

Complete Information on Autoimmune hemolytic anemia with Treatment and Prevention

Autoimmune hemolytic anemia is a serious, life threatening disease. About half of the time, the cause of autoimmune hemolytic anemia cannot be determined. There are two main types of autoimmune hemolytic anemia: warm antibody hemolytic anemia and cold antibody hemolytic anemia. Autoimmune hemolytic anemia can also be caused by or occur with another disease, such as systemic lupus erythematosus, and rarely it follows the use of certain drugs, such as penicillin. In the warm antibody type, the autoantibodies attach to and destroy red blood cells at temperatures equal to or in excess of normal body temperature. In the cold antibody type, the autoantibodies become most active and attack red blood cells only at temperatures well below normal body temperature. Autoimmune hemolytic anemia is more likely to occur in middle-aged and older individuals.

Autoimmune hemolytic anemia is a rare group of disorders that can happen at any age. These disorders impact women more frequently than men. Destruction of crimson blood cells by autoantibodies may happen abruptly, or it may produce gradually. In some people, the destruction may stop after a period of time, whereas in other people, it persists and becomes chronic. When the cause of autoimmune hemolytic anemia is another disease, symptoms of the underlying disease, such as swollen and tender lymph nodes and fever, may dominate. Autoimmune hemolytic anemia can also be induced by several drugs including methyl-dopa and fluarabine. In people with hemolytic anemia, red blood cells have an abnormally short life span. Hemolytic antibodies also may be induced by incompatible blood transfusions and be encountered in isoimmune anemias of newborn piglets, puppies, or foals.

Some folk with autoimmune hemolytic anemia may get no symptoms, particularly when the devastation of crimson blood cells is balmy and develops gradually. Autoimmune hemolytic anemia is characterized by a malfunction of the exempt structure that produces autoantibodies, which assault crimson blood cells as if they were substances international to the system. Others have symptoms similar to those that occur with other types of anemia, especially when the destruction is more severe or rapid. When severe or rapid destruction of red blood cells occurs, mild jaundice may also develop. Penicillin, quinine, quinidine, L-dopa, and other agents may cause immune hemolysis. A patient who needs a transfusion but does not show evidence of blood loss or bone marrow suppression may have hemolytic anemia. The onset of the disease may be quite rapid and very serious.

If symptoms are balmy or if devastation of crimson blood cells seems to be slowing on its own, no handling is needed. If crimson blood cubicle devastation is worsening, a corticosteroid drug such as prednisone is normally the best selection for handling. If prednisone does not improve the condition, a splenectomy may be considered. High doses are used at first, followed by a gradual tapering of the dose over many weeks or months. When destruction of red blood cells persists after removal of the spleen or when surgery cannot be performed, immunosuppressive drugs, such as cyclophosphamide or azathioprine, are used. Plasmapheresis, which involves filtering blood to remove antibodies, is occasionally helpful when other treatments fail. Blood transfusions are given with caution, if indicated for severe anemia, because of the potential that blood may not be compatible and may bring on a reaction.

About the Author

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