



Blood Group Types and Cutaneous Leishmaniasis in Iraq

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Abstract

Background: Leishmaniasis remains an important cause of morbidity and mortality in numerous areas throughout the world. The association of certain human blood groups with parasites is a controversial subject. In some cases positive relations have been demonstrated, as in schistomiasis. **Aim of the study:** is any association between blood group and cutaneous leishmaniasis. **Materials and Methods:** Three hundred patients who were clinically suspected for CL were enrolled. The diagnosis was mainly clinical from Department of Dermatology and Venereology, Al-Hussain Teaching Hospital, Nasiriyah-Iraq and Department of Dermatology and Venereology, Baquba Teaching Hospital, Diyala-Iraq and control group of 300 normal donors. **Results:** there is no statistical significant between them. This lead that blood group and Rh factor no risk factor in cutaneous leishmaniasis in Iraqi patients.

Keywords: Leishmaniasis, blood group, Rh factor, Iraqi patients.

Introduction

Leishmaniasis is a protozoal disease transmitted by sand fly vectors. There are several species of vector, each occupying a particular zoogeographical zone.

This disease is considered to be a zoonosis (an infectious disease that is naturally transmissible from vertebrate animals to humans), with the exception of *Leishmania tropica* which is often an anthroponotic disease (an infectious disease that is naturally transmissible from humans to vertebrate animals).

The disease is endemic in 88 countries, most commonly in tropical and subtropical regions. Clinical

manifestations of disease range from aggressive cutaneous ulcers to systemic multiorgan disease. (1, 9) It's still one of the world's most neglected diseases, affecting largely the poorest of the poor, mainly in developing countries; 350 million people are considered at risk of contracting leishmaniasis, and some 2 million new cases occur yearly. In the past 10 years, major scientific breakthroughs have been made in the treatment, diagnosis and prevention of leishmaniasis, and the prices of several key medicines have been reduced. These developments have facilitated implementation of sustainable national and regional control programmes; however, functioning control programmes are still rare, and mortality and morbidity from leishmaniasis worldwide show a worrying increasing trend. (2)

More than 90% of localized cutaneous leishmaniasis (LCL) cases occur in Afghanistan, Algeria, Iran, Iraq, Saudi Arabia, Syria, Brazil, and Peru.(3)

The various types of leishmaniasis are confined primarily to the Mediterranean basin, Southern Europe, Central Africa, and parts of Southern and Central Asia [Old World (OW)], and Central and South America [New World (NW)]. The infection is endemic in 88 countries, 72 of which are developing countries.

In Western countries, the incidence is increasing due to human immunodeficiency virus (HIV)-Leishmaniacoinfection, military appointment, and tourism in endemic countries. (4, 5)

There are several types of lesion. All tend to occur on exposed parts because all are transmitted by the sandfly.

Old World leishmaniasis manifests mainly in the skin and has also been called Baghdad boil, Oriental sore, leishmaniasistropica, Biskra button, Delhi boil, Aleppo boil, Kandahar sore, and Lahore sore. Mild visceral disease may occur. Skin lesions of New World infection have been termed uta, pian bois, and bay sore or chiclero ulcer.(6)

In Old World leishmaniasis, lesions may present in two distinct types. One is the moist or rural type, a slowly growing, indurated, livid, indolent papule, which enlarges in a few months to form a nodule that may ulcerate in a few weeks to form an ulcer as large as 5 cm in diameter. Spontaneous healing usually takes place within 6 months, leaving a characteristic scar. This type is contracted from rodent reservoirs such as gerbils via the sandfly vector. The incubation period is relatively short (1–4 weeks). The dry or urban type has a longer incubation period (2–8 months or longer), develops much more slowly, and heals more slowly than the rural type. In both types, the ulcer or crust forms on a bed of edematous tissue.

Rarely, after the initial or “mother” lesion is healed, at the borders of the healed area, a few soft red papules may appear that are covered with whitish scales and have the “apple jelly” characteristics of granulomatous diseases such as lupus vulgaris. These spread peripherally on a common erythematous base and are the lupoid type. This is also known as leishmaniasisrecidivans and occurs most often with the urban type of disease, caused by *Leishmaniatropica*. (7, 8)

In endemic areas where transmission is stable, children are especially affected, and the cumulative rate of infection as determined by the presence of scars and positive leishmanin tests may approach 100%. (10)

Pathogenesis

The resulting disease depends on the fate of the phagocytosedamastigotes. This in turn is function of numerous parasite- and host-related factors, as well as other factors that may account for geographical differences.(11), In general, parasites interfere with the signaling pathways, intracellular kinases, transcription factors, and gene expression of macrophages, compromising their ability to generate leishmanicidal substances. In addition, they impair dendritic cell activation, migration, and ability to secrete T helper 1 (Th1) cytokines. (12)

In the majority of cases, CL is a self-healing disease. Nonetheless, nodular lymphangitis and MCL can lead to disabling and atrocious tissue destruction. Full recovery can take months to years, and this period can be characterized by function impairment, susceptibility to secondary infection, and the development of disfiguring permanent scars. Because little evidence-based data are available, most therapy options have to rely on expert opinions (13), options are associated with significant toxicity and side effects. Therefore, a risk-benefit assessment must be made by an experienced clinician for each CL patient, and, in mild and indolent cases, a wait-and-see policy can sometimes be the best advisable option. Moreover, drug resistance is an emerging problem in the control of CL (14)

Several treatment options for CL are available. Pentavalentantimonials (sodium stibogluconate, Pentostam® or meglumineantimoniate, Glucantime) remain the first-choice treatment for CL in most countries. Alternative treatment regimens include miltefosine, pentamidineisethionate, amphotericin B, antifungal agents (e.g., ketoconazole, fluconazole, itraconazol), paromomycine, granulocyte macrophage colony-stimulating factor, and heat therapy or cryotherapy (15, 16).

In cases of a few (less than five) lesions, local therapy is preferred. (17)

Local treatment combining intralesional antimony and cryotherapy proved more effective than antimony or cryotherapy alone, although as monotherapies, both also show high cure rates (18, 19, 20).

Heat therapy has also proven to be effective but requires special equipment (21, 22).

Systemic treatment can be considered for multiple lesions, disfiguring facial lesions, or lesions at sites that make topical treatment less desirable. Systemic (oral) miltefosine treatment is a promising option for patients with multiple or complicated Old World CL (L. major) lesions (23, 24).

The diagnosis of CL is based on clinical features (supported by epidemiologic data) and laboratory testing. Numerous diagnostic methods have been described with a huge variation in diagnostic accuracy, including direct parasitologic examination (microscopy, histopathology, and parasite culture) and/or indirect testing with serology and molecular diagnostics.

The selection of the diagnostic test employed often depends on the available infrastructure and resources of the diagnostic facility and not on diagnostic accuracy. Here, we selected only general employed diagnostic methodologies for discussion. (25)

Direct Microscopy, Histopathology, and Culture

Parasitologic diagnosis is still considered the gold standard in leishmaniasis diagnosis because of its high specificity. This is typically undertaken by histopathologic examination of fixed tissue or parasite in vitro culture from material from suspected lesions. Microscopical diagnosis of CL is performed by the direct identification of amastigotes in Giemsa-stained lesion smears of biopsies, scrapings, or impression smears. Amastigotes appear as round or oval bodies, about 2–4 μm in diameter, with characteristic nuclei and kinetoplasts. The material from the ulcer margin usually has the highest yield. A comparative study between widely used scraping smears and fine needle aspiration cytology found a significant difference between the two methods in favor of fine needle aspiration in the detection of amastigotes and microgranuloma, slide background, and patient comfort (26). A simplified collection method is the press-imprint-smear (PIS). When compared with histopathology for the diagnosis of CL, PIS was positive in 85.3 % in study cases suspected of CL, and

histopathology was only positive in 44 %. PIS is considered a rapid and relatively sensitive method for the diagnosis of CL (27)

Blood Groups

Blood group antigens represent polymorphic traits inherited among individuals and populations. At present, there are 34 recognized human blood groups and hundreds of individual blood group antigens and alleles. (28, 29) blood group antigen expression differences can lead to variable host susceptibility to many infections which might be elevated or reduced. Blood groups can play a direct role in infection by serving as receptors and/or coreceptors for microorganisms, parasites, and viruses. In addition, many blood group antigens facilitate intracellular uptake, signal transduction, or adhesion through the organization of membrane microdomains. (30, 31)

Several blood groups can modify the innate immune response to infection.(32). Several distinct phenotypes associated with increased host resistance to malaria are overrepresented in populations living in areas where malaria is endemic, as a result of evolutionary pressures. (33, 34). Microorganisms can also stimulate antibodies against blood group antigens, including ABO, T, and Kell. (35) Finally, there is a symbiotic relationship between blood group expression and maturation of the gastrointestinal microbiome. (36)

Materials and Methods

This study was carried out in the Department of Dermatology and Venereology, Al-Hussain Teaching Hospital, Nasiriyah, Iraq and in Department of Dermatology and Venereology, Baquba Teaching Hospital Diyala, Iraq during the period from September 2016 to April 2017.

Three hundred patients who were clinically suspected for CL were enrolled. The diagnosis was mainly clinical -a typical chronic nonhealing indurated papule, nodule or plaque with or without crusting in patients referring to Outpatient Of Dermatology and Venereology in Al-hussain Teaching Hospital, Diagnosis were made clinically according to characteristics clinical features by more than one dermatologist in period of high outbreak of CL. The distribution of blood group type of all infected patients was compared with that of a control group of 300 normal donors. Statistical comparison was performed to find out the relationship between blood group type and CL, using t- test.

Both control group blood donors and CL patient were selected randomly regardless the age, sex or number of lesions

Results

There were 210 males and 90 females among CL patients (total 300 patients) .ninety two (31%)of the

patients with blood group A ,82 with Rh+ve and 10 Rh-ve. And 104 (34%) patients with blood group B, 98 patients with Rh+ve and 6 patients only with Rh-ve. Eighty (27%) patients with blood group O, 74 patients with Rh+ve and 6 patients only with Rh-ve. Twenty four (8%) patients with blood group AB,20 patients with Rh+ve and4 patients only with Rh-ve.,as show in fig.(1) and fig (2).

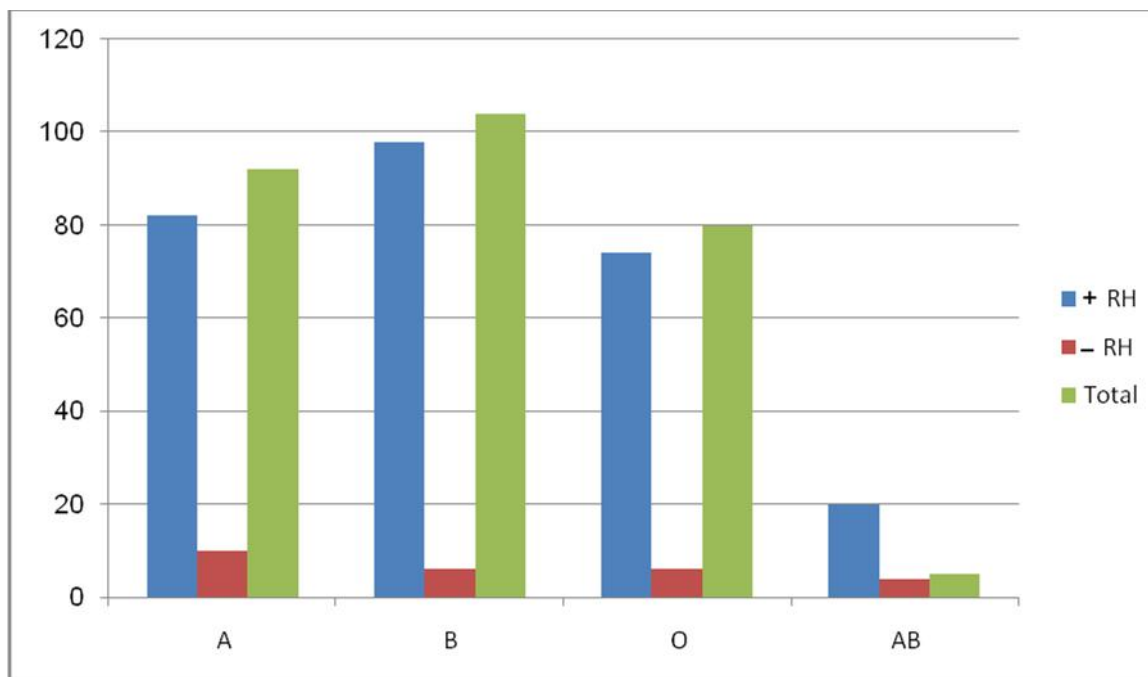


Figure (1): Number of patients with their blood groups.

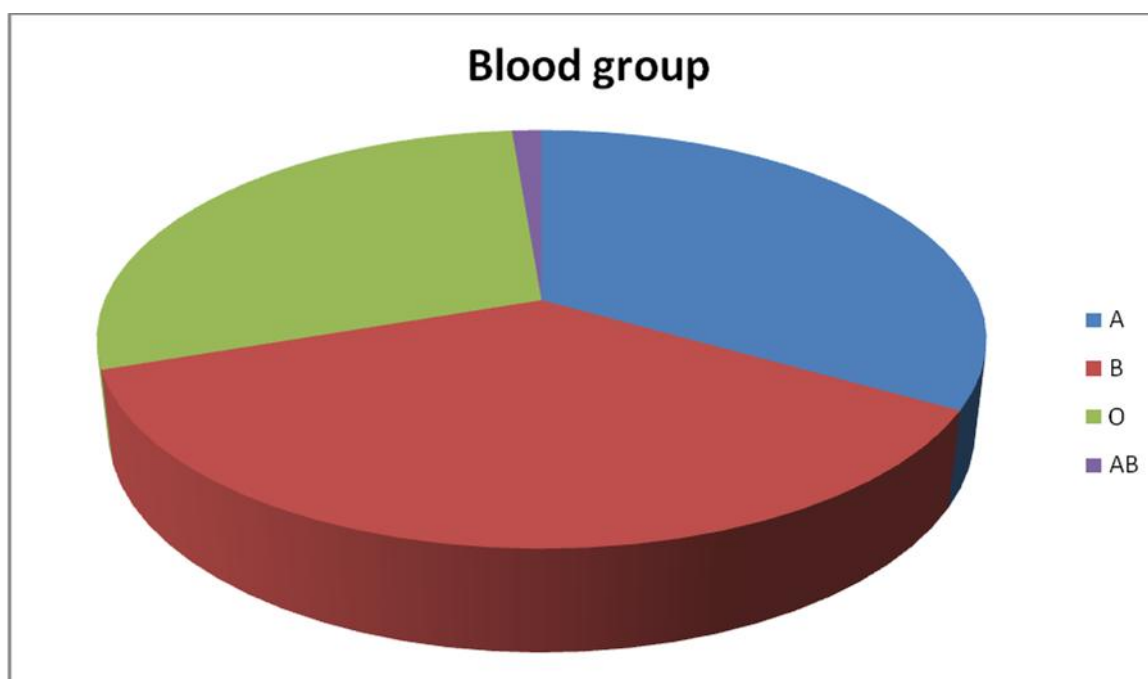


Figure (2): Percentage of blood groups of patients.

There were 210 males and 90 females among control patients (total 300 patients). ninety seven (32%) of the patients with blood group A, 90 with Rh+ve and 7 Rh-ve. And 110 (37%) patients with blood group B, 102 patients with Rh+ve and 8 patients only with

Rh-ve. Seventy three (24%) patients with blood group O, 67 patients with Rh+ve and 6 patients only with Rh-ve. Twenty (7%) patients with blood group AB, 18 patients with Rh+ve and 2 patients only with Rh-ve., as show in fig.(3) and fig (4).

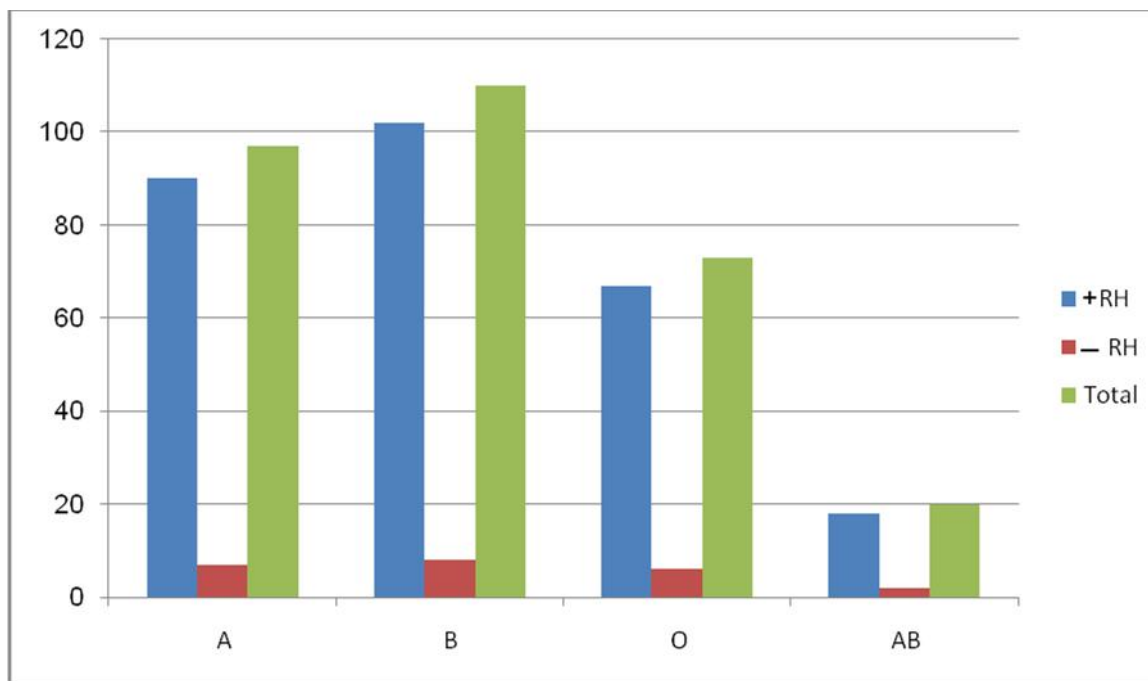


Figure (3): Number of control group with their blood groups.

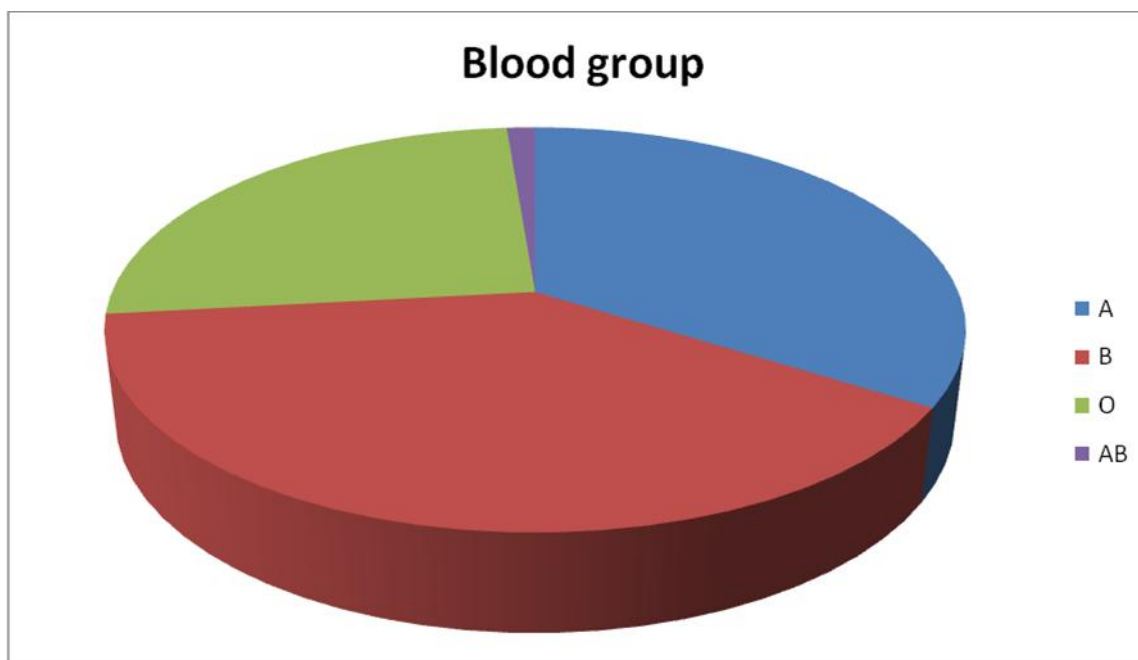


Figure (4): Percentage of blood groups of control group.

By use t-test to compare the results of patients with cutaneous leishmaniasis and control group the p value is 0.5 which mean there is no statistical significant

between them. this lead that blood group and Rh factor no risk factor in cutaneous leishmaniasis in Iraqi patients, as show in fig (5).

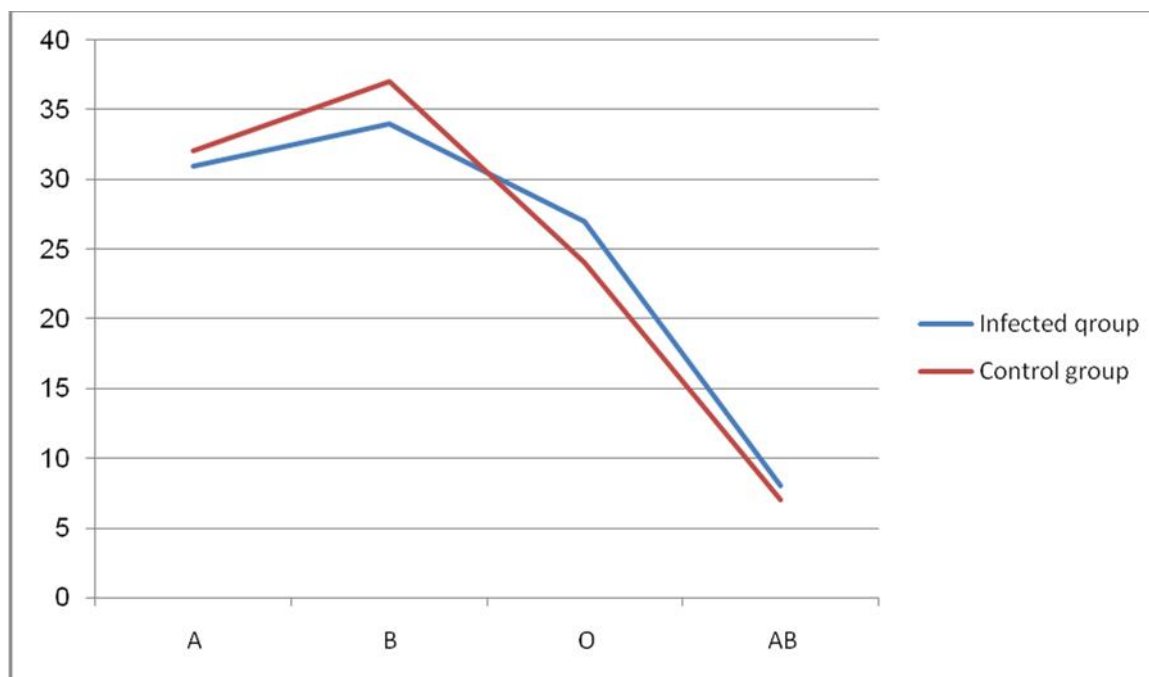


Figure (5) : Results of patients with cutaneous leishmaniasis and control group

Discussion

Leishmaniasis remains an important cause of morbidity and mortality in numerous areas throughout the world. The association of certain human blood groups with parasites is a controversial subject. In some cases positive relations have been demonstrated, as in schistomiasis [1] and Giardiasis[2], while in other cases, such as filariasis [3].

Cutaneous leishmaniasis (ACL) is still endemic disease in Iraq. (37)

For Iraq, the reported cases of CL were 1655/ year for 2004-2008, while the estimated annual CL incidence was 8300 to 16,500, which considered moderate underreporting (41). CL is endemic in majority of Middle Eastern countries including Iraq. The incidence is declining in time in some Middle East countries but continues to spread in others such as Iraq (42,43).

Our study was done in two cities first in Nasiriyah (**Nasiriyah** (is a city in Iraq. It is situated along the banks of the Euphrates River, about 225 miles (370 km) southeast of Baghdad, near the ruins of the ancient city of Ur. It is the capital of the DhiQar Governorate. Its population 2003 was about 560,000, making it the fourth largest city in Iraq), and the second one is Baquba. (is the capital of Iraq's Diyala Governorate. The city is located some 50 km (31 mi) to the northeast of Baghdad, on the Diyala River. In 2003 it had an estimated population of some 467,900 people).

Our results showed that the blood group was not a risk factor in the occurrence of CL. The findings failed to support the hypothesis of Greenblatt et al. (38) So, we conclude that ABO-Rh blood groups are not associated with the occurrence of CL in Nasiriyah and Baquba Iraqi patients. The conclusion of our study is similar to that of Aflatoonian M.R et al. (2007) (39) and Kumar et al. (2008) (40)which failed to support the hypothesis of camouflage, using blood groups.

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