The Use of Systemic Lupus Erythematosis (SLE) Biomarkers in Forensic Investigation: A Suggested Approach

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Received date: Feb 08, 2014, Accepted date: Apr 01, 2014, Published date: April 05, 2014

Abstract

Forensic investigations usually depend on collecting clues, evidences and references. Long time ago, gene banks and finger prints have found their application in forensic analysis of crimes and for identification of individuals in mass disasters. Teeth and oral tissues were also included before. Recent studies used the characteristics DNA extracted from teeth remains to identify totally deteriorated bodies. This article direct the attention toward the possibility of using diseases-associated biomarkers as a provisional tool for identifying individuals located in complicated forensic challenges. Systemic Lupus erythematosis (SLE) is one among the diseases that associated with the release of different biomarkers in body fluids. These biomarkers could help a differential identification of individuals at acceptable level of validity, however a disease-related biomarker banks should be established first.

Keywords: Biomarkers; Forensic investigation; Systemic lupus erythematosis; Autoimmune diseases; Adiponectin

Background

The oral cavity is a mirror reflecting the existing illness of different human body parts. A wide number of dermatological conditions can be manifested orally [1]. Dermatoses have frequently been reported with different frequencies in the oral cavity [1]. One of the most important concerns about systemic lupus erythematosis (SLE) is the presence of oral lesions, which is one of its diagnostic criteria [2]. In many cases, mucosal ulcers are the earliest manifestation of SLE that probably identify the disease. Khatibi et al. [2], proved that 102 out of 188 SLE Iranian patients (54.3%) had oral lesions, where both buccal and labial ulcers were the most prevalent (28.1%) [2].

SLE is a chronic autoimmune disorder of the connective tissue and blood vessels. With the active disease usually develops anti-nuclear antibodies (ANA) and anti-double strand DNA (Anti-dsDNA) autoantibodies against various components of cell nucleus leading to inflammation, vasculitis and immune complex deposition [3]. The immune complex deposition along with complement activation could be the cause of lupus nephritis, that frequently accompanied with hypocomplementemia [4].

The exact etiology of SLE is still unclear, although multifactorial interaction with genetic and environmental factors has been implicated. Children and adolescents represent 15-20% of all SLE patients [3] however its frequencies differ among racial and ethnic groups [5–7]. The overall prevalence of SLE-affected individuals in the general population is 1 in 2000 with a predilection for women [5,8]. The possibilities of using diseases’ biomarkers have not hitherto been applied in forensic analysis. Therefore, this article aims to suggest below some practical implications of SLE-associated biomarkers that may help characterizing individuals:

Adipocytokines (adiponectin) are soluble mediators derived mainly from adipocytes and are thought to play an important role in both inflammatory and immunity processes [9]. Adiponectin normally show a strong genetic references, with an additive genetic heritability of 46% [10]. The ADIPOQ gene consists of three exons and two introns spanning a 17-kb region and has been located on chromosome 3q27 [10,11]. The ADIPOQ gene was found to be the only major gene responsible for plasma adiponectin [12-15].

Different adiponectin levels were found to increasing the risk toward type 2 diabetes, atherosclerosis, coronary artery disease, and SLE too [9,10]. Some researchers [10] recently discovered low adiponectin levels in plasma of obese individuals suffering from type 2 diabetes, hypertension, atherosclerosis or other diseases [10]. However, persons having SLE, diabetic nephropathy, and chronic renal failure showed higher levels of both plasma and urine adiponectin levels [11,12].

Immunosenescence is a normal biological process that occurs in all organisms involving marked decline in cell functions. These alterations make elderly individuals not only prone to infectious diseases but also malignancy and autoimmunity [16]. Those changes were reported to occur in the immune system during aging affect the onset of autoimmune diseases (ADs). This is due to the fact that aging is related to increased reactivity to self-antigens and loss of tolerance. Elderly people usually experience general systemic inflammation and aggravate degenerative diseases [17], which in turn, increase the risk of developing ADs due to telomere length alteration leading to rheumatic arthritis, scleroderma and SLE [18-23].

Moreover, anti–dsDNA autoantibodies are usually detected before clinical symptoms of SLE show up, [24,25] and are implicated in the pathogenesis of lupus nephritis which is the major cause of morbidity and mortality in SLE [26,27]. Referring to its high specificity, anti–dsDNA autoantibody production is one of SLE eleven classification criteria developed by the American College of Rheumatology (ACR) [28,29]. Men with SLE are more likely to have disability, hypertension, thrombosis, and renal, hematological, and serological manifestations than women. The end organs’ damage and death are also more frequent in men [30]. On the other hand, women with SLE used to
experience malar rash, photosensitivity, oral ulcers, alopecia, Raynaud's phenomenon and arthralgia [31]. Drug-induced lupus erythematosus (DI-LE) can develop clinical and immunopathological symptoms similar to idiopathic lupus in patients with no prior history of the disease [32]. All DI-LE symptoms normally resolved after discontinuation of the offending drug and relapse after re-exposure. The first case of DI-LE was described in 1945 [33] and its estimated incidence is almost 10% out of LE identified cases. Many anti-hypertensive preparations could provoke LE, [34,35] and the tumor necrosis factor-antagonists were additionally reported [36-38].

Conclusion

Depending on the aforementioned literature, the following conclusion could be deduced; the use of SLE-associated markers probably help the provisional identification of individuals when the implication of routine forensic approaches is challenging, but establishing diseases-associated biomarkers’ bank is mandatory.

Acknowledgement

Author would like to express his sincere gratitude and appreciation to Dr. Khalid M. Abdelaziz, Associate Professor and Deputy chairman, scientific and research committee, College of Dentistry, King Khalid University, Abha, KSA for the help provided at the time of writing this article.

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