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A case report of pulmonary anthracosis in a free-ranging infant rhesus macaque (Macaca mulatta): histopathology and environmental health implications

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Abstract

The study aimed to describe a rare case of pulmonary anthracosis in a free-ranging infant rhesus monkey in Bangladesh. An infant Rhesus monkey (*Macaca mulatta*) just under 1 year of age, was admitted to a veterinary hospital in Bangladesh with clinical signs including loss of appetite, gradual weight loss, weakness, dehydration, depression, breathing difficulty, and mild nasal discharge. The animal died suddenly within 24 hours of admission. A necropsy was performed on the infant monkey, and tissue samples were collected and sent to the Department of Anatomy and Histology at Chittagong University of Veterinary and Animal Sciences (CVASU) for histopathological analysis. Gross examination revealed numerous uniformly distributed black spots in both lungs. The lungs showed significant inflammation, were firm, swollen, and abnormally colored, but no nodules or fibrosis were observed. Microscopic examination revealed black carbon particles, inflammatory cells, edema fluid, and brown hemosiderin-containing macrophages in the pulmonary interstitium. Diffuse alveolar damage with exudate and carbon particles was also observed. The infant monkey died from cardiopulmonary failure due to chronic respiratory distress. The presence of pulmonary carbonization indicates poor air quality in the animal's habitat, which may pose a threat to the health of both wildlife and humans.

Keywords: Infant Rhesus monkey, Post-mortem, Pneumonia, Carbon particles, Poor-quality air.

1. Introduction

The Rhesus monkey (*Macaca mulatta*) is a member of the genus *Macaca*, the family *Cercopithecidae* of Old World monkeys, the order of the *Primates*, and the class of mammals (*Mammalia*) [1]. The largest native range of any non-human primate is thought to belong to the Rhesus macaque, which is endemic to the whole continent of Asia. It is one of the most often utilized laboratory animals worldwide, particularly for biomedical research [2]. In Bangladesh, Rhesus macaques have been seen in urban and woodland environments. The monkeys are split into many groups and found in Dhaka, Madaripur, Rangamati, Bandarban, Chittagong, Khulna, and Satkhira districts of Bangladesh. The largest population of Rhesus in urban areas was recorded at Charmuguria in the Madaripur district [3]. Furthermore, Rhesus monkeys are housed in cages in several zoos and safari parks for display and education [4]. They have coats that are either brown or grey, often lighter above the waist and on the belly and darker on the lower back. The rump and face are hairless and have a reddish hue. The average height of adult males is 53.1 cm (1.74 ft), while that of adult females is 46.8 cm (1.54 ft). Every population has a different average weight, but normally, males are 2-3 kg heavier than females [5]. Births usually take place in the spring or summer, while breeding often takes place in the fall and winter. The female gives birth to one infant a year [6]. Infant weight at birth is 0.4 – 0.55 kg and yearlings weigh an average of 1.3 kg [6, 7].

The macaque is classified as a vulnerable species in Bangladesh by the International Union for Conservation of Nature (IUCN) as one of the second most susceptible species [8]. The species faces challenges because of fragmented populations, shifting terrain, habitat destruction, and illicit commerce [9]. They are affected by various infectious diseases, including hepatitis B, pox virus, respiratory syncytial virus, campylobacteriosis, tuberculosis, and balantidiasis. Furthermore, pulmonary and gastrointestinal issues are among the most prevalent issues that captive monkeys face [10, 11]. Infant monkeys are the most affected by respiratory diseases such as pneumonia and their death rate is relatively high. An increased risk of pneumonia in infants has been linked to poor air quality [12]. Bangladesh ranks 166th out of 180 countries in the Environmental Performance Index for Air Quality, highlighting

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that air pollution is a serious problem for the country. Toxic pollutants in the air, soils, and water sources pose a significant negative impact on both wildlife [13] and captive zoo animals, e.g., aerosolized lead [14].

Anthracosis is the blackish pigmentation of the respiratory system involving the lung parenchyma [15] and tracheobronchial tree [16]. The inhalation of dust and smoke is to be blamed for anthracosis [17]. The main agents causing anthracosis are carbon, free crystalline silica, mica, kaolin, and other silicates [18, 19]. When particles enter the interstitium, it's deposited there and are ingested by macrophages in the alveoli. Chronic exposure to these substances is thought to be the cause of anthracosis development [20]. Reports on anthracosis in domestic and wild animals [21], dogs [22], and lion-tailed macaque [23] can be found.

Nevertheless, relatively few studies have been conducted on diseases of wild monkeys in Bangladesh, and this case aims to describe gross postmortem and histopathological findings of a single infant wild Rhesus macaque, which died after being rescued from a roadside.

2. Case presentation

2.1. Animal

In January 2024, an infant rhesus monkey (*Macaca mulatta*) was found lying on its side, extremely weak, and struggling to breathe by a forest road in Ukia, Cox's Bazar, Bangladesh. The monkey was rescued and taken to the Ukia Veterinary Hospital for clinical evaluation and treatment. Upon admission, based on its weight (2.15 kg) and physical development, the infant was estimated to be less than one year old. Clinical examination revealed anorexia, progressive weight loss, dehydration, lethargy, difficulty breathing, and small amounts of serous nasal discharge; the monkey preferred to stay alone in a corner of its cage. Despite supportive treatment, the monkey's condition rapidly deteriorated, and it died within 24 hours of admission without exhibiting any characteristic symptoms. An autopsy was performed 30 minutes post-mortem (Figure 1). The carcass was extremely emaciated, with brown fur, a pink face, and exposed hindquarters. No external injuries or markings were observed. The body measurements include: height 1.4 feet, tail length 1.0 foot, forelimb length 0.8 feet, hindlimb length 1.4 feet, chest circumference, abdominal circumference and waist circumference 0.13 feet, 0.12 feet and 0.11 feet respectively. However, the infant monkey's clinical signs included decreased appetite, gradual weight loss, weakness, dehydration, depression, sitting in the corner of the cage, breathing difficulty, and mild nasal discharge. After 30 minutes of death, the infant was submitted for a full post-mortem examination (Figure 1).

2.2. Necropsy

The infant monkey was evaluated by two veterinary specialists who also conducted the postmortem assessment following established guidelines [24]. A comprehensive set of tissues (trachea, lungs, stomach, small and large intestine, liver, spleen, kidney, and heart) was fixed in 10% neutral buffered formalin solution for a period of 72 hours and submitted for histopathological examination to CVASU's Department of Anatomy and Histology.

2.3. Histopathology

The collected tissue samples were fixed in 10% buffered formalin, followed by washing, dehydration, cleaning by xylene, paraffin infiltration, and embedding. The tissue was cut at a thickness of 5 µm and stained with hematoxylin and eosin.

2.4. External morphology

The Rhesus monkey (Macaca mulatta) exhibited a brown to grey coat, a pink face, and a bare rump. It had a body height of 1.4 ft, a tail length of 1 ft, and a weight of 2.15 kg. The forelimbs and hindlimbs measured 0.8 ft and 1.4 ft, respectively. The cachectic infant monkey's skin color was brown but had no external marks or lesions.

2.5. Macro and microscopic observation of internal organs

Gross lesions were primarily limited to the lungs, in which all lobes of the right lung and three lobes of the left lung were pale and diffusely blackish-spotted. The blackish deposits were found externally in the shapes of minute dots of more or less the same size, scattered uniformly across all the lungs. Both lungs were inflamed, firm, swollen, and discolored. However, the lungs showed no fibrosis or nodules. Furthermore, there was no inflammatory fluid or exudate in the trachea. The spleen, liver, and heart were all normal in terms of size and appearance. Microscopically, exudative fluid and carbon particles were seen in the alveoli. Black-colored carbon particles, hemorrhage, and brown-colored hemosiderin-laden macrophages were seen in the interalveolar space. Exudate with inflammatory cells was seen within the bronchioles of the lungs, which coincided with the gross findings.

The visible lesions were mainly confined to the lungs, with all lobes of the right lung and three lobes of the left lung appearing pale and diffusely dotted with black spots (Fig. 2A). The spleen, liver, and heart were normal in size and appearance (Figs. 2B-D).

Microscopic examination revealed exudate and carbon particles within the alveoli (Figs. 3D and 3E). Black carbon particles, hemorrhages, and brown hemosiderin-containing macrophages were visible in the interalveolar spaces (Figs. 3B and 3C). Exudate containing inflammatory cells was observed within the bronchioles (Fig. 3A), consistent with macroscopic findings.

3. Discussion

Respiratory diseases become more severe, especially in infants, due to air pollution. A high prevalence of several acute lung diseases, which together account for approxi-



Fig. 1. Dead infant Rhesus monkey in the Ukhiya Veterinary Hospital, Cox's Bazar, Chattogram, Bangladesh.

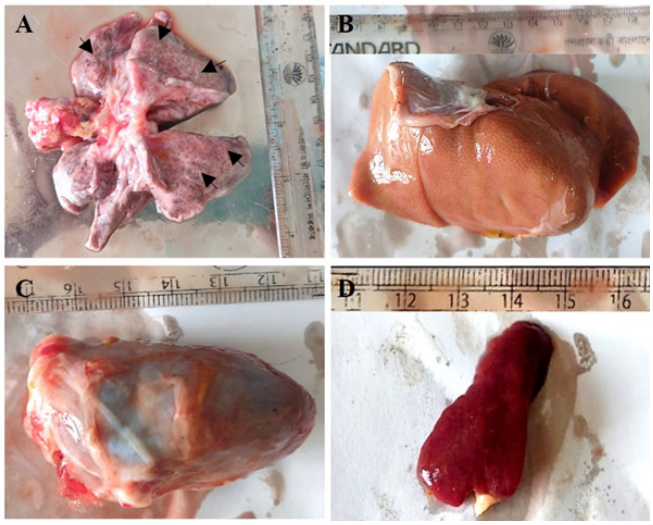


Fig. 2. Different organs of the infant rhesus monkey, A. Lungs (blackish color, firm and swollen), Arrows indicating numerous black spots, B. Liver (normal), C. Heart (normal), and D. Spleen (normal).

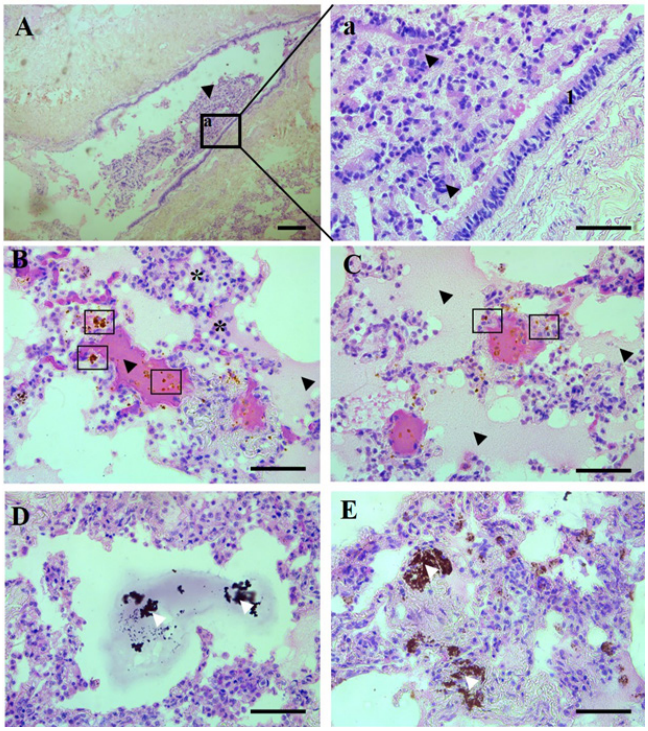


Fig. 3. Histopathological findings of the lung (H&E staining). (A) Inflammatory cells and exudate within a bronchiole (arrows). (a) Higher magnification of inflammatory cells in exudate. (1) Bronchiolar epithelial lining. (2) Cilia. (B, C) Hemosiderin-laden macrophages (square boxes) and hemosiderin pigment in the interalveolar space. Stars indicate edematous fluid. Arrows indicate exudative fluid. (D, E) Carbon particles (arrows) in the alveoli and interalveolar space. Scale bars: 100 µm (low magnification), 50 µm (high magnification).

mately a third of all pediatric fatalities, is particularly connected with outdoor pollution in large cities [25, 26]. In the current investigation, abundant block spots were found evenly distributed throughout the lungs due to the deposition of carbon particles in the alveolus and interalveolar space. It has been reported that the inhalation of polluted air gradually develops blackening of the lung [27] and microscopically, carbon particles are seen in alveolar macrophages in the interstitial space [28]. Because alveolar macrophages are the first line of defense against invading inhaled environmental particles like coal, silica, asbestos, and tissue debris [29]. Thus, specific environmental particles like carbon are engulfed by macrophages in an attempt to keep them out of the bloodstream, causing carbon particles to accumulate in the interalveolar space [28] called anthracosis.

The presence of hemosiderin-rich macrophages in the alveoli and interstitium provides further insight into the pathophysiology of this case. The accumulation of hemosiderin usually indicates a previous alveolar hemorrhage, which may be due to damage to the alveolar-capillary membrane.[35] Inhaled particulate matter, including carbon particles, can directly damage type I alveolar cells and microvascular endothelial cells, leading to disruption of the air-blood barrier.[32, 33] This damage results in cytoplasmic blistering, necrosis, and basement membrane shedding, followed by leakage of fibrin-rich exudate and red blood cells into the alveolar space.[34] The exudated red blood cells are then phagocytosed by alveolar macrophages, where heme is broken down and iron is stored as ferritin, eventually being converted into hemosiderin, which appears as golden-brown granules within the macrophages.[35, 36] These hemosiderin-rich macrophages, also known as “heart failure cells,” have traditionally been associated with pulmonary congestion caused by cardiac dysfunction. However, in this case, the autopsy revealed no cardiac abnormalities, suggesting that the hemosiderin deposition was more likely caused by repeated alveolar hemorrhage and subsequent inflammatory damage resulting from long-term carbon particle inhalation, rather than primary cardiogenic pulmonary edema. The presence of edematous effusion in the interalveolar spaces further supports the view that particulate damage leads to increased capillary permeability. This pathophysiological cascade, triggered by carbon particle deposition, followed by inflammatory cell infiltration, alveolar hemorrhage, and edema, ultimately resulted in diffuse alveolar damage and progressive respiratory dysfunction, ultimately leading to cardiopulmonary failure in the infant monkey.

Apart from this, diffuse infiltration of the inflammatory cell in the alveolar wall, interstitial space and bronchiole was found in this study, along with the accumulation of exudative fluid in the alveolus and interstitial space. Environmental particles are associated with the incidence and severity of chronic obstructive pulmonary disease (COPD) [30, 31]. Moreover, several noxious chemicals can harm the two main cells that compose the air-blood barrier, type I pneumocytes and capillary endothelial cells. Injury to these two types of cells shows cytoplasmic blebbing, which is soon followed by necrosis, resulting in denudation of the basement membrane. The consequences of this destruction include the leakage of fibrin-rich exudates into the interstitial and air spaces, loss of the surface-active alveolar lining film, and pulmonary collapse [32, 33], the capillaries of the alveoli leading to bleeding into the alveolar space [34]. The alveolar hemorrhage causes hemosiderin to accumulate inside the pulmonary macrophages. The removal of extra blood is done by the alveolar macrophages. The extra iron from heme breakdown in the alveolar macrophages promotes intracellular ferritin molecules as the macrophages break down the erythrocytes. Hemosiderin complexes are produced as a result of further ferritin processing [35]. Our study also revealed that the

golden-brown color of hemosiderin-laden macrophages was found in the alveolar space and the interstitial space. It has already been reported that hemosiderin appears as golden-brown granules in macrophages in routine staining [36]. These macrophages are sometimes called "heart failure cells" because of their association with pulmonary congestion and congestive heart failure. This hemosiderin and pulmonary macrophages have been proposed as indicators of prior asphyxia events [37], which coincides with the clinical signs of an infant monkey. Children who died from sudden infant death syndrome had greater hemosiderin deposits in their lungs [38]. Accumulation of hemosiderin-loaded macrophages in the lungs induces pulmonary fibrosis, resulting in the progressive collapse of the lungs and diminishing gas exchange [39]. Additionally, particulate matter exposure can increase the likelihood of someone developing respiratory diseases. Thus, long-term exposure can lead to more non-human primates being affected by lung and cardiovascular conditions.

Air pollution is a serious environmental health hazard affecting urban humans and wildlife in Bangladesh [13]. Anthracosis is seen when high carbon particles are present in the lungs and is an indicator of poor air quality in the zoo [40]. Similarly, there have been reports indicating that the poor quality air is considered a risk factor for radiographically confirmed pneumonia in children under the age of 5 in Dhaka, Bangladesh [41]. The age groups of 5 to 10 years and over 10 years had the highest frequency of anthracosis [42]. Several factors may have contributed to the high frequencies observed in older animals, such as continuous exposure to air pollutants, environmental pollution, and animals' immunity [17]. A study has reported that lung samples of rhesus macaques in Dhaka Zoo were necropsied and showed anthracosis lesions as a public health forecast for its city dwellers [21]. Therefore, this study strongly suggests that the poor-quality air in the area may be a predisposing factor to pulmonary anthracosis. Studies reported that dust exposure is strongly linked to lung conditions such as inflammation, lung fibrosis, and pulmonary neoplasia. The limitations of this study were that there was no clear association between pulmonary inflammation and anthracosis grades. Although infiltration of inflammatory cells around deposited dust elements was observed in some cases, the possibility of other respiratory infections could not be ruled out [43–45].

According to the current study, the infant monkey died of cardiopulmonary failure due to chronic respiratory distress. The cause of death was not pulmonary anthracosis, but this condition indicated that the infant free-ranging monkey inhaled poor-quality air without any confusion. The poor-quality air extends into the urban and the forest, causing life-threatening conditions for wildlife. The government and non-government organizations need to take any initiative to minimize dust and smoke in animal habitats, thereby reducing the risks of anthracosis in wildlife.

This study has several limitations. First, due to a lack of advanced diagnostic equipment, microbiological or toxicological analyses could not be performed to rule out infection or poisoning factors. Second, the sample size was limited to only one animal, which restricts the general applicability of the results. Third, the specific duration and concentration of the animal's exposure to air pollutants could not be determined.

In conclusion, the infant monkey died of cardiopulmonary failure due to chronic respiratory distress. Diffuse black spots were found in the gross and histopathological findings in the lungs. This pulmonary anthracosis indicates that air quality was poor in the habitat and may pose a health risk to wild animals and humans. Deforestation and industrial development around the forest may be the main causes of the poor-quality air in the wildlife habitat. To further lower the amount of carbon dioxide, industrial sites must be located far from wildlife habitat and deforestation must be restrained.

This case study provides the first record of pulmonary anthrax in young wild rhesus macaques in Bangladesh, highlighting the potential health risks posed by ambient air pollution to wildlife. The findings call for urgent action to monitor and improve air quality in wildlife habitats to prevent similar lung diseases in animals and humans.

Data Availability

All data are available within the manuscript.

Acknowledgments

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Author contribution

MRP and SKS conceived and designed the study with comments from all authors. MRP wrote the original manuscript and SKS, SH, and ALA edited the manuscript. MRP, SH, SKS, and ALA performed the experiments. SH performed the postmortem examination and transported the samples to the Department of Anatomy and Histology, CVASU.

Conflict of interest

None declared

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Ethics approval statement

It was a dead infant monkey. Nevertheless, the study was conducted according to the guidelines provided by the ethical committee of the Chattogram Veterinary and Animal Sciences University, Bangladesh. The study was approved by the Ethical Committee of Chattogram Veterinary and Animal Sciences University (Approval No. CVASU/EC/2023/012).

Consent for publication

The Upazila livestock officer provides consent to publish the clinical details and any accompanying images.

Use of artificial intelligence tools

Some sentences in this manuscript were revised with the assistance of artificial intelligence, based language models to improve clarity and readability. All intellectual content, interpretations, and conclusions are solely the responsibility of the authors.

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