

Editorial

Babesiosis as an emerging zoonotic disease is caused by infection with hemoparasites of the protozoan genus *Babesia* which are the second most common blood-borne parasites of mammals after the trypanosomes [1]. *Babesia*, the first recognized arthropod-borne pathogen of vertebrates, was discovered originally by the Romanian bacteriologist Victor Babeș as a cause of febrile bovine hemoglobinuria or red water fever at the end of the 19th century [2].

The first case of human babesiosis has been reported in 1957 near Zagreb, Croatia in a splenectomized young farmer and subsequently several *Babesia* species have been documented to involve in human infections in United States, Europe, China, Taiwan, Korea, Japan, India, Egypt, South Africa, Brazil and Mexico [3-10]. Up to now, over 100 species of *Babesia* have been recorded, infecting many mammalian and some avian species making this disease a global health threat [11].

It is well known that hemolytic anemia as a major clinical manifestation of this infection progression can lead to blood supply disruption, tissue hypoxia and eventually cell death [12]. Moreover, erythrocytes adherence to the microvasculature endothelium can also lead to excessive pro-inflammatory cytokine release and intensification of tissue hypoxia [13]. It is noteworthy to mention that blood reperfusion can also result in cell membrane damages and tissue devastation through reactive oxygen species over-generation and neutrophil infiltration [14,15]. Also, it should be borne in mind that activated neutrophils play critical roles in reactive oxygen species, proteases and elastases production resulting in endothelial cell dysfunctions and injuries as well as microvasculopathies [16]. Additionally, hemolysis and hemolytic anemia associated iron overload in tissues can augment oxidative damages, demolish essential macromolecules and exacerbate cytopathies [17].

On the other hand, it is well established that blood flow reduction is associated with testicular germ cell degeneration as well as

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