Commentary Report
Volume 4:1, 2021

Journal of
Anaesthesiology and Pain Research

Open Access

Development in the Treatment of Hemophilia: A Commentary

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Commentary

Hemophilia A and B are X-chromosome-linked bleeding disorders caused by mutations in the factor VIII (FVIII) and factor IX (FIX) genes, respectively, and affect 1 in 5,000 and 1 in 30,000 male live births, respectively. The diagnosis of hemophilia A or B is confirmed by the identification of a low level of the specific factor using laboratory assays. Affected individuals have a lifelong bleeding tendency, caused by the failure of secondary hemostasis, with the degree of severity being proportional to the degree of reduction of the specific coagulation factor [1]. The existence of autoantibodies directed against clotting factors causes acquired hemophilia, an autoimmune illness. While this condition can afflict anyone at any age, it is most common in the elderly. Unprovoked haemorrhage into the skin and muscles, as well as iatrogenic bleeding, is common symptoms. Hemophilia, on the other hand, is a congenital lack of either factor VIII or factor IX that is primarily linked to hemarthroses. Antibodies against factor VIII or factor IX can form in people with hemophilia, however these are alloantibodies triggered by exogenous factor concentrates. Autoantibodies have been found against all of the coagulation factors however factor VIII autoantibodies are the most common. Recent improvements in clarifying the structure-functional links of factor VIII have aided in the identification of 'hot spots' in the molecule that are common autoantibody targets [2]. Hemophilia has been a model of excellent management of a congenital chronic condition since the introduction of modern replacement treatment in the early 1970s. This has been linked to a significant improvement in patient life expectancy, which had been less than 30 years prior. Simultaneously, increased use of prophylaxis in children has resulted in the avoidance or major decrease of the devastating effects of hemophilic arthropathy. The development of effective viral inactivation techniques, as well as methods for screening viruses in blood donations and plasma pools, has greatly improved the safety of plasma-derived products, as evidenced by the fact that no hepatitis or human immunodeficiency virus transmission has occurred in the last 15 years. The emergence of recombinant products, on the other hand, has substantially contributed to the perception of enhanced replacement therapy safety, as well as the growing usage of prophylactic and home treatment. Because of the unknown pathogens and the potential influence of prion disease on the hemophilic population who rely on blood derivatives, there is still a lot of uncertainty [3]. Gene therapy has now proven to be a promising therapeutic technique with long-term advantages for a number of hereditary and acquired disorders, after decades of dedicated research and multiple disappointments. Recently, promising results of hemophilia gene therapy clinical trials were published, revealing maintained factor IX and factor VIII expression, as well as significant reductions in bleeding episodes and factor concentrate intake [4]. As individuals with hemophilia who have been treated with newer, safer, more efficient factor concentrates live longer, their morbidity and healthcare costs rise correspondingly. A reduction in factor utilisation is associated with FIX levels just above 1%. This is consistent with the findings of the Swedish prophylaxis studies, which showed a reduction in hemorrhages when concentrate was dosed to maintain nadir levels of approximately 1% [5]. There have been many advancement in the level of hemophilia treatment and still more study need to be conducted to achieve more efficient method of treatment of hemophilia.

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How to cite this article: Khuong Lahn. "Development in the Treatment of Hemophilia: A Commentary". *J Anesthesiol Pain Res* 4 (2021): 119.

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Received 29 June 2021; **Accepted** 01 July 2021; **Published** 08 July 2021