Publication of the Month

Phadia
Setting the Standard

July 07/11: IgG anti-IgA – a screening tool for hypogammaglobulinemic patients

Key messages:

- *Anti-IgA antibodies can cause severe anaphylactoid reactions in the case of blood transfusion or substitution immunoglobulin therapy in patients with hypogammaglobulinemia.*
- *Anti-IgA antibodies occur in 10% of patients with IgA deficiency.*
- *Quantitative detection of anti-IgA antibodies and measurement of serum IgA concentration can prevent anaphylactoid reactions after blood transfusion or gammaglobulin treatment.*

Thon V.
Screening of IgG anti-IgA antibodies in hypogammaglobulinemic subjects to prevent anaphylactoid reactions
Clinical Laboratory International, 12.07.2010, Online article (link to the article)

Background: Patients with selective IgA deficiency (IgAD) and anti-IgA antibodies have a higher risk of severe anaphylactoid reaction after blood transfusion or substitution therapy with gammaglobulins containing IgA. The prevalence of primary immunodeficiencies studied in the Czech Republic is 5.8/100 000 inhabitants. Similar results were reported from a more recent European ESID database of primary immunodeficiencies.
Selective IgA deficiency is the most common primary immunodeficiency (about 75%) and is characterised by low serum levels of IgA. Some individuals with IgA deficiency may be clinically healthy, while others are susceptible to respiratory and gastrointestinal infections, allergy, autoimmune diseases and malignancy.

Summary: Thon V et al. systematically studied the prevalence of anti-IgA antibodies and related anaphylactoid reactions following immunoglobulin infusions in patients with common variable immunodeficiency from Germany and Czech Republic. They found that 10% of hypogammaglobulinemic patients were positive for anti-IgA antibodies. Anti-IgA antibodies occur only in patients with undetectable IgA serum level (IgA lower than 0.05 g/L).

A positive finding of anti-IgA antibodies enables better recognition and management of true cases of anti-IgA anaphylactoid reactions and enables assessment of the risk for the blood transfusion services. Anti-IgA mediated anaphylactoid reaction can be prevented by auto transfusion (if this procedure is possible) or by using blood from donors completely lacking IgA, or by thoroughly washing the blood cells to remove donor's IgA.

Screening for anti-IgA antibodies in patients lacking IgA may be helpful for determining the best therapeutic strategy, including the route of administration of substitution immunoglobulin therapy or the transfusion of IgA-free blood products. The combined use of quantitative detection of anti-IgA antibodies and the measurement of serum IgA concentration provides an effective and safe strategy for the diagnosis and prevention of IgA anaphylactic transfusion reactions.

Comment: This publication shows that anti-IgA antibodies are often present in selective IgA deficiency implying a high risk for an anaphylactoid reaction due to substitution immunoglobulin therapy. Therefore the evaluation of anti-IgA antibodies should be mandatory prior to therapy initiation.