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Review Article

Inclusions bodies in blood dyscrasias: A diagnostic aid

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ABSTRACT

The analysis of inclusions includes a detailed study of cellular and nuclear structures and their modified presentation in a given pathology. This is done using a lot of staining procedures and various very small procedures. Among the many cases that can be encountered with the oral cavity, those in the field of hematological diseases should represent, at the dentist, the area with the highest load. The presence of implants is often an important diagnostic aid in diagnosing an underlying disease. Therefore, the current article is an attempt to incorporate the distinct implants seen in blood dyscrasias.

1. Introduction

Among the many cases that may be encountered in the oral cavity, those in the field of Hematological Diseases should represent, at the dentist, the area of excessive responsibility for the treatment, as Dental blood work performed without a detailed analysis of the patient's general condition is possible. It has led to serious problems and sometimes even death.[1]

The clinical signs of various blood dyscrasias in the oral cavity vary widely and are sometimes very similar to other local or general conditions, forcing the dentist to have a thorough knowledge of specific signs and symptoms in each condition manifested at this stage, in order to make a more accurate and different diagnosis.[2] Of course, not all symptoms of blood dyscrasia are commonly encountered in stomatological practice, but some of them, and especially in acute leukemia and all its forms, should always be in the hands of staff, both motivated by the fact that they threaten the patient's health, but also because they are often especially these days, due to many natural features, which are not fully clarified, yet.[1] The presence of inclusions is often an important diagnostic aid in identifying the underlying disease. Therefore, the current article is an attempt to incorporate the distinct implants that appear in blood dyscrasias.

2. Howell-Jolly Bodies (Figure 1)

Howell-Jolly's carcasses are nuclear fossils containing DNA in red blood cells. They are 1 to 2 um in size and can appear on their own or double in the eccentric area on the periphery of the cell membrane. They are thought to develop during periods of rapid or abnormal erythropoiesis.[3] This introduction of red blood cells is due to the remaining DNA residue during bone marrow transplantation and was first recognized by William Henry Howell and Justin Marie Jolly. These bodies appear basophilic and their ring resembles the appearance of red cells that mimic parasites.[4] They can be seen in Romanowsky, i.e., Wright's, Giemsa, or smears with large spots. The chromosome clip is removed and left floating in the cytoplasm after the nucleus is removed. Under normal circumstances, the spleen fits snugly into the noncompressed bodies from the cell.[5] However, in times of erythroid stress, the parking method

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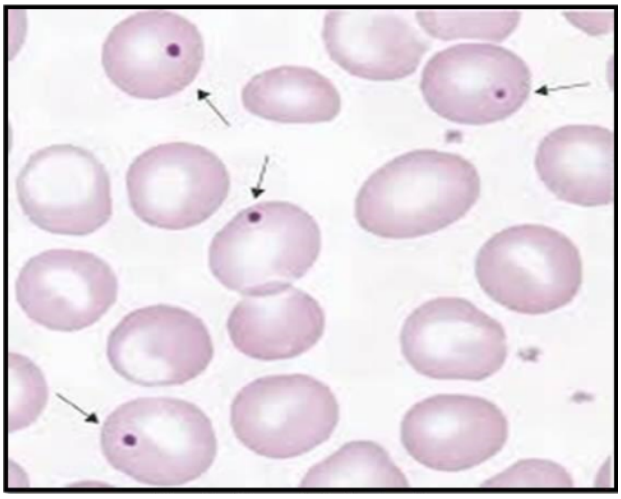

Fig. 1: Howell-jolly bodies

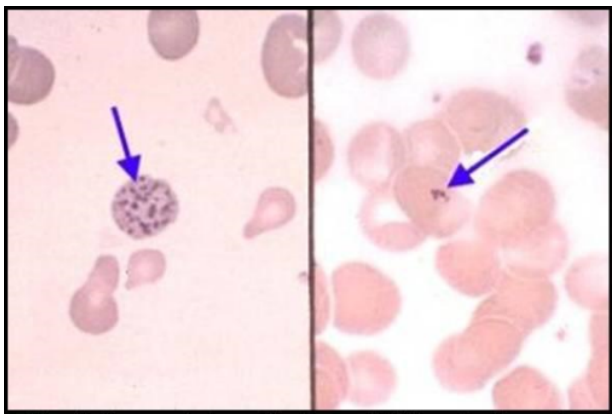

Fig. 2: Pappenheimer and sidrotic granules

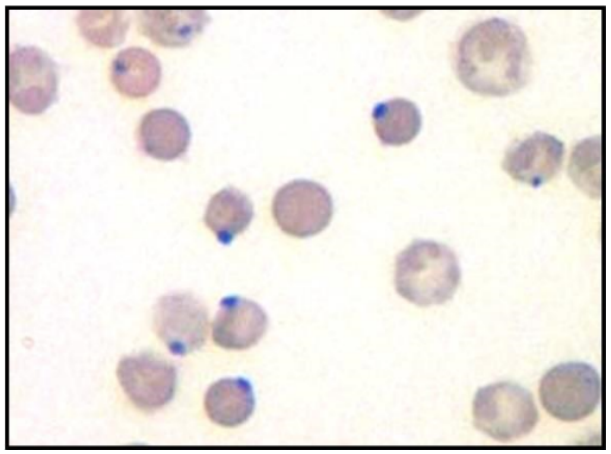

Fig. 3: Heinz bodies

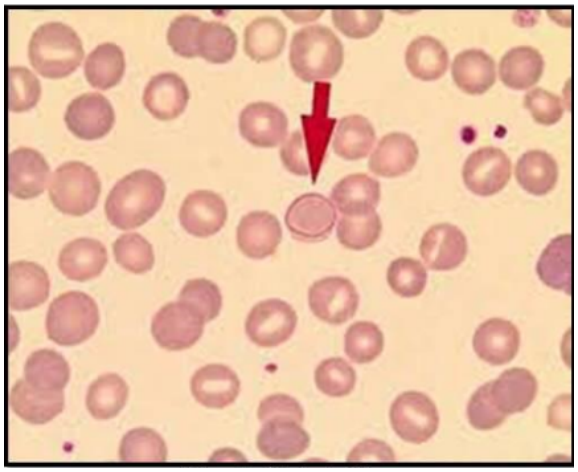

Fig. 4: Cabot ring

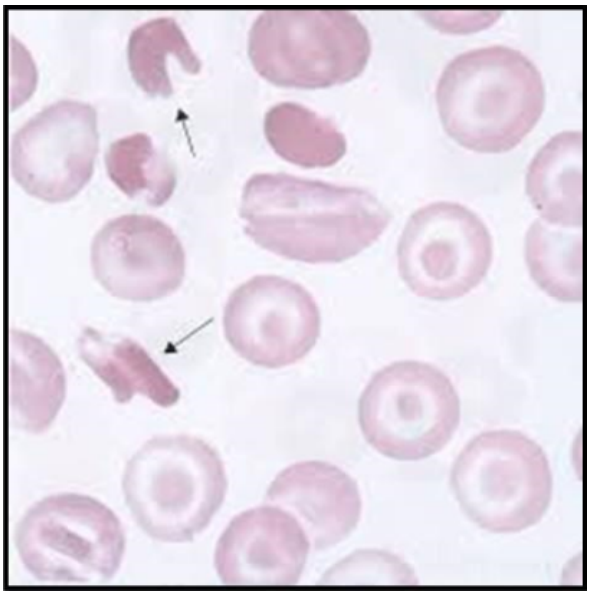

Fig. 5: HbCC inclusions

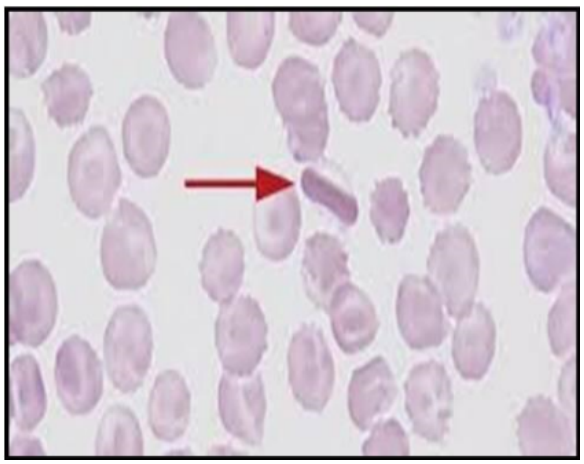

Fig. 6: Hb SC inclusions

Howell-Jolly's bodies can be seen after splenectomy surgery, spinal cord injury, or splenic atrophy after multiple complications. They can also be seen in patients with thalassemic syndromes, sickle cell anemia and other hemolytic anemias, and in megaloblastic anemias.[6]

3. Pappenheimer Bodies and Siderotic Granules (Figure 2)

In 1945, Pappenheimer et al. described three patients whose red blood cells, after splenectomy, showed implants when they had Giemsa spots or Wright spots. They described these bodies as reddish-purple, usually coccoid, and close to the cell membrane, and showed that the cells had metallic spots in Prussia blue. throughout the body.[7] Although Pappenheimers and siderotic granules are not the same, they are selected differently depending on the color used. Inclusions are called Pappenheimer bodies when seen in Wright-stain smear and siderotic granules when seen in Prussian blue or other types of metallic pigment. The definition of the term variation is Pappenheimer (Wright color) Romanowsky spots that show Pappenheimer's bodies by contaminating granule proteins, while Prussia's blue color is responsible for contaminating part of the granule metal. When the presence of siderotic granules is confirmed by metal spots, the cells in which they are found are called siderocytes. Siderocytes containing the nucleus are described as sideroblasts and are commonly seen in sideroblastic anemias.[8] Sideroblasts exhibit numerous siderotic granules found within mitochondria that form a ring around at least one-third of the nucleus, labeled as pathologic sideroblasts. Siderocytes appear in any condition where there is an overload of iron such as hemochromatosis or hemosiderosis. They can also be seen in hemoglobinopathies (e.g., sickle cell anemia and thalassemia) and in patients undergoing splenectomy.[9]

4. Heinz Bodies (Figure 3)

In 1890, Robert Heinz, a German physician, described the transfusion of red blood cells for patients with hemolytic anemia. They are named after Heinz's body. Hundreds (0.3 to 211m) inputs are strong and distort the cell membrane. Heinz 'bodies were formed as a result of damaged DNA, usually by oxidation or by changes in the internal morphology of the amino acid residue within red blood cells. These damaged cells are corded prematurely and removed by macrophages. They can be formed by in vitro detection by incubation with phenylhydrazine (a powerful oxidizing agent). In the first exposure to phenylhydrazine, tiny crystalline bodies appear, combine, and migrate to the underside of the cell membrane. This procedure is applied before applying crystal violet or cresyl blue where the presence of Heinz bodies can be seen in the peripheral smear. Heinz's bodies cannot be seen with Romanowsky spots. Special colors like crystal violet and Wright color can be used to show these bodies. Heinz bodies can be detected in thalassemic syndromes, glucose-ti-phosphate dehydrogenase (G6PD) deficiency under oxidant pressure, and in any unstable hemoglobin syndromes (i.e., hemoglobin Koln, hemoglobin Zur). They can also be detected by damage to red blood cells caused by chemical exposure.[10,11]

5. Cabot Rings (Figure 4)

The exact physiologic effect on the construction of Cabot rings remains to be seen. Using ammoniacal silver dye, Cabot rings are obtained from blood erythrocytes from patients with untreated malignant anemia. This structure can represent part of a mitotic spinning handle, remnants of microtubules, or a piece of nuclear membrane. Studies of the structure of these erythrocytes have shown silver deposition in partial strands and eight forms, suggesting that arginine-rich histone may be a prominent feature of the Cabot ring. Cabot rings can be found in megaloblastic anemias, dyserythropoiesis, homozygous thalassemia syndromes, and in postsplenectomy.[11,12]

6. Hemoglobin CC Crystals (Figure 5)

Hemoglobin (Hb) C crystals can be found in hemoglobin CC disease. HbC disease is a chronic hemolytic anemia in which the patient has a homozygous hemoglobin C. Hb CC crystals are formed by crystallization of abnormal hemoglobin at the end of the red cell membrane. The crystal forms are in a permanent state with dull boundaries, leaving the rest of the cell appear empty. These particles often contaminate the red color and are said to resemble a "gold bar" and can be referred to as such. HbCC crystals may not always be shown in HbC disease, but their appearance has been found to increase after splenectomy. HbCC crystals are not visible on HbC trait (HbAC).[11–13]

7. Hemoglobin SC Crystals (Figure 6)

Hemoglobin SC (HbSC) crystals can be found in smears in parts of patients diagnosed with HbSC. SC is a chronic hemolytic disease that is caused by a serious condition and various bodily injuries, exacerbated by the presence of HbS and HbC. Each common as a disease from HbS and crystallization from HbC. The result of this combination is the formation of finger-shaped projection crystals from the cell membrane. The hypothesis is said to be "similar to the Washington Monument and therefore SC crystals can be called "Washington Monument" crystals.[11,14]

8. Fessas Body

Blood smears of the borderline patient from the homozygous form of Thalassemia show intracellular

implants known as the Fessas body. Decreased production of β-chain hemoglobin occurs resulting in excessive α-chain hemoglobin production in red blood cells. Improper production of hemoglobin leads to severe hypochromic red blood cell count and increased α-chain hemoglobin deposits leads to premature cleansing of the red blood cell wall. [15]

9. Source of Funding

None.

10. Conflict of Interest

None.

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