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MICROBIOLOGICAL INDUCTION OF ACANTHOCYTOSIS

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Abstract

Acantocytosis is associated with various diseases among which are a β-lipoproteinemia, a genetic disease, following splenectomy, following exposure to toxic chemical, and massive carcinoid tumor. No previous studies have, up to the best knowledge of the author, reported acanthocytosis to be induced by pathogens.

The aim of the present study was to explore the potential of β-hemolytic Streptococcus group A to induce acanthocytosis.

The methodology of the present study involved incubation of β-hemolytic Streptococcus group A with blood samples, examining blood films of patients diagnosed with β-hemolytic Streptococcus group A and examining urine samples showing acanthocytosis for β-hemolytic Streptococcus group A.

Study findings confirmed that acanthocytosis was induced by β-hemolytic Streptococcus group A, and patients diagnosed with β-hemolytic Streptococcus group A showed acanthocytosis in their blood smears. Urine samples showing acnathocytes in microscopic examination were positive for β-hemolytic Streptococcus group A.

Taken together, Acanthocytosis is a diagnostic feature of β-lipoproteinemia, a genetic disease. Our data pointed to a new causative agent in which β-hemolytic Streptococcus group A induces acanthocytosis with high potential to play a new etiological agent in pathogenesis of diseases.

Our data are preliminary findings that need to be further investigations since pathogens seem to have non-classical roles and new pathological studies are required to explore such roles.

Keywords: Acanthocytosis, β-lipoproteinemia, β-hemolytic Streptococcus group A

Introduction

The acanthocyte has been described as a red blood cell that is being an irregularly speculated with normal volume. It has a sphere shape with 3-12 (up to 20) finger-like projections that are irregularly distributed on its surface. These projections exhibit variations in length and width and typically have blunt or clubbed tips (Bessis, 1977).

From a historical point of view, in 1950, Bassen and Komzweig were the first to describe acanthocyte in humans as abnormal erythrocytes, crenated cells with bizarre shapes. The researchers found the abnormal consistently in fresh and dried preparations of blood from a young woman whose parents were first cousins. The patient was diagnosed with an atypical retinitis pigmentosa associated with unusual neurological signs. Te researchers were not sure about the etiology, but when the blood was re-examined a year later, the abnormal red blood cells were still present in the same numbers (Bassen and Komzweig 1950).

Two years later, another case with acanthocytosis was described by Singer et al (1952) in a young boy from a consanguineous marriage that presented with progressive neurological signs.

According to Salt et al (1960), the name "acanthrocyte" (Greek akantha = thorn) was given for this abnormal cell which was considered the characteristic feature of the new hereditary syndrome. The name acanthrocyte was modified to acanthocyte.

Experimental studies by Singer et al (1952) demonstrated that the acanthocytes are characterized by having an increased osmotic resistance in hypotonic solution but an increased mechanical fragility when rotated with glass beads. In a further study, Salt et al (1960) found that acanthocytosis originated from an inborn error of lipoprotein metabolism resulting in very low levels of β-lipoprotein in affected patients.

This condition was called abetalipoproteinemia. It has been demonstrated that red blood cells with defective membranes to have mildly increased cholesterol: phospholipid ratio with l5-20% increase in the cholesterol content (McBride and Jacob 1970).

In a study by Smith and Lonergan (1964), it was demonstrated that in acquired acanthocytosis, when washed acanthocytic cells were placed in normal serum, they returned back to a normal discoid conformation. Furthermore, when normal homologous red blood cells were placed in patient plasma, they acquired the speculated shape typical of the patient's altered red blood cells. This experiment suggested that a factor in the plasma was responsible for the change.

It has been postulated that increased serum cholesterol in acquired acanthocytosis to be resulted from a deficiency of lecithyl cholesterol acyl tramferase (LCAT), the enzyme working in esterification of free cholesterol

(Smith and Lonergan, 1964). Another study showed that the deficiency of LCAT leads to an increase in plasma free cholesterol and the phospholipid lecithin. Because the cholesterol and phospholipid in the red cell membrane have dynamic equilibrium with the plasma, the cell membrane takes on extra lecithin and cholesterol, which works to increases the surface area of the membrane and causes it to become folded and ruffled and to take on the appearance of the acanthocyte (Cooper et al., 1975).

It is worth to mention that the increased membrane lipid leads to increased osmotic resistance and increased membrane rigidity as indicated by reduced filterability which participates in mechanical fragility, fragmentation and extravascular hemolysis because the spleen removes damaged red blood cells (Cooper 1969). In his study, Silber et al (1966) showed that even hemolysis can be reduced by splenectomy but the acanthocytes remain.

Other studies have described the occurrence of acquired acanthocytes to be associated with massive hepatic metastasis of a rectal carcinoid (Keller et al., 1971). In another study, acanthocytes have also been described in people following splenectomy (Brecher et al., 1973). In another study, Frederique et al (2010) reported the occurrence of thrombocytopenia and severe hemolytic anemia with acanthocytosis in rats at SCH 900875 doses of 75, 100, and 150 mg/kg/day.

Wandel (1996) showed that acanthocyte is associated significantly with glomerular disease and acanthocytes prove to be the most characteristic red cell type for glomerular haematuria.

Up to the best knowledge of researcher, no studies have identified direct relationship between the infection of β-hemolytic Streptococcus group A and acanthocytosis.

Materials and Methods

This section involved obtaining purified isolations of β-hemolytic Streptococcus group A, which then diluted and incubated in broth medium for 24 hrs to be ready for use. Blood samples were obtained from hematology laboratory. We took a drop of broth medium that contains β-hemolytic Streptococcus group A and incubated with a drop of blood and spread on a microscopic slide to investigate for acanthocytosis. Some slides were examined immediately for acanthocytosis, while other slides were dried and stained using Wright and Giemsa stain to examine the red cell morphology.

The other part of methodology involved taking blood samples from patients diagnosed with β-hemolytic Streptococcus group A to examine blood films to confirm the occurrence of acanthocytosis.

The third part involved examining if urine samples exhibiting acanthocytes in microscopic examination would have positive cultures for β-hemolytic Streptococcus group A.

Results

The results of the present study showed that acanthocytosis was induced in 100% of all cases in which β-hemolytic Streptococcus group A was incubated with blood samples. Stained blood films also showed acanthocytosis.

When blood films from patients diagnosed with β-hemolytic Streptococcus group A were examined microscopically, acanthocytosis was revealed. 10-15% of red blood cells showed the appearance of acanthocytosis.

Finally, urine samples with acanthocytosis gave positive cultures for β-hemolytic Streptococcus group A.

Discussion

In the present study, we reported our experience in induction of acanthocytosis by unusual way. Acanthocytosis has been associated since its first time of reporting by genetic diseases in which alterations in membrane lipids (Bassen and Komzweig, 1950; Singer et al., 1952; Bessis, 1977).

Other studies reported the occurrence of acanthocytosis in other diseases including massive hepatic metastasis of a rectal carcinoid (Keller et al., 1971), following splenectomy (Brecher et al., 1973), glomerular disease (Wandel, 1996), and following the exposure to a toxic chemical (Frederique et al., 2010).

The etiology of acanthocytosis is not well clear but rotates on the axis of problems in lipid metabolism in membranes of red blood cells (Smith and Lonergan, 1964; Cooper et al., 1975).

The importance of the present findings come from several considerations among which are the infectious agent should be considered upon management of cases involving acanthocytosis including a β-lipoproteinemia; we have also to consider acanthocytosis when examining urine microscopically. Another significant point, but needs further investigations, is the ability of β-hemolytic Streptococcus group A to affect the membranes of other cells playing a new etiological role in pathogenesis.

Conclusions

Acanthocytosis is a diagnostic feature of β-lipoproteinemia, a genetic disease. Our data pointed to a new causative agent in which β-hemolytic Streptococcus group A induces acanthocytosis with high potential to play a new etiological agent in pathogenesis of diseases.

Recommendations

Our data are preliminary findings that need to be further investigations since pathogens seem to have non-classical roles and new pathological studies are required to explore such roles.

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