *et al.*, 2005). More than 100 toxic of aluminium have been identified and many are damaging to the human brain. The progressive deterioration in cognitive function associated with Alzheimer had been correlated with loss of cholinergic function and the degeneration of cholinergic neurons (Collerton, 1988; Francis *et al.*, 1985; Smith and Swash, 1978; Whitehouse *et al.*, 1985). Aluminium is generally a recognized neurotoxin that is believed to be at the root cause of Alzheimer Disease (AD). Aluminium becomes more toxic when combined with a high cholesterol diet. They work together by means that have yet to be fully determined to create the senile plagues and ultimately the mental deterioration known as AD. However, there are at least two recognized synergistic ways that these factors contribute to brain damage. First, an acid-forming diet-one high in meat, poultry, eggs, and cheese-leads to increased senile and brain concentrations of aluminium. Second, aluminium enhances inflammation. The immune enhancing properties of aluminium discovered after immunization with diphtheria and tetanus vaccines in studies performed in the 1940s and 1950s and aluminium is used today to enhance the effectiveness of the inflammatory response of most vaccines given to adults and children. In the brain, aluminium enhances the inflammation that may result from the formation of senile plagues driven by cholesterol build up initial injury-this metal is known toxic to the nervous system-that starts the disease processes, leading to brain cell death, senile plagues neurofibillary tangles. However, according to Dougall (2004), aluminium is present in our water, foods, medications and air. The healthy human body has effective barriers such as skin, lungs, and gastrointestinal tracts, against aluminium. Aluminium is not a nutrient-in other words and the body has no need for this metal and avoidance has no negative consequences. All foods naturally contain aluminium, but some, such as tea, are partly high in this metal. Fortunately, most of the aluminium in natural plant foods is bound with other substances such as Silicon which prevents absorption of the aluminium into the body. The harmful (unbound, more absorbable) forms of aluminium enter our foods additives such as leaving agent and emulsion.Over the Counter; Deodorants, vaginal douches, baby wipes, skin creams, suntan lotions, toothpaste, buffered aspirin, some hemorrhoid and diarrhea products. Medical; Vaccinations, allergy testing, intravenous solutions, allergens, wound and antacid, ulcer treatment, Foods; Aluminium cans, foils, containers, baking powder, cake mixes, frozen dough, pancake mixes, self-rising flour, grains, processed cheese. (Dougall,2004).

Role of aluminium intoxication in different organs in neurodegenerative diseases has been recently emphasized (Somova, *et al.*, 1997; Exley, 1999). Aluminium was said to have contributed to a variety of cognitive impairments in mice, rabbits, and rat pups (Muller *et al.*, 1990; Yoke, 1985, Bilkei-Gorzo, 1993; Mari, 2001). Studies on workers exposed to aluminium dust in industrial environments demonstrate similar effects (Rifat *et al.*, 1991; Bast-pettersen *et al.*, 1994; White *et al.*, 1992; Akila *et al.*, 1999). The purpose of this study was to evaluate the possible effects that aluminium chloride exposure could have on the histology of lungs of wistar rats.

MATERIALS AND METHODS

This experiment was conducted in the Department of Human Anatomy, Faculty of Medicine, Ahmadu Bello University, Samaru, Zaria, Nigeria. The rules and regulations governing animal handling were strictly observed.

Experimental Animals

Total of twenty wistar rats were used for this experiment. The wistar rats were kept for two weeks before commencement of aluminium chloride administration. This was in order to allow the wistar rats acclimatized to the environment. The wistar rats were given adequate feed (grower mash), water and kept under good ventilation.

Experimental Design

The wistar rats were divided into five groups. The group I was the control that received distil water only while the remaining four groups received different concentrations of aluminium chloride as follows:

Group II received 475mg/Kg,
Group III received 950mg/kg,
Group IV received 1,425mg/kg,

And group V received 1,900mg/kg via oral intubation for duration of eight weeks.

Tissue processing and staining:

At the end of eight weeks of oral administration of aluminium chloride to the wistar rats, except group I that received distil water only, they were humanely sacrificed by anesthetizing them in a suffocating chamber using chloroform. The thoracic regions of the wistar rats were dissected and the lungs were removed, and immediately fixed in formalin. After fixation, the lungs were transferred into an automatic processor where they went through a process of dehydration in ascending grades of alcohol (ethanol) 70%, 80%, 95% and absolute alcohol for 2 changes each. The tissues were then cleared in xylene and embedded in paraffin wax. Serial sections of 5 micron thick were obtained using a rotary microtome. The tissue sections were deparaffinised, hydrated and stained using the routine haematoxylin and eosin staining method (H&E). The stained sections were examined under the light microscope fitted to a digital camera and lap top.

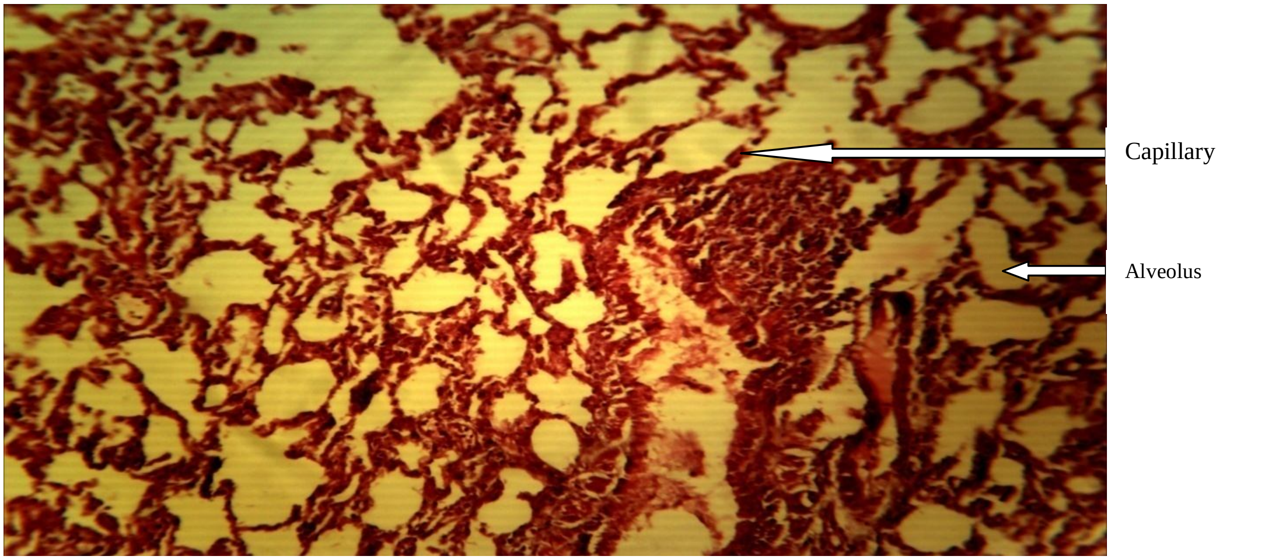

Plate. 1: Photomicrograph of Normal lung of Wistar Rat of group I. X100, H&E.

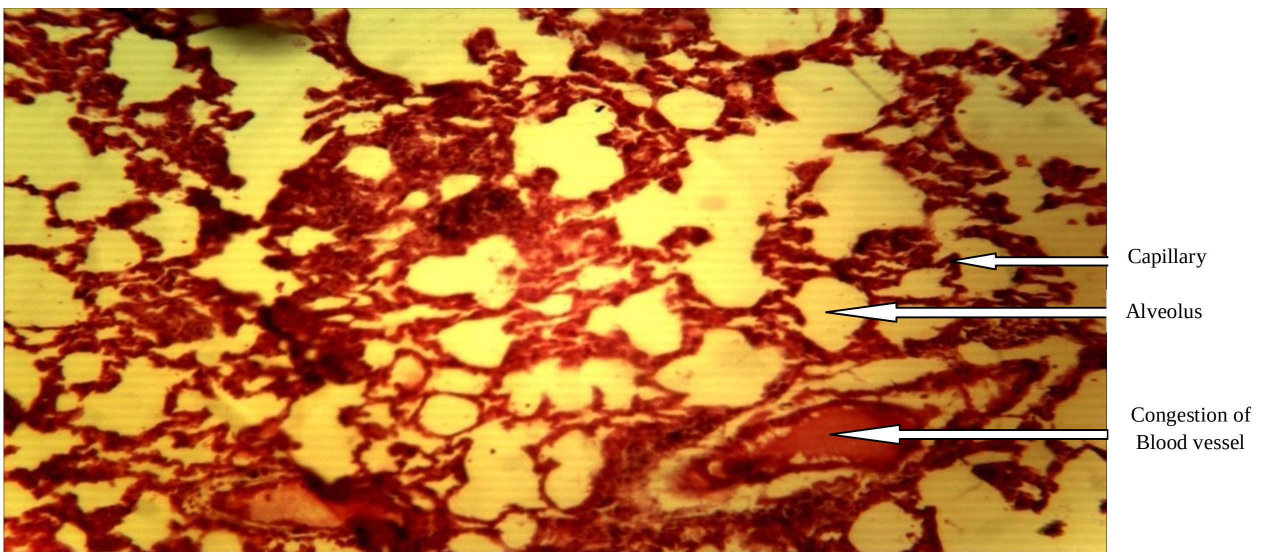

Plate. 2: Photomicrograph of lung of Wistar Rat of group II showing congested blood vessel engorged with blood. X100, H&E.

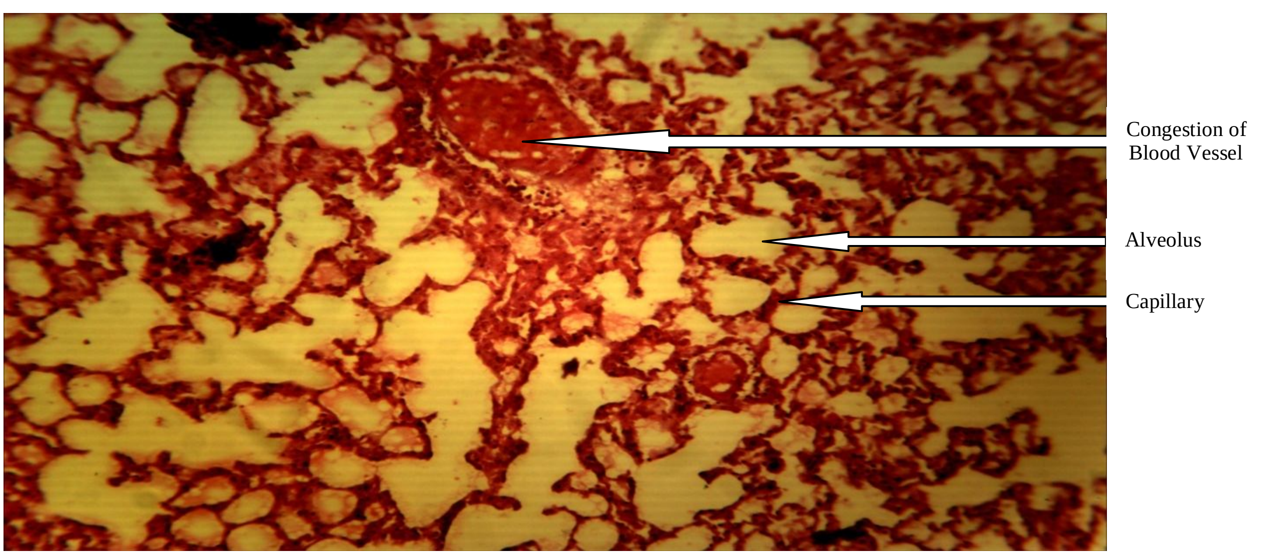

Plate. 3: Photomicrograph of lung of Wistar Rat of group III showing congested blood vessel engorged with blood. X100, H&E.

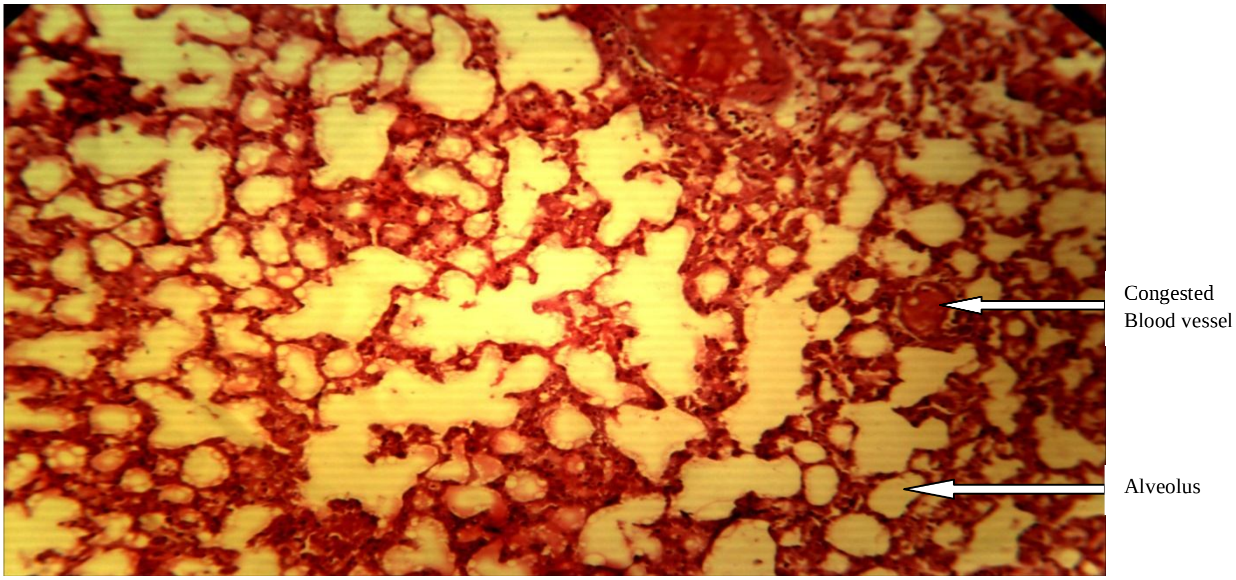

Plate. 4: Photomicrograph of lung of Wistar Rats of group IV showing congested blood vessel engorged with blood. X100, H&E.

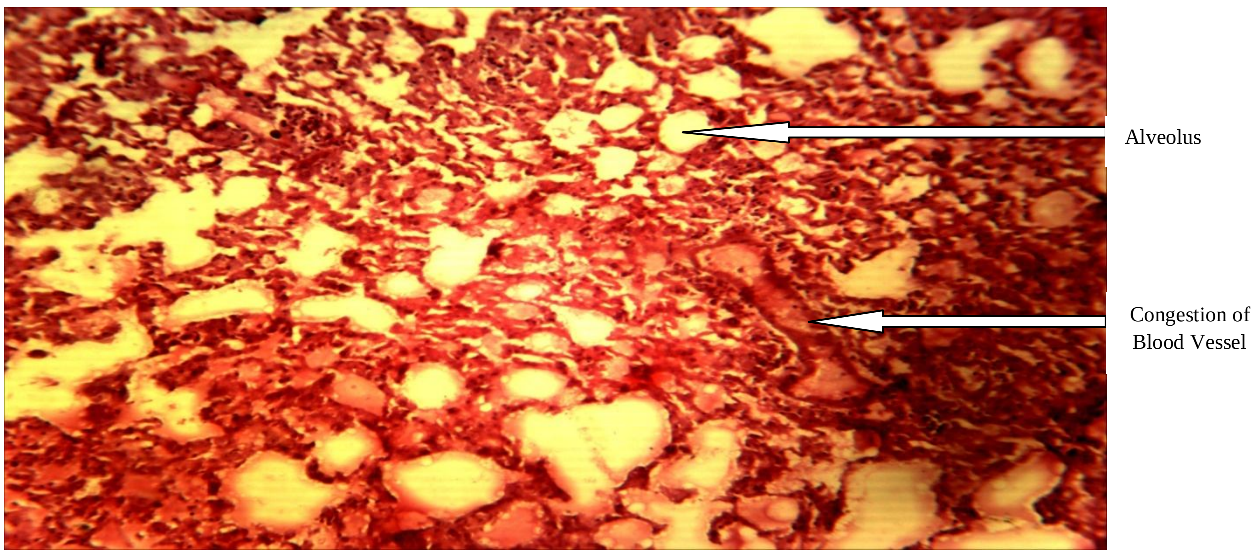

Plate. 5: Photomicrograph of lung of Wistar Rats of group II showing congested blood vessel engorged with blood with areas of the alveolar spaces filled with materials. X100, H&E.

There is little indication that aluminium is acutely toxic by oral exposure despite its widespread occurrence in foods, drinking-water, and many antacid preparations (WHO, 1997). In 1988, a population of about 20 000 individuals in Camelford, England, was exposed for at least 5 days to unknown but increased levels of aluminium accidentally distributed to the population from a water supply facility using aluminium sulfate for treatment. Symptoms including nausea, vomiting, diarrhoea, mouth ulcers, skin ulcers, skin rashes, and arthritic pain were noted. It was concluded that the symptoms were mostly mild and short-lived. No lasting effects on health could be attributed to the known exposures from aluminium in the drinking-water (Clayton, 1989). Many researchers have found elevated Aluminium levels to be associated with a decline in visual memory, attention, concentration, frontal lobe function and lower vocabulary scores in hemodialysis patients (Bolla *et al.,* 1992). In our study, the histology of lungs of wistar rats in control group I showed normal structures of the tissue; the aluminium treated groups II to V showed congested blood vessel engorged with blood, with areas of the alveolar spaces/ducts filled with materials that covered some of the alveolar ducts (See Plates II-V) when compared with the control group (See plate I). This was in concord with findings that revealed that aluminium chloride exposure was implicated to have negative effects on behavioural endpoints of wistar rats (i.e. alters behaviour), have negative effects on anxiety-related behaviour of wistar rats as it increased the rate of anxiety in aluminium treated rats, said to have neurodegenerative effects on the histology of cerebral cortex of adult wistar rats especially at higher dose and had deterimental effects on the kidney of wistar rats (Buraimoh, *et al.,* 2011a; Buraimoh, *et al.,* 2011b; Buraimoh, *et al.,* 2012a;

Buraimoh and Ojo 2012a). But it was in contrast with other studies which revealed that aluminium chloride exposure had no effects on the histology of the epididymis, stomach and that the effects on the cerebral cortex of adult wistar rats were not transferable to the offspring (Buraimoh, *et al.,* 2012b; Buraimoh and Ojo, 2012b; Buraimoh, *et al.,* 2012c). Our present study therefore revealed that aluminium chloride exposure was detrimental to the histology of the lungs of wistar rats (Plates II-V) and hence could in turn negatively affect respiration. Therefore, caution should be taken in its usage.

CONCLUSION

Based on our observations, we therefore conclude that aluminium chloride exposure had detrimental effects on the histology of the lungs of wistar rats, which was eminent in the congested blood vessel engorged with blood, with evidence of congestion and hemorrhage.

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