INTRODUCTION

One specific feature is that this area has been since millennia and continues to be endemic for a vector born infection leishmaniasis. This infection was noted for at least a century in both its anthroponotic and zoonotic forms, expressed clinically either as a cutaneous disease or as a systemic infection [1-3]. The Cutaneous form, in general, is a self-limiting infection that heals irrespective of whether treated or not. Still in some of these subjects (close to one third) the parasites from the skin lesion invade the blood and remain in circulation even after total healing of the skin lesion [4]. In contrast the systemic form, visceral leishmaniasis belongs to the list of “Neglected Tropical Diseases” and is considered a public health threat worldwide. It is referred to as Kala azar, which is often fatal in infants and children less than three years old and in old age (people above seventy) [5]. This parasite is propagated, in endemic zones, by sand flies which have been proven to exist in the Middle East for at least 120 million years [1,2,6]. After the sandfly bite the incubation period varies greatly. It extends from several months to more than ten years [5,7]. According to WHO report (2016) an estimated 900,000-1.3 million new cases and 20,000 to 30,000 deaths occur annually” [8]. Apparently this infection is spreading, although the report mentions that “only a small fraction of those infected by Leishmania parasites will develop the disease”. The rest of infected subjects (who resist clinical manifestation) represent a reservoir for these organisms. Over the past few decades, reports of patients with Kala azar in vector free areas started to be published and attracted a great deal of attention [5]. It was reported that not everyone who harbors this parasite develops the clinically described systemic leishmaniasis with its classical symptoms. Some patients may present with much milder symptoms depending on the integrity of the immune system and on, probably, the size of the inoculums when bitten by the infected sand fly. Such patients may present with non-specific symptoms, or remain unidentified with no symptoms at all. They are the larger proportion of the parasite infected population and are now referred to as “carriers” [9-12]. Leishmania is as pathogenic to many other mammals such as canines and feline animals in the Mediterranean basin [12-14] the infection develops very slowly with the clinical signs and symptoms appearing over a long stretch of time [13]. This reservoir for the parasite existed strictly in endemic zones although more recently it seems to be increasing in number in non-endemic regions [13]. The implications of all of the above reports, on public health are very serious not only in endemic regions but also in non-endemic areas.

DISCUSSION

As already mentioned, we chose leishmaniasis, a parasitic infection, to illustrate our objective. The disease was first described in 1902 in India by a British medical officer C. Donavan [15]. Then this was followed by mapping around the globe, all the regions where this infection existed. The presence of the infection with this parasites was organismically bound with the presence of the sand fly vector and the presence of the reservoir animal mainly canines. However, in the past three decades, reports of kala azar in vector free areas started to be published and attracted a great deal of attention [5]. It was reported that not everyone who harbors this parasite develops the clinically described systemic leishmaniasis with its classical symptoms. Some patients may present with much milder symptoms depending on the integrity of the immune system and on, probably, the size of the inoculums when bitten by the infected sand fly. Such patients may present with non-specific symptoms, or remain unidentified with no symptoms at all. They are the larger proportion of the Leishmania parasite infected population and are now referred to as carriers” [16]. Displacement of any group of Leishmania carriers into regions non-endemic for leishmania parasites makes out of such populations a time bomb. They can transmit the parasite by donating any organ including blood [16-26] (Figure 1,2) their body fluids carry the parasites, thus making the human carrier the center of a focus that can propagate unknowingly the infection
in the new environment [21]. Unfortunately most tests available rely on the presence of a positive immune reaction to Leishmania antigens, yet unfortunately this does not indicate whether the parasite is still present in the subject or not. A better test is the use of Skin Window [16] which aggregates pure agranulocytes on a slide in a high enough number to examine scores of cells for the presence of amastigotes. If inconclusive we can use indirect immunofluorescence tagged antibodies (sandwich technique) to detect the parasite. Certainly PCR can be used but all it reveals is DNA of the parasite in the carrier blood and again this is no proof whether the intact parasite is there or not unless it is used on scrapings from Skin Window. Furthermore the cost of PCR makes it available on a very limited scale, and not on waves of refugees.

CONCLUSION

To conclude we have witnessed over the last few decades, the remarkable increase in mobility of subjects among countries and even among continents such as from Africa, and the Middle East to Europe, and even more to Europe (central and northern), in addition to north America. More recently, we have been observing exodus of much larger groups of people from one geographic zone to another, with sometimes almost insignificant control. The objectives of this short review is to express concern on the potential hazard of a rapid spread of infections resulting from large scale migrations of peoples from endemic to non-endemic regions.

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