

Research on hemophilia essay sample



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The word Hemophilia is derived from two Greek words haima, blood, and philein, to love – and, despite being senseless in the description of excess bleeding, it remains in general use. In the older literature various names have been used, e. g. hemorrhaphilia, hematophilia, hemorrhhea, hemorrhagophilia, idiosyncrasia haemorrhagica and morbus haematicus. This first use of the word ‘ hemophilia’ is attributed to Schonlein, who gave a dissertation entitled Die Hamophiliet at Wurzburg in 1828.

The disease is now called haemophilia A or classic haemophilia and is due to deficient activity of antihemophilic factor (AHF, antihemophilic globulin (AHG) or factor VIII. A variety has been described which is called Christmas disease in the UK but in Europe and the USA is often called haemophilia B or plasma thromboplastin component (PTC) deficiency. This is due to deficient activity of Christmas factor (CF, PTC or factor IX).

The clinical manifestations of these two diseases are essentially similar and no differentiation is made between them with regard to bleeding tendency. It is probable that genetically determined bleeding disorders are as old as time but they seem to have been neglected by the Egyptian, Romans and Greek physicians. The earliest documented case of a familial bleeding disorder occurs in the fourth-century Talmud and in Rabbinic writings thereafter.

Perhaps the most famous hemophilic family internationally is that of Queen Victoria, who in 1853 gave birth to her eighth child, Leopold, Duke of Albany, and he was a haemophiliac. In addition to this, two of her daughters proved to be carriers. It can truly be said that the result of the transmission of haemophilia into the royal families of Europe had a profound effect on the

course of European history. Nowhere is this more poignant than in the case of Alexandra, who became the consort of Tsar Nicholas the Second of Russia, who produced Alexis, a severely affected haemophiliac, in 1904. In Victoria's family there was no evidence of any ancestor with hemophilia and it must be presumed that the disease was due to mutant gene.

Aims

The proponent aims to:

1. compare and contrast the differences of hemophilia A and hemophilia B.
2. explain how hemophilia is diagnosed.
3. explain the complications that may arise if hemophilia is not diagnosed early.

Research Questions

The proponent endeavors to provide answers to the following:

1. What are differences between hemophilia A and hemophilia B?
2. How is hemophilia diagnosed?
3. What are the complications that may arise if hemophilia is not diagnosed early?

Chapter 2

METHOD

Research Method

A comparison and contrast pattern arranges information according to how two or more things are similar to or different from one another (or both). This

is an effective pattern to use when the reader can better understand one subject when it is described in relation to another. If the reader is familiar with one topic, the writer can compare or contrast it with another topic to shed insight on it.

The proponent will be using compare and contrast for the topic “ Hemophilia” by comparing “ Hemophilia A” and “ Hemophilia B”. This principle of organization will present the similarities and differences between these two types of hemophilia which are clinically almost identical. The proponent thinks that Comparison and Contrast would be an effective way for developing this paper about the topic “ Hemophilia”.

Due to Due to

Factor VIII only affect men Factor IX

Deficiency Deficiency have a genetic mutation on the X chromosome 1 in 5, 000 1 in 30, 000

Males affects all people of all Males races and ethnics

Both types of hemophilia is caused by the lack of clotting factor in the blood which makes them bleed internally, mainly into muscles and joints Both types of hemophilia is caused by the lack of clotting factor in the blood which makes them bleed internally, mainly into muscles and joints globally

Explanation

Hemophilia is a rare disease wherein it slows down the process of blood clotting. If you have hemophilia, you may bleed for a longer time than others after an injury. It has its two major forms – Hemophilia A and Hemophilia B. Both have several similarities and differences as well as its various

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characteristics as of some of which seen in the framework above. Both are diagnosed in the same way except only that they have different factors which they lack in the blood in order for it to have a normal blood clotting process. This disease is already known and does have remedies and diagnoses. But one thing I can say, we do not yet what effects does this kind of disease bring about if a person with hemophilia is not diagnosed at an early time as this disease is very critical for it does not have any permanent or stable treatment at this moment in time with the given present level of technology. We will be able to know it as this paper is enriched with further researches and studies with the help of the principle of organization – comparison and contrast.

Chapter 3

RELATED READINGS

Hemophilia Defined

The word Hemophilia is derived from two Greek words haima, blood, and philein, to love – and, despite being senseless in the description of excess bleeding, it remains in general use. Hemophilia is the term given to a bleeding disorder wherein the person experiences bleeding spontaneously or following after an injury (Forbes, 1997, 3).

Nature of Hemophilia

Hemophilia is the oldest known hereditary bleeding disorder. There are two types of hemophilia, A and B. Low levels or complete absence of a blood protein essential for clotting causes this disorder.

Patients with hemophilia A lack the blood clotting protein, factor VIII, and those with hemophilia B lack factor IX. Approximately 85% have hemophilia A and the remainder has hemophilia B. The severity of hemophilia is related to the amount of the clotting factor in the blood. About 70% of hemophilia patients have less than one percent of the normal amount and, thus, have severe hemophilia. A small increase in the blood level of the clotting factor, up to five percent of normal, results in mild hemophilia with rare bleeding except after injuries or surgery (Kemball-Cook, 1997, 21).

How Hemophilia is inherited

Hemophilia types A and B are inherited diseases passed on from a gene located on the X chromosome; therefore it is a sex-linked disease. About 70% of all individuals with hemophilia A or B inherited the disease. The other 30% have hemophilia because of a spontaneous genetic mutation. Females have two X chromosomes, while males have one X and one Y chromosome. A female carrier of hemophilia has the hemophilia gene on one of her X chromosomes, and there is a 50 percent chance that she may pass the defective gene to her male offspring.

Males who inherit the defective gene will develop hemophilia. Males with hemophilia do not pass the gene to their sons; however, they do pass the gene to their daughters.

Females who inherit the defective gene will become carriers who may, in turn, have a 50 percent chance of passing it on to their children. Although females who inherit the gene generally have no active problems related to hemophilia, some may have other problems associated with bleeding, such

as excessive menstrual bleeding, frequent or severe nosebleeds, or bleeding after dental procedures or surgery. In about one-third of hemophilia cases, there is no family history of the disease. These cases are due to a new or spontaneous development of the defective gene in the female.

In the rare event of a hemophiliac father and a carrier mother, the right combination of parental chromosomes will result in a hemophiliac female child. This situation, however, is extraordinarily rare. The vast majority of individuals with either hemophilia A or B, then, are male.

As mentioned earlier, about 30% of all individuals with hemophilia A or B are the first members of their families to ever present with the disease. These individuals had the unfortunate occurrence of a spontaneous genetic mutation, meaning that early in development, some random genetic accident befell their X chromosome, resulting in the defect causing hemophilia A or B (Yhe, 1997, 229).

Why Hemophiliacs Bleed

The ability to stem blood loss after vascular injury is vital for all vertebrate species. Essentially simultaneously, escape of blood through the gap created by minimal vascular damage is normally staunched by platelets, a nuclear cells that arise from the break-up of the of the cytoplasm of megakaryocytes, a process stimulated by a trace plasma polypeptide, thrombopoeitin, that promotes both the proliferation and differentiation of megakaryocyte progenitor cells. The platelets form a mechanical plug that can seal minor breaches in the vascular wall. Platelets do this by sticking to the edges of severed vessels, to exposed sub endothelial structures such as collagen, and <https://assignbuster.com/research-on-hemophilia-essay-sample/>

to each other. Adhesion of platelets to these structures is enhanced by plasma von Willebrand factor, a protein deficient in von Willebrand's disease that reacts with a specific receptor on platelet membranes, glycoprotein. Contact of platelets with the collagen induces activation of these cells which release biologically active agents from their cytoplasmic granules that enhance the formation of platelet aggregates.

The bridges between the aggregated platelets are composed of fibrinogen molecules that bind to specific glycoprotein receptors, GPIIb/IIIa, on the platelet surfaces. The adherent and aggregated platelets clump rapidly, forming a hemostatic plug that can close small gaps in the walls of blood vessels. Platelet aggregation is brought about by a cyclic endoperoxide, thromboxane A₂, a derivative of arachidonic acid that is released from the platelet membrane phospholipids when these cells have been stimulated by collagen, adenosine diphosphate (ADP) or other agonists. Essentially simultaneously, gaps in the vascular wall are closed by generation of a blood clot, that is, by transformation of liquid blood to a gel-like coagulum, which consolidates the platelet aggregates. Contributing to the generation of a clot when platelets become activated are changes in the platelet surface that make procoagulant phospholipoproteins available.

Coagulation of blood at the site of injury can diminish or halt blood loss from vascular defects larger than those that can be controlled by platelets alone. When bleeding occurs into a closed space such as the tip of a finger or toe, blood loss is minimized by the back-pressure of extravasated blood; the importance of this device can be appreciated by the extensive loss of blood that may occur in otherwise normal individuals after injury to periorbital

tissues or to an intraperitoneal vessel. In these situations, the accumulated shed blood usually does not provide sufficient back-pressure to stop blood flow. Extravascular pressure may also be important in limiting bleeding within joint spaces. In the special case of the uterus, bleeding after delivery is further controlled by contraction of the myometrium, applying external pressure on blood vessels at the site of the avulsed placenta. A similar mechanism may participate in the control of menstrual bleeding.

The importance of blood coagulation for the control of bleeding is readily understood from the hemorrhagic tendency of individuals with hereditary clotting defects. Clotting is the end-result of a series of chemical events that are initiated upon contact of blood with either injured tissue or with negatively charged surfaces such as glass. Perturbation of blood in these ways starts sequential reaction that result, in the end, in the release of a plasma protease, thrombin that brings about coagulation. At each step, a plasma protein clotting factor undergoes limited proteolysis that results in its conversion to a form with enzymatic activity until, in the end; prothrombin is cleaved proteolytically, releasing enzymatically active thrombin. A characteristic of these clotting factors is that they may have multiple functions, suggesting that their role has been determined by the forces of evolution (Ratnoff, 1997, 7). Factor VIII

The hemostatic system, consisting of the blood vessels and their content, blood, plays a crucial role in human survival. The importance of the plasma coagulation system in protecting life by preventing further blood loss following transection of a blood vessel is well recognized. Blood is usually maintained in a fluid state, without evidence of bleeding or clotting. The <https://assignbuster.com/research-on-hemophilia-essay-sample/>

presence of an X-linked pattern of inheritance of a bleeding diathesis in families, referred to as hemophilia, has been recognized for hundreds of years.

That hemophilia is due to a deficiency of a factor (F) in the blood was proven in 1840 by correction of the bleeding defect with transfusion of whole blood; this was followed in 1911 by the demonstration that normal plasma could shorten the whole blood clotting time of hemophilic blood. Then, in 1937, a factor from normal plasma was shown to be effective in accelerating the coagulation of hemophilic blood, and the term antihemophilic globulin was coined; this protein is now referred to as factor VIII-C (FVIII-C).

Further progress was achieved in the 1950s with the development of cryoprecipitate and plasma concentrates to treat hemophilia A (FVIII deficiency). The clinical and therapeutic observation that clotting time was corrected after transfusion of blood from one hemophilic patient to another was followed by the description of “ plasma thromboplastin component” or factor IX deficiency. This second type of deficiency was referred to as hemophilia B to differentiate it from hemophilia A.

Clarification of the structure and function of the factor VIII molecule (FVIII-C, an X-linked gene product, also known as antihemophilic globulin) noncovalently bound to von Willebrand factor (vWF, an autosomal 12p gene product) in plasma clarified the separate roles of factor VIII-C (antihemophilic globulin) and von Willebrand factor proteins. This led to an understanding of the role of the different components of the factor VIII molecule in the physiology of normal hemostasis and to a recognition that hemophilia A and

von Willebrand disease were caused by a deficiency of different proteins in the factor VIII complex.

An understanding of the reasons for the development of factor VIII inhibitors in persons with hemophilia or in persons with previously normal hemostasis (referred to as acquired hemophilia) expanded understanding of the antigenic structure of the factor VIII molecule. Cloning of the factor VIII gene was followed by the preparation of recombinantly derived factor VIII (rFVIII) as replacement therapy for the missing factor. Several different vectors have now been used to correct factor VIII deficiency in humans, with many questions still to be resolved. The potential role of increased levels of factor VIII in thrombophilic states continues to be explored.

Primary immunodeficiency diseases (PIDs) are associated with various autoimmune complications and several manifestations of autoimmunity. Acquired hemophilia is rare in childhood even though autoantibodies may develop in various forms of primary immunodeficiency diseases. However, acquired hemophilia may rarely form factor VIII inhibitors in patients with undefined primary immunodeficiency disease features that are suggestive of autosomal recessive hyper-immunoglobulin (Ig) E syndrome (Berntorp, 1997, 181).

Factor IX

The most significant breakthroughs in comprehending the mechanisms associated with coagulation first came from an understanding of the individual causes of the bleeding disorders. The recognition in 1952 that hemophilia B was due to a deficiency of a coagulation factor followed the <https://assignbuster.com/research-on-hemophilia-essay-sample/>

discovery that hemophilia A was caused by the deficiency of another clotting factor. Also termed Christmas disease, hemophilia B is an X-linked inherited bleeding disorder, usually manifested in males and transmitted by females when they carry the abnormality on the X chromosome. Hemophilia B is caused by a deficiency or dysfunction of factor IX (FIX) resulting from a variety of defects in the FIX gene. FIX deficiency is 4-6 times less prevalent than factor VIII (FVIII) deficiency. The newspaper item below demonstrates what appears to be a late 19th-century record of hemophilia passed from mother to sons.

Mutations in human coagulation factor IX that cause hemophilia B may be classified as severe, moderately severe and mild based on the plasma levels of factor IX among affected individuals (< 1%, 2-5%, 6-30%, respectively). Recently, hemophilia B was shown to be a disease with mutations showing clinical variation (Lusher, 1997, 203).

Diagnosis of Hemophilia A and B Carriers

Hemophilia A and B are X-linked diseases where precise carrier detection and prenatal diagnosis are increasingly an integral part of care of the patient and family. Carrier assessment can be made in several ways:

1. Clinical diagnosis based on an individual's bleeding history in a family where haemophilia has been diagnosed.
2. Diagnosis within a family with haemophilia based upon a detailed family tree where no uncertainties (e. g. questions of paternity) are present.
3. Diagnosis based on reduced levels of plasma factor VIII (FVIII) or factor IX (FIX).
4. Genetic diagnosis by analysis of

the FVIII or FIX genes within families either by polymorphism-based gene tracking (linkage analysis) or mutation detection (Goodeve, 1997, 63).

Clinical and phenotypic diagnosis of carrier status

Female carriers of haemophilia A or B have generally inherited their abnormal FVIII or FIX gene from one of their parents, together with a normal gene. Thus, on average, carriers of severe disease, where affected males have very low or undetectable levels of FVIII or FIX, will have a plasma levels around 50% of normal. Carriers of milder conditions will tend to have levels greater than 50%, since the defective gene will be able to produce some clotting factor.

Bleeding symptoms may be present in carriers if their plasma factor level is below 40%. This generally occurs as a result of extreme lyonization, although other rare possibilities, including homozygosity, Turner's syndrome and co-inheritance of von Willebrand's disease should be considered.

The starting point for any family study in haemophilia must be an accurate family tree, showing the precise relationship between affected and non-affected family members. The nature of the inherited condition (haemophilia A or B) must be certain and the ethnic background of the family may also be important.

With good family data it may be immediately possible to exclude or include carrier status in certain females. Thus, carriership can be excluded for a female if haemophilia only occurs in her paternal family and her father has haemophilia or she has more than one hemophilic son or she has one

affected son and her family includes a well-documented haemophiliac on her maternal side.

The remaining females within a hemophilic family should be considered to be possible carriers, particularly on families where the individual concerned has one hemophilic son and no family history of the disease (isolated cases). The assignment to a possible carrier of a probability of carriership is the essence of carrier detection and is only absolute when the precise genetic defect within the FVIII or FIX gene has been identified within her family and herself. Conversely, the absence of the mutation confers to the status of non-carrier. However, the procedures necessary for such diagnoses are unavailable to the majority of the world's hemophilic families at present and phenotypic methods are still widely used in carrier analysis.

The assessment of carrier status based on a family tree is calculated using anterior and descendent pedigree information to calculate an overall probability or odds of carriership of 1.0 (obligate carrier), and her daughters a probability of 0.5. Her granddaughters will have a probability of 0.25 (0.5×0.5) (Goodeve, 1997, 64).

Complications that may arise from the treatment

- Deep internal bleeding – Hemophilia may cause deep muscle bleeding that leads to swelling of a limb. The swelling may press on nerves and lead to numbness or pain. This may result in a reluctance to use that limb.
- Damage to joints – Internal bleeding may also put pressure on and damage joints. Pain sometimes may be severe, and you may be reluctant to use a

limb or move a joint. If bleeding occurs frequently and you don't receive adequate treatment, the irritation may lead to destruction of the joint or the development of arthritis.

- Infection – People with hemophilia are more likely to receive blood transfusions and are at greater risk of receiving contaminated blood products. Until the mid-1980s, it was more likely for people with hemophilia to become infected with the human immunodeficiency virus (HIV) or with hepatitis through contaminated blood products. Since then, blood products are much safer because of steps taken to screen the supply of donated blood. The risk of infection through blood products also has decreased substantially since the introduction of genetically engineered clotting products called recombinant factors, which are free of infection. However, it's still possible for people who rely on blood products to contract diseases. If you have hemophilia, consider receiving immunization against hepatitis A and B.

- Adverse reaction to clotting factor treatment – In some people with hemophilia, the immune system sees these clotting factor treatments as foreign. When this happens, the immune system develops proteins that inactivate the clotting factors used to treat bleeding. Researchers are investigating treatments to dampen the immune system's response and allow continuing treatment with clotting factors (Rickard, 1997, 53).

Chapter 4

PROPOSITIONS

Proposition 1: Hemophilia is the oldest known hereditary bleeding disorder.

The earliest documented case of hemophilia occurs in the fourth century Talmud and in Rabbanic writings thereafter. This bleeding disorder is as old as time but they seem to have been neglected by the Egyptian, Roman, and Greek physicians (Kemball-Cook, 1997, 21).

Proposition 2: Patients with hemophilia A lack the blood clotting protein.

Factor VIII.

Hemophilia A is the most common type of hemophilia. It is also known as factor VIII deficiency or classic hemophilia. Factor VIII is an essential blood clotting factor also known as anti-hemophilic factor (AHF). In humans, Factor VIII is encoded by the F8 gene. Defects in this gene result in hemophilia A, a well-known recessive X-linked coagulation disorder (Kemball-Cook, 1997, 21).

Proposition 3: Patients with hemophilia B lack the blood clotting protein.

Factor IX.

Hemophilia B is the second most common type of hemophilia. It can also be known as factor IX deficiency, or Christmas disease. Factor IX is produced as a zymogen, an inactive precursor. It is processed to remove the signal peptide, glycosylated and then cleaved by factor XIa (of the contact pathway) or factor VIIa (of the tissue factor pathway) to produce a two-chain form where the chains are linked by a disulphide bridge (Kemball-Cook, 1997, 21).

Proposition 4: The severity of hemophilia is related to the amount of the clotting factor in the blood. About 70% of hemophilia patients have less than

one percent of the normal amount, and, thus, have severe hemophilia. A small increase in the blood level of the clotting factor, up to five percent of normal, results in mild hemophilia with rare bleeding except after injuries or surgery (Yhe, 1997, 229).

Proposition 5: Hemophilia is a sex-linked disease.

Factor VIII and factor IX are located on the long arm of the X chromosome. Females have two X chromosomes, while males have one X and one Y chromosome. A female carrier of hemophilia has the hemophilia gene on one of her X chromosomes, and there is a 50 percent chance that she may pass the defective gene to her male offspring (Yhe, 1997, 229).

Proposition 6: Males who inherit the defective gene will develop hemophilia. Males have one X chromosome and one Y chromosome. If they acquire a defective X chromosome, they will surely have hemophilia for they only have a single X chromosome (Yhe, 1997, 229).

Proposition 7: Females who inherit the defective gene will become carriers who may, in turn, have a 50 percent chance of passing it on to their children. Females have two X chromosomes. If they acquire a defective X chromosome, they will still not have the disease for they have another X chromosome that will prevent them from having the disease. Thus, there will only be a probability that they will pass this defective gene on to her male offspring (Yhe, 1997, 229).

Proposition 8: The ability to stem blood loss after vascular injury is vital for all vertebrate species.

Blood clotting is very important to all vertebrate species for it stops the

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blood from coming out of our body. If will not be able to clot blood, it will continuously bleed out until we run out of it and die (Ratnoff, 1997, 7).

Proposition 9: When bleeding occurs in a closed space such as the tip of a finger or toe, blood loss is less. This is such because of the back-pressure of extravastated blood which pushes the blood back at the same time while it is bleeding out (Ratnoff, 1997, 7).

Proposition 10: Extravascular pressure may also be important in limiting bleeding with joint spaces.

In the special case of the uterus, bleeding after delivery is further controlled by contraction of the myometrium, applying external pressure on blood vessels at the site of the avulsed placenta. A similar mechanism may participate in the control of menstrual bleeding (Ratnoff, 1997, 7).

Proposition 11: The hemostatic system, consisting of the blood vessels and the blood, plays a crucial role in human survival.

Blood is a body fluid that delivers essential substances like nutrients and oxygen to the cells of the body. Blood also transports carbon dioxide and other waste products away from the cells, to the lungs, kidneys, and digestive system; from there they are removed from the body. Our body has billions of cells that need regular supply of fuel and oxygen to function. Blood meets these requirements and ensures proper functioning of cells, thus, also making sure that our body keeps in good health (Berntorp, 1997, 181).

Proposition 12: Factor IX deficiency is 4-6 times less prevalent than factor VIII deficiency.

1 in 5, 000 males worldwide are born with hemophilia A, while 1 in 30, 000 males worldwide are born with hemophilia B (Lusher, 1997, 203).

Proposition 13: Hemophilia carrier assessment can be made in several ways. There are number of ways in order to know or evaluate whether someone is a possible carrier of hemophilia or not. One is based upon the detailed family tree where no uncertainties are present (Goodeve, 1997, 63).

Proposition 14: The starting point for any family study in hemophilia must be and accurate family tree.

An accurate family tree is needed in showing the precise relationship between affected and non-affected family members. The nature of the inherited condition must be certain and ethnic background of the family may also be important (Goodeve, 1997, 64).

Proposition 15: Hemophilia may cause deep muscle bleeding that leads to swelling of a limb.

The swelling may press on nerves and lead to numbness or pain. This may result in a reluctance to use that limb (Rickard, 1997, 53).

Proposition 16: Patients with hemophilia who undergo liver transplantation are cured of their hemophilia.

This is because factor VIII and factor IX are synthesized in the liver. FIX is produced by hepatocytes. The exact site of synthesis of FVIII in the liver is unknown (Rickard, 1997, 53).

Proposition 17: Patients with hemophilia should not be deprived of engaging in sports.

Those with hemophilia are still recommended to engage in sports and various physical activities in order to have a healthy lifestyle, however, they are advised to avoid sports that may cause them to bleed (Rickard, 1997, 55).

Proposition 18: People with hemophilia are at greater risks of receiving contaminated blood products.

Until the mid-1980s, it was more likely for people with hemophilia to become infected with the human immunodeficiency virus (HIV) or hepatitis through contaminated blood products (Rickard, 1997, 56).

Proposition 19: People with or without hemophilia just have the same life expectancy.

Hemophilia doesn't affect one's life span as long as it is diagnosed and treated as early as possible. One's lifespan depends on his lifestyle and way of living (Rickard, 1997, 58).

Chapter 5

CONCLUSION

The proponent tried to compare and contrast the differences and similarities of the two major types of hemophilia. The proponent concludes that there is really no permanent treatment to hemophilia; however, patients undergoing liver transplant are cured of their disease. Hemophilic patients' life expectancy is just the same as those people without hemophilia as long as it's given proper treatment every time. Hemophilic patients should not be deprived of being engaged in sports, but are recommended take sports with less physical contact and refrain from bleeding.

There are certain facts that the two types of hemophilia are different in some of its aspects. First, the reason why patients with hemophilia A and hemophilia B bleeds is because they lack protein factors in their blood. The former lacks in factor VIII while the latter lacks in factor IX. Second, factor IX deficiency is 4-6 times less prevalent than factor VIII deficiency for there is only 1 person with hemophilia A in every 5, 000 males; on the other hand, there is 1 person with hemophilia B in every 30, 000 males.

However, the two main types of the disease also have their similarities as it is almost clinically identical in nature. Both types of hemophilia only affect men because this is a sex-linked disease and is found in the X chromosome. And men have an XY chromosome, so if they acquired a defected X chromosome, they will surely acquire the disease. Unlike on the case of the women, they have an XX chromosome, so if they acquired a defected X chromosome, they still have another X chromosome which can protect them from having the disease. This disease affects all people of all races and ethnics globally.

For further study, I recommend the readers to continually research for the disease hemophilia specifically their treatment and diagnoses. At the present time with the given present level of technology, there is still no permanent treatment for this disease. What we have are just merely “ remedies” which cures the disease only for a certain period of time but it doesn’t really cure it permanently.

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