Dracunculiasis

An incidental diagnosis in a Saudi female

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ABSTRACT

Dracunculiasis is a disabling, and economically crippling parasitic infestation transmitted by drinking contaminated water. Although the disease has been eradicated from most parts of the world, it is still endemic in some tropical African countries. Here we report a 65-year-old female from the southern region of Saudi Arabia with radiological evidence of heavy load of guinea worms. This case could represent the local reemergence of the disease.

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Dracunculiasis or Guinea worm disease, a tissue parasitic infection, has been eradicated from historically endemic areas, and is presently endemic in some African countries notably Sudan, Ghana, Mali, Togo, Ethiopia, and Somalia. Residual disease may be present in the Kingdom of Saudi Arabia (KSA) and Yemen.[1] Through PubMed search, we found that the last case of guinea worm infestation from KSA was reported in 1975.[2] Here we present a case of dracunculiasis after a gap of 3 decades. Our aim is to raise the awareness of clinicians on local reemergence of the disease, possibly from reservoirs of past residual infection, or new infection imported by the expatriates from endemic areas.

Case Report. A 65-year old, previously healthy Saudi female from Al-Baha, KSA, presented to us with 5 days history of a sudden onset painful, and itching erythematous macular rash over the dorsum of the feet, which progressed to blisters over 2 days. The pain worsened and became unbearable. She denied exposure to any systemic or local medication. Her systemic review was unremarkable. On examination, her vital signs were stable. She was in agony due to pain. She had multiple violet and red colored, tense blisters, over the dorsal aspects of the feet (more on the right foot). The underlying skin was erythematous, warm, edematous, and tender (**Figure 1**). There were several erythematous, tender macular lesions over the palmar aspect of the hands. Her conjunctivae, oral mucosa, and genitalia were free of lesions. Systemic examination was unremarkable. Initial laboratory work up was significant for mild microcytic anemia (hemoglobin 11 gms/dl, normal range (nr): 13-18g/dl, mean red cell volume 75 cμ, nr: 80-94 fL), thrombocytosis (platelet count 646/μl, nr: 140-450/μL), and high erythrocyte sedimentation rate (ESR) (103 mm 1st hour, nr: 10-20 mm 1st hour). Urea electrolyte, fasting blood sugar, and serum ferritin were normal. Her chest x-ray, and electrocardiogram revealed no abnormality. She was started on intravenous cloxacillin and ibuprofen. Blood cultures drawn at admission were negative. On day 8 of illness, she developed sudden onset shortness of breath associated with nonproductive cough, fever (temperature 38°C), generalized itching, and joint pains. The chest examination revealed diffuse bilateral wheezes. While repeat chest x-ray was normal, the complete blood cell count was significant for mild eiosinophilia (absolute eiosinophil count 824/μL, nr: 40-440/μL). Pulmonary function tests revealed obstructive pattern. Her respiratory symptoms responded dramatically to inhaled steroids and beta-2 agonists. In view of the persistent pain, magnetic resonance imaging of the feet and legs was obtained to rule out deep tissue infection, and an echocardiogram was requested to rule out septic emboli. Both studies were negative. The gram staining, and cultures of blister

fluid were negative. Histopathology of skin biopsy from the foot lesions revealed infiltration of dermis with eiosinophils and histiocytes, with granuloma formation that was suggestive of a foreign body reaction. The x-ray of the feet incidentally revealed soft tissue liner, and segmental calcifications suggestive of guinea worm infestation. Further radiographs revealed similar calcifications in other parts of the body suggesting a huge load of guinea worms (**Figures 2a-2c**). On further questioning, she admitted to drinking water from local wells, for many years. Cloxacillin was stopped, and she was started on oral metronidazole. Codeine was added to control her unbearable pain. Her clinical condition improved over the next 7 days. The pain subsided, and the skin lesions healed with desquamation. Repeat ESR, platelet count, and eiosinophil count were normal, and she was discharged home. Her follow up examination was unremarkable.

Discussion. Guinea worm infestation or dracunculiasis is a parasitic infestation caused by a tissue nematode *Dracunculus medinensis* (also known as serpent worm, dragon worm, Medina worm, and so forth), which is transmitted by ingesting microfilaria present in contaminated water from stagnant water from ponds and shallow, open wells. Microfilaria, released in the small intestine, penetrates the gut wall to settle in connective tissue of the abdominal and thoracic wall. The impregnated female worm (rarely more than one worm) migrates through the connective tissue to emerge from the skin to discharge microfilaria. The migration of the worm through the tissues is asymptomatic. A painful blister forms at the point of emergence (usually the feet and the legs), which ruptures on contact with water. The uterine contents are released causing severe

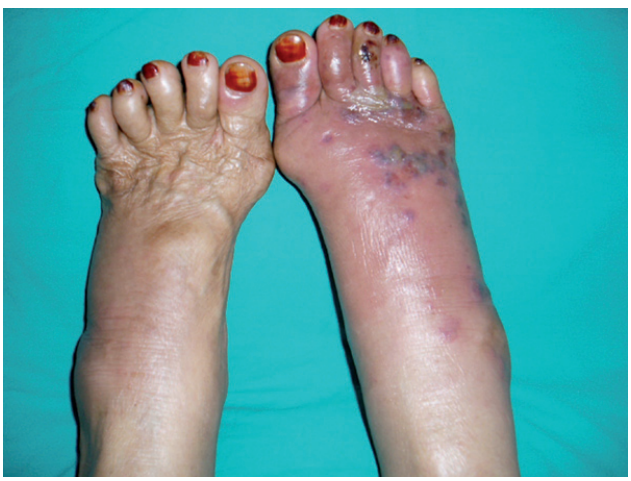

Figure 1 - Photograph of patient's feet showing blisters, and inflamed skin (more notable on the right foot).

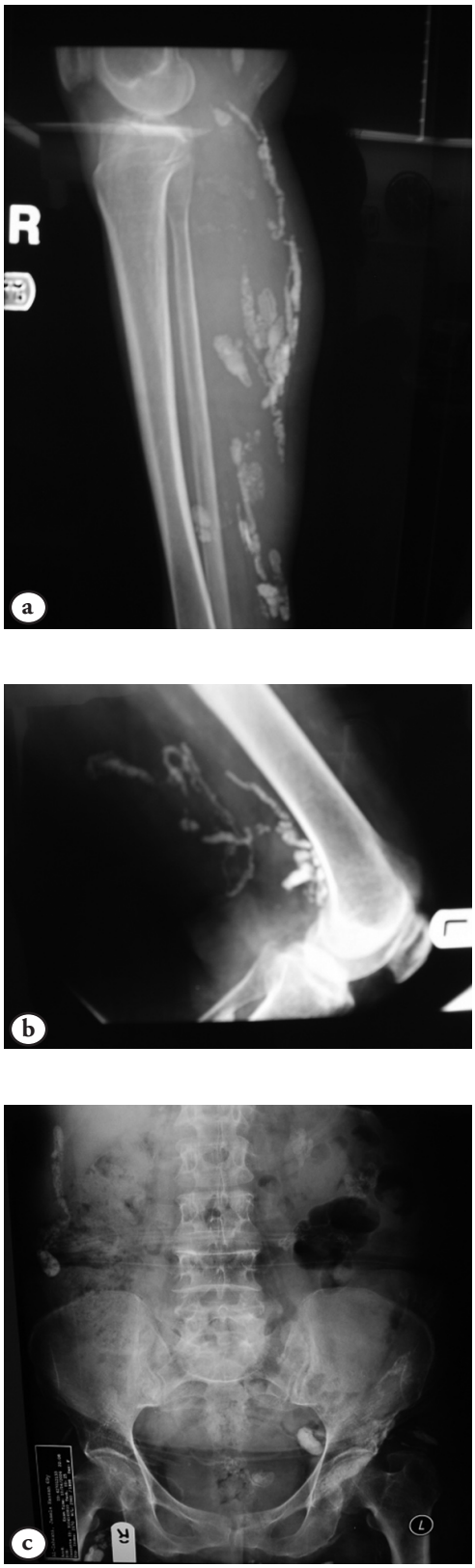

Figure 2 - Radiographs of **a**) right lower leg, **b**) right thigh and knee, and **c**) abdomen, showing linear and coiled segmental calcifications. Similar calcifications were also seen in radiographs of arms, hands, and neck.

tissue inflammation and pain incapacitating patients, for weeks to month. The worm emerges, and gradually extrudes out over weeks to months causing significant agony to the patient. Rupture of the blister leaves behind an ulcer that predispose the patients to cellulitis and tetanus. Sometimes, there is aberrant migration of the worms to various tissue organs.[3] An allergic reaction immediately prior to rupture of the blister may occur in 30-80% of patients manifesting as rash, fever, joint pains, urticaria, and bronchial asthma.[4] Adult onset bronchial asthma has been reported to occur as a sole manifestation of an allergic reaction to guinea worm.[5] Typical presentation and witnessing the worm emerging from the blister establishes the diagnosis of the disease. Alternatively, microfilaria can be seen under microscopy in blister fluid. Winding the worm around the stick as it emerges is the most effective treatment practiced for centuries. There is no effective pharmacological treatment for guinea worm. Nitroimidazoles have been shown to have an anti-inflammatory effect without aborting the infection, or facilitating expulsion of the worms.[6] Metronidazole is the preferred agent, and supportive measures include non-steroidal anti-inflammatory drugs, antiseptic dressings, and administration of tetanus toxide. The only way to prevent and eradicate the disease is to educate people, and provide safe drinking water. The calcified worms represent dead parasites, are asymptomatic in the majority of cases, and do not need to be treated. Sometimes, calcified worms may cause chronic, or intermittent foot and leg pain, and swelling. The diagnosis of dracunculiasis could not be established substantially in our patient as no worm emerged, and blister fluid was not sent for microfilaria as we initially did not consider the diagnosis. However, the clinical presentation, allergic manifestation, adult onset bronchial asthma, foreign body granulomas on the skin biopsy, together with the characteristic radiological findings strongly support the diagnosis of dracunculiasis. The radiological features, segmental linear, coiled or serpentine calcifications, with distal dense curvilinear opacities (**Figures 2a-2c**), are so characteristic of the guinea worm, that there is no differential diagnosis.[7] Sometimes, guinea worm infestation is incidentally diagnosed by radiology performed for other reasons, or during the evaluation of a painful limb. Our patient was fortunate not to have suffered in the past, despite having a heavy burden of worms. She most likely acquired infection while drinking contaminated water from the local wells, harboring the guinea worm microfilaria.

Dracunculiasis has not been reported from KSA since 1975. It appears from our case, that there might be reservoirs of guinea worm infection in the southern region of KSA. These might be residual infection from the past, or new infection imported by expatriates from endemic areas coming over to the Kingdom. There may be more patients asymptomatic or symptomatic, who are not seeking medical advice owing to the taboo attached to the disease. Also, health care workers are either under-reporting the condition or missing the diagnosis. Since many people from the southern regions continue to drink water from the wells, as revealed to us by the patient, such reservoirs of infection need to be identified so that preventive and eradication measures can be applied.

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