

Al Easkan disease: Desert storm pneumonitis: New data and round-up

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Abstract

Al Easkan Disease (Desert Storm Pneumonitis) is a desert related ailment that has been observed and studied in Coalition Forces of the Gulf War. In the first phase of the disease the fine Saudi sand initially triggered a hyperergic lung condition. This was further aggravated by a multitude of noxious agents present in the theater of operation. Various kinds of organic and inorganic pathogenic components contributed to an opportunistic infection of the lung. The second phase developed either in continuation with persistent symptoms or evolved after a few months latency period and features of complications of silicosis such as rheumatoid pneumoconiosis, lupus erithematosus, scleroderma, progressive crescentic glomerulonephritis, poliarteritis nodosa, ataxia sensory neuropathy, polymyositis and monoclonal gammopathy. Ultrastructural as well as microanalytical studies were completed by Scanning Electron Microscopy and where the Saudi sand dust elemental composition was determined by energy dispersive spectroscopy and X-ray photoelectron spectroscopy.

Contrary to previous beliefs sand particles less than one μm in diameter were present in substantial quantities in the Saudi sand and were pathogenic causing hyperergia and immunodeficiency. Pathogenesis of the sand dust induced immunodepression and its pathologic background are discussed. These support the recognition of a new clinicopathological entity. Al Easkan Disease or Desert Storm Pneumonitis.

1. Informed consent was obtained from study subjects, and human experimentation guidelines of Department of Military Affairs, Commonwealth of Pennsylvania were followed.
2. Views, opinions and findings contained in this report are those of the authors and should not be considered as an official Department of Defense USA, Department of Military Affairs PA, or NASA, USA, policy and decision.
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Introduction

The purpose of this paper is to report a disease specific to the Arabian Peninsula. Recent deployment of Coalition Military Forces to the Arabian Gulf and the successful campaign "Operation Desert Shield/Storm" have generated after-action reports which have provided us with "lessons learned" for years to come.

Al-Eskan village in Riyadh is one of the several villages King Khalid established. During operation Desert Shield U.S. Forces moved into the Al Eskan village. The allied soldier efforts to make their living quarters habitable resulted in freeing the dust which became aerosol in large quantities.

696,562 U.S.A. soldiers, airmen, sailors and coast guard personnel were deployed to Saudi Arabia and neighbouring countries and abruptly exposed to the desert environment. Other countries that deployed military force were Argentina 100 troops, Bangladesh 2000 troops, Czech Republic 170 anti-chemical warfare troops, Egypt 45,000 troops, France 15,200 troops including 4,000 rapid deployment and 1,200 chemical warfare troops, Great Britain 25,000 troops including the 1st Armored Division plus 9,000 support troops, Gulf Cooperation Council (Saudi Arabia, Oman, Qatar, U.A.E., Bahrain, Kuwait) 145,000 troops including rapid deployment force upto 10,000 troops, and 7,000 Kuwait troops who escaped after Iraqi invasion, Honduras 150 troops, Hungary 36 troops a military hospital unit, Morocco 4,700 troops, Niger 480 troops, Pakistan 15,000 troops, Senegal 500 troops, Sierra Leona 200 troops, and Syria 15,000 troops [1].

The primary pathomechanism of exposure to the etiological agent(s) was inhalation. In Riyadh, the average temperature ranged from 65 to 75°F, occasionally reaching 80°F and frequently dropping to the 50°F's overnight. The yearly average of suspended dust was found in 1982 already to be 3,810 $\mu\text{g}/\text{m}^3$ in the periphery of the city of Riyadh, 1,190 $\mu\text{g}/\text{m}^3$ in residential areas, and 667 $\mu\text{g}/\text{m}^3$ in a tree lined park. These Fig.s were over 10 times higher than those recorded in that years in Europe and in the U.S.A. [2]. The sand dust was laden with many allergic substances. Spores and pollens were also recorded. In spite of low natural humidity, generally less than 40%, a large number of fungal genera of the class Deuteromycotina (fungi imperfecti) mostly of a dry nature, were present in the atmosphere of the central, desert area of Saudi Arabia.

Local laundry services in Eskan village for exam

ple sprayed the U.S.A. Forces uniforms with Pyrethryn. Common pesticides used included pirethroids, d-phenothrin, chlorpiriphos, resmethrin, malothion, methamyl, lindane, oromethiphos, and DEET.

Materials and Methods

The troops were vaccinated for protection against: tetanus/diphtheria, polio, typhoid, influenza and received 5 cc gamma immune globulin (IgG).

The average hospital's troop strength was 282. The prevalence of respiratory disease was 43% (5% - 82%) of total sick call involving 7% of hospital personnel strength. The peak incidence appeared on the second week of deployment. It is reasonable to assume that the incident rate was much higher than this data suggests. These data do not include patients who were treated in Medical Treatment Facilities other than in the U.S.A. Compound; patients who did not report to any Medical Treatment Facility at all; or regarded their symptoms as trivial and were not documented.

Another area, Thumamah, was an abandoned summer palace of King Khalid. It was located with its surrounding hamlet near Riyadh. After a few days of exposure to the dusty environment. Al-Eskan disease occurred, accompanied by gastrointestinal complaints of diarrhea. The water was contaminated by *Escherichia coli* in pathogenic concentrations. The water supply, other than drinking water, came from a local artesian well through a processing plant and was collected in a water tower. Hundreds of pigeon bodies and dozens of feline' bodies were discovered in the water tower. This cistern was identified as the most plausible source of contamination.

Corticosteroids and immunosuppressive agents were added to the therapeutic interventions if have been indicated. No single drug or combination of drugs has been shown to halt the autoimmune manifestations of Al Eskan Disease.

We collected sand samples from different locations in the Theater of Operation; storing them in sealed plastic bags.

Sand Analysis

The sand samples were analyzed using Scanning Electron Microscopy (SEM), X-ray microanalysis by Energy Dispersive Spectroscopy (EDS), and X-ray Photoelectron Spectroscopy (XPS).

Specimen Preparation

The sand specimen, due to its small particle size, was directly poured onto indium foil which is extremely malleable. The foil was folded in half over the sand and compressed manually. The foil was reopened and the non-embedded particles were shaken off. For SEM and EDS analysis the foil was attached to an aluminum SEM mounting stub with carbon paint, and the entire specimen mount was coated with a 200Å layer of carbon or gold to prevent charging of the sand samples. For XPS analysis, the foil, with the embedded sand particles, was carbon painted onto a specimen stub.

Specimen Analysis

Photomicrographs were taken at different magnifications ranging from $\times 15$ to $\times 20,000$ using a JEOL JSM 840A Scanning Electron Microscope. Energy Dispersive Spectroscopy via an EDAX spectrometer was carried out. Elemental dot mapping, which images the site of X-ray emission for a specified element, was also performed. The effective sampling depth of EDS was $0.5\text{ }\mu\text{m}$ to $1\text{ }\mu\text{m}$ for the accelerating voltages used. X-ray Photoelectron Spectroscopy was carried out by a VG ESCA LAB MARK II system. The effective sampling depth of XPS was 10Å to 100Å . The first few atomic layers were probed.

Results

In the first phase the conjunctiva and nasal mucosa were congested. A few complained of epistaxis. There was a slight irritating dry cough during the first few days which increased at times in intensity to painful paroxysms. The sputum was scanty, absent, or at first mucoid, which later become mucopurulent and tan-colored like the ubiquitous sand dust in the environment. Abnormal signs about the lungs included dullness to percussion in the upper lobes and crepitating. Pleural reaction was minimal or absent. Symptoms and physical signs tended to disappear by the third week. When the gastrointestinal tract was involved nausea and vomiting were common early symptoms. The patients' tongues were also furry. Either constipation or diarrhea was present. In the more serious cases, radiologic examination revealed no or minimal signs of atypical pneumonia. In the second phase, patients exhibited general malaise, soreness and sores, fatigue, recurrent low fever (usually upto 108°F), frequent headaches, difficulties in concentration, memory loss, sleeping difficulties, inter-mittent mental confusion,

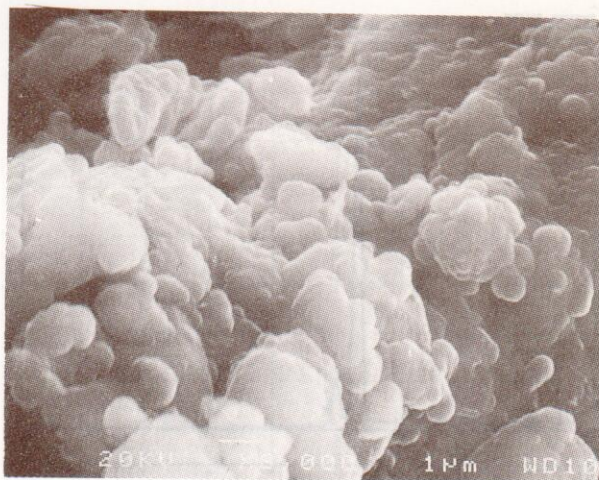


Fig. 1: Scanning electron micrograph of Saudi sand. Clusters of globoid particles. The individuals particles average size is $1\text{ }\mu\text{m}$ ($\times 5,000$)

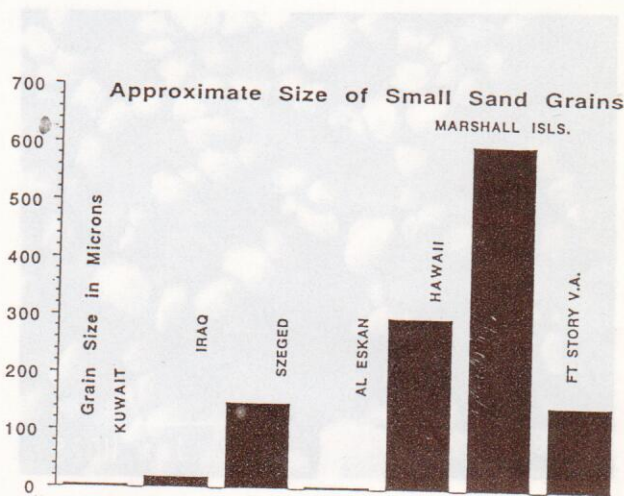


Fig. 2: Approximate size of small sand grains

irritability, significant increase insensitivity to multiple chemicals, newly acquired sensitivity to low level concentration chemicals, alcohol intolerance, lymph node enlargements, vertigo, tingling feeling and numbness of the extremities, blurred vision, recurrent eye infections, hoarseness, shortness of breath, chronic cough, recurrent paranasal sinusitis, chest pains, sensation of cardiac arrhythmias, halitosis, gum atrophy, and bleeding gums, loosening of teeth, abdominal pains, frequent nausea vomiting, recurrent

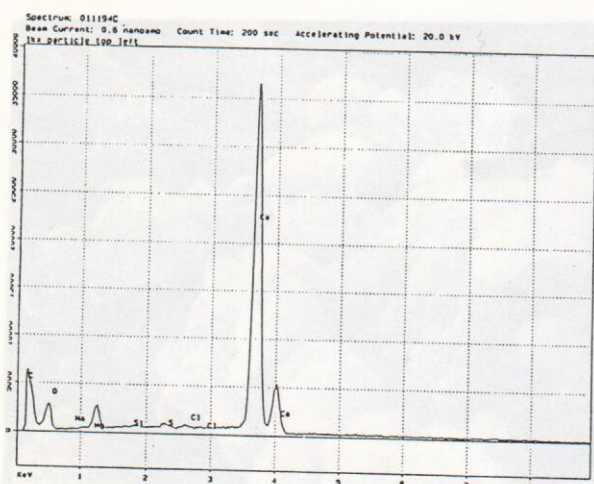


Fig. 3: Results of X-ray photoelectron spectroscopy a survey spectrum of Saudi Arabian sand

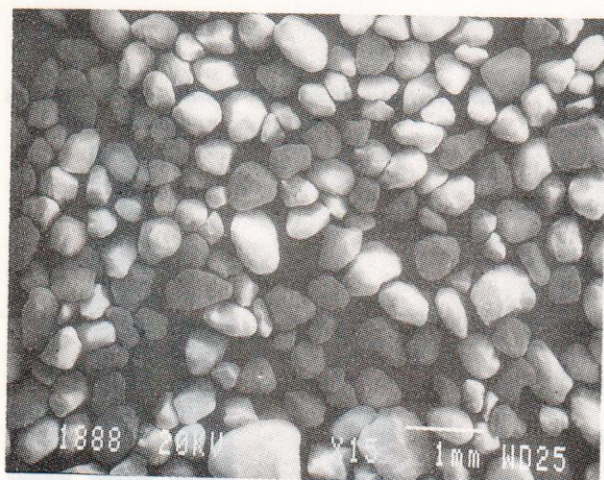


Fig. 4: Scanning electromicrograph of an odd sand sample from the Wadi of Batin vicinity of King Khalid Military City. Average grain size was from 0.5 to 1 mm. No free floating particles. The individual grains were rather faceted than rounded (x15)

diarrheas, rectal bleeding loss of anal sphincter tone, urinary incontinence, loss of urinary sphincter tone, water retention, peripheral edema, skin rashes recurred without predilection or with or without induration, pruritus, hyperpigmentations, photosensitivity, alopecia areata, hair loss. Laboratory studies usually revealed elevated erythrocyte sedimentation rate, pol

clonal hypergammaglobulinaemia, monocytosis and eosinophilia. Anemia was frequently observed. In some of the cases the serologic hallmark was the production of high titer autoantibodies directed against a variety of nuclear components.

In descending rank order the most common genera species of *Cladosporium* (24.7% annual means) smuts (21.2%), *Alternaria* (4%), *Ulocladium* (3.2%), *Basidiomycetes* (3.0%) and *Chaetomium* (2%) were the major components. The maximum concentration in February for *Cladosporium* was as high as 14,000/m³ of air in the humid coastal site and close to 6,000/m³ of air at a non coastal, central, dry desert site (Riyadh and vicinity).

Depleted uranium entered the body via inhalation of aerosolized uranium wound contamination and depleted uranium shrapnel (36 cases followed up as of 1944 among the U.S.A. Gulf War veterans. The effect of uranium on health when combined with sand or other putative causal agents of Al Eskan disease can only be speculated upon.

The sand grains appeared heavily agglomerated. The grains stacked upon themselves to create fibrous agglomerates with a high similarity to ferruginous bodies (Fig. 1). Photomicrographs revealed the average non-agglomerated grain to be in the range of 0.1 μ m to 0.25 μ m (Figs 1 and 2). Individual particles from the Saudi/Kuwait samples were measured in the range of 1 μ m to 1.5 μ m. Approximate 18% of suspended "free floating" particles had a range of 0.1 μ m to 0.25 μ m. Also smaller suspended or "free floating" grains and larger 5-7 μ m grains were present. The grain structure appeared rounded and smooth surfaced (Fig. 2). A typical EDAX microanalysis showed calcium to be in a much higher concentration than silicon itself, by a factor of 5 or higher. Other elements present were magnesium, aluminum and iron. Spectroscopy did show a peak for potassium however potassium and indium cannot be distinguished. Indium foil was used for the mount. The material studied appeared to be silicate with calcium, magnesium, aluminum, and iron substituted in the crystalline matrix. Some naturally occurring silicate which contain the above elements are anorthite (CaO Al₂O₃ 2SiO₂), diopside (MgO CaO 2SiO₂), akermanite (2 CaO MgO 2SiO₂) monticellite (CaO MgO SiO₂) and feldspars-lime (CaO Al₂O₃ 2SiO₂). In most silica minerals the concentration of Si predominates. Some minerals show a higher concentration of Al, Mg, Fe relative to Si, but not in a factor of 5 Ca to Si ratio as is found with Saudi sand. The bulk of the Saudi Arabian sand reveals an unusually

high concentration of Ca.

XPS reveals the surface of the Saudi sand to contain Al, Ca, Fe, Mg, O, C, Si. The C found is an adventitious species. After peak fitting and integration, the relative concentration of these elements reveal O to be in the highest concentration followed by Al. Ca is present on the surface in much less concentration than in the bulk. Qualitative XPS is extremely surface sensitive so the results reveal only the species residing on the top few atomic layers of the material.

SEM/EDS analysis was also performed on sand samples collected from the immediate vicinities of Al Eskan village, King Khalid Military City at Dhahran. Analysis revealed Si, S, Cl, Fe, and an excessive amount of Ca. Morphology of the silicates was identical to Al-Eskan samples. A single sample from King Khalid Military City was different from the rest of Saudi sand samples. In this sample the average grain size was in the range of 0.5-1 mm. No free floating particles were observed. The grains were faceted, rather than rounded (Fig. 3). Si/Ca ratio was 25 to 1. Other elements were Au, C, O, Al, Mg, K. The Au peak was an artifact due to the thin coating of specimens to dissipate electric discharges of the sand samples. In our continuing effort to further collect information on the physical and chemical nature of the elementary cause of El Eskan Disease we analyzed sand samples from some randomly chosen terrestrial locations. The sand from Al Eskan village in relation to the other samples exhibits a high calcium concentration and significantly small grain size. Note to the Tables: Szeged is in Hungary and Ft. Story is in Virginia Beach VA, U.S.A. (Fig. 4).

Discussion

During the war about 600 oil wells in Kuwait were set on fire. One million barrels of oil, weighing a million tons, went up in smoke daily which spewed 50,000 tons of sulfur dioxide (the chief constituent of acid rain) and 100,000 tons of sooty smoke into the atmosphere. The EPA found 1,4 dichlorobenzene, diethylphtalate and dimethylphtalate to be present, all of which irritated the respiratory system. Additional contributors were the emissions of internal combustion engines of thousands of trucks and tracked vehicles that use diesel fuel and fixed and rotary wing aircraft that burn a kerosene-like fuel. Every present electric

power generators contributed to hydrocarbon emissions. One can regard the petrochemicals as adjuvant factors acting upon the development of the second phase of Al Eskan Disease [3-5]. Although it seemed reasonable to conclude that these combined environmental emissions contributed to respiratory ailments, it is noteworthy to add that the theory of hydrocarbon poisoning was the cause of Al Eskan Disease of the service members of the South West Asia Theater of Operation is interesting.

From 1 September to 1 November, 1990, 20% of daily sick call was due to respiratory illnesses including allergic symptoms, involving 0.5% of the personnel daily, with a consequent 48-96 hours loss of duty time per person. Weekly medical survey reports of a Division further North of Al Eskan village in the Theater of Operations indicated that from 18 November 1990 until 16 February 1991, the prevalence of respiratory disease was 19% (9% - 28%) of total sick cell involving 1.3% of division strength. The peak incidence appeared at the end of December and the beginning of January.

It is significant that in the Forces deployed to the Theater of Operation the respiratory tract demonstrated a "locus minoris resistanciae" toward inflammatory disease. Hyams and his colleagues in later study, confirmed our observation of the significant prevalence of acute upper respiratory tract infection symptoms; the phase I of Al Eskan Disease [6].

In a desert environment, silicosis should be a main concern and, logically when the population is exposed to mixed dust of sand and pigeon droppings, ornitosis, psittacosis should be a consideration. It is observed that respiratory distress is very common in Saudi Arabia. The allergenic genera may remain airborne throughout the year providing extrinsic factors for allergenic and immunocompromised individuals at risk. It is reasonable to state that fungus spores may be important outdoors as well as indoor allergens and may also be responsible for perennial symptoms. Davis used a similar method (Hirst spore trap) in Kuwait and identified *Cladosporium* and *Ustilago* as two major spores in the Kuwait atmosphere [7].

The large majority of deployed service members were in a stable stage of mental health, and developed Al Eskan Disease. They did not have exposure to traumatic stress at all. Additionally it is reasonable to assume that neurologic involvement of the brain could affect the mind. It is hoped that we will avoid to recreate Charcot Disease when we try to explain all signs

and symptoms of the first and second phase of Al Eskin Disease as Post Traumatic Stress Disorder. However, it is interesting to note here that it has been theorized that irritation of the olfactory nerve endings beyond their sensitivity threshold may trigger a pathological response. This response would be the cause of a disorder called Multi Chemical Sensitivity. Parosmia sometimes follows viral infections of the upper respiratory tract. The sense of smell is disturbed in such fashion that most or all substances have a disagreeable odor. We suggest that the sand can be held responsible for the development of Multi Chemical Sensitivity Disorder in Al Eskin Disease, in some cases, rather than vivid anxiety neurosis.

The high and acostumed Calcium pulmonary intake and its stimulation by the busy trafficking electromagnetic environment, created by the high tech war equipment, needs further consideration in the pathogenesis of Al Eskin Disease. Hypercalcemia of any etiology can have dire consequences. The predominant and most devastating lesion usually occurs in the kidney: prominently in the collecting ducts, and distal tubes. On some occasions, hypercalcemia is in itself a life threatening situation. We want to stress that the high calcium content of Saud/Kuwait sand samples bear significance because of their ability to carry other organic compounds with it (allergens for example).

What is known is that the silicon particles of less than 1 μm average size were present in substantial quantities of what and were presumed to have been inhaled in large quantities. Irey from the Armed Forces Institute of Pathology autopsied 86 casualties from the Kuwait Theater of Operation. His report indicated no toxic findings but obstructive bronchitis and bronchiolitis. He observed birefringent sand particles in the lung parenchyma [8]. Hawass reported that, in his cases of Desert Lung Syndrome, transbronchial needle biopsy was performed and showed that the pulmonary miliary nodules, seen on the chest radiography were full of dust particles, being birefringent under polarized light (35).

Victims of silicosis are highly susceptible to pneumonia and the disease is likely to be atypical, with delayed resolution, organization, empyema and subsequent tuberculosis and carcinoma. It is suggested that dormant autoimmune conditions may predispose patients to silicosis. However, controversies and misconceptions regarding the pathogenicity of silicates, and silicon, data back to the recognition of silicone pneumoconiosis.

The silica grains are engulfed by the alveolar migratory macrophages. These cells have surface receptors both for IgG and serum complement. When these substances bind to the silica, particle uptake is enhanced (immune phagocytosis). The engulfed silica resides within phagosomes. Primary lysosomes (about 0.5 μm in diameter) empty a wide range of hydrolytic enzymes into these phagosomes, converting the phagosomes to digestive chambers called phagolysosomes. The Ergastoplasm and the Golgi complex became stimulated to produce and condense peptides, lysozyme and 1 L-1 immune interferon. The peptides invade the resident macrophages and the inter alveolar septal dendritic cells, enhancing a T cell reaction (CD4 and CD8 positive T cells). The peptides convert more virgin, resident macrophages to be committed to silica incorporation. The dendritic cells entrap antigens in the presence of the antibody and retain the antigen-antibody complexes [9]. It has been proposed that tissue dendritic cells carrying processed antigen from the periphery lose the capacity to process new antigens in the lymph nodes but acquire a unique capacity to stimulate naive T cells (10).

Upon interaction of silica grains with type II pneumocytes cellular hypertrophy, hyperplasia, and increased production of phospholipids identical to the surfactant have been observed.

The size and the shape of the inhaled particles is an important determinant of the nature and severity of the disease produced, fibrous shapes usually being the most pathogenic. The smooth rounded surface seems to promote an unchallenged passage of the particles all the way down to the Mononuclear Phagocyte System at the alveoli. Silica particles less than 1 μm are believed to be most pathogenic.

The initial early lung lesion is an extrinsic hyperergic alveolitis adjusted by cellular and humoral immune complexes and complement binding complexes. Subsequently, a fibrosing macrophagic alveolitis develops around the dust deposits. It is microscopic in extension (11).

Clinical Manifestations of Silicosis

- a) *Acute silicosis* occurs in subjects exposed to very high concentration of silica over few weeks to few months and results in rapid death [2].
- b) *Accelerated silicosis* develops after 5-10 years period of heavy exposure to silicates.
- c) *Chronic silicosis* with characteristic fibrogenicity observed that long exposure to silicon dust which could lead to three distinct pulmonary disorders:

silicosis associated pulmonary dysfunction with effort dyspnea; chronic airflow limitation; and chronic bronchitis symptoms.

Late Effect of Silicosis

- a) *Association of silicosis with other diseases:* The association of silicosis with tuberculosis and with lung cancer was recognized a long time ago [12,2,13]. The increased incidence of tuberculosis observed among silicate workers could be attributed to the accumulation of iron complexes by dust particles in the lung. When the iron complex made available to dormant mycobacteria it may act as a virulence factor, and also could be adjuvated by ineffective microbicidal responses of the silica laden macrophages. In 1938, Matz estimated that 75% of silicotics died of pulmonary tuberculosis [14].
- b) It was theorized that the condition was misinterpreted as tuberculosis in earlier centuries and was accountable for the high percentage of "tuberculosis" cases [15]. A desert related lung condition has been observed and reported as Desert Lung Syndrome from the Libyan Desert, the Negev Desert, the Najd are (the central province of Saudi Arabia) and from the Sahara Desert. The Desert Lung Syndrome is a simple siliceous pneumoconiosis and is connected with the living conditions of Bedouins in desert areas with frequent sand storms. Its cause is prolonged, excessive exposure to atmospheric sand dust. This is one of the manifestations of the environmental hazard in the desert. An association between "Desert Lung" and cataract has been observed.

Conclusion

Troops of Coalition Military Forces were abruptly exposed to desert environment. While Great Britain reported 11 cases with symptoms to the second phase of Al Eskin Disease, the Arabic Nationals reported none. It may support our assumption that by thousands of years of natural selection the inhabitants of the desert, the nomadic Bedouins acclimated to the environment but individuals with selected vulnerability may still react pathologically to the frequent sand storms, the victims of Desert Lung Syndrome. The U.S.A. compound in Riyadh was established in a village never inhabited by a human population. The village was

covered with a fine dust, a mixture of Saudi sand and pigeon droppings. A significantly large number of service member in an immunologically naive stage were abruptly exposed to this environmental hazard.

These all lead to the recognition of a clinicopathological entity "Al-Eskin Disease" as follows: The free floating sand particles less than 1 μm in diameter were easily inhaled into the alveolar sacs and alveoli. This area of the respiratory system responds by triggering immunopathologic reactions concomitant with gas exchange. The U.S.A. service members were in a special psychosomatic condition created by the War. They did not have a gradual exposure to this highly irritable environment. The sand dust triggered a hyperergic reaction which concluded in this clinically as well as pathologically recognizable condition. In some cases this condition was complicated by organic pathogens present with the dust, leading to various opportunistic infections. In other cases contributing factors, like inhaled petrochemical fumes, lead to a non-infectious respiratory ailment in the immunocompromised lung, or a mixture of these two [12,16].

During the second, immunodeficient phase of Al Eskin disease old diseases have presented unconventional clinicopathological features and revealed themselves as new disease (Table 6).

Some veterans-spouses reported illnesses and birth defects in children conceived after redeployment from the Theater of Operation. Concerns are raised about the possibility of indirect late effect(s) of sand-induced immunodeficiency. Our future plans include the assessment of the functional integrity of human spermatozoa, parameters of sperm concentration, motility, forward progression and morphology [17]. Our current investigators focus on the determination of immunosuppression on victims of Al Eskin disease, and the direct effect of the Saudi sand on in vitro macrophages and the host immunoresponse to the Saudi sand in comparison to other sand samples. Controlled reexposure procedures; inhalation challenges; and animal modeling of Al Eskin disease are paramount [18]. Further studies combining the disciplines of pathology, microbiology and studies of physical characteristics of various silicate substances should, hopefully, lead to a better understanding of this and related ailments; to more effective treatment, and better preventive medicine in the desert environment. As further symptoms and effects of service in the Gulf War surface we must direct our undivided attention to all possibilities and issues at hand.

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