On the function of the Megakaryocyte (Motility, Separation of the Platelet and Phagocytosis), Observations Both in Idiopathic Thrombocytopenic Purpura and in Normal Adult

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Abstract

The idiopathic thrombocytopenic purpura was described by Werlhof as an independent disease first in 1738. Kaznelson reported the excellent effect of splenectomy for its chronic type in 1915. For the genesis of its thrombocytopenia, there have been many theories to be concluded into the followings, 1) the development of an auto-immune mechanism resulting in platelet destruction, 2) increased platelet destruction in the spleen, 3) the inhibition of platelet production from the marrow megakaryocytes by a humoral factor produced in the spleen, 4) both increased destruction and decreased production of the platelet. Among the above four theories, the third one is the most popular in the chronic type.

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(MOTILITY, SEPARATION OF THE PLATELET, PHAGOCYTOSIS)
OBSERVATIONS BOTH IN IDIOPATHIC THROMBOCYTOPENIC PURPURA AND IN NORMAL ADULT

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The idiopathic thrombocytopenic purpura was described by Werlhof as an independent disease first in 1738. Kaznelson reported the excellent effect of splenectomy for its chronic type in 1915. For the genesis of its thrombocytopenia, there have been many theories to be concluded into the followings, 1) the development of an auto-immune mechanism resulting in platelet destruction, 2) increased platelet destruction in the spleen, 3) the inhibition of platelet production from the marrow megakaryocytes by a humoral factor produced in the spleen, 4) both increased destruction and decreased production of the platelet. Among the above four theories, the third one is the most popular in the chronic type.

We have had recently three cases (No. 1, age 45 years, No. 2, 39, No. 3, 58, all female) of the chronic type of this disease. All of the cases showed remarkable increase of platelets by splenectomy and the tissue culture of their bone marrow was studied before and after the operation.

In the tissue culture of the patients, the growth of the tissue, the cell density and the migratory power of the neutrophiles were similar to the normal, but the changes of the megakaryocytes were very peculiar and showed hyperfunction and accelerated motility immediately after the operation, they were at the site of severe hypofunction before the operation compared with the normal.
The normal megakaryocytes

The mature megakaryocytes showed a complicated shape and stretched out the pseudopodia at places of the margin of the cytoplasm. Rarely they stucked out tentacle-like filaments, which showed considerably active movement and their top made a platelet sometimes. The average of the migration velocity 3.3 mic./min.

The immature megakaryocytes showed only the rotatory movement and no migration. The phagocytosis of carbon particles was observed neither in the mature nor in the immature. The nucleus could not be clearly recognized during the movement.
K. Hiraki, T. Ofuji, T. Kobayashi, H. Sunami and K. Awai:

The megakaryocytes of I. T. P.

The number of the megakaryocytes decreased in two cases and increased in one case of the three compared with the normal.

Most of them had neither motility nor tentacle-like filaments in spite of their maturity, and thus showed a severe hypofunction.

The megakaryocytes of I. T. P. after splenectomy

The number of the megakaryocytes in all cases recovered
to the normal. Their motility improved remarkably after the operation and far more active than normal. They stretched out many strange and complicated pseudopodia in all direction, and showed vigorous migration as well as striking formation of tentacle-like filaments.

A phenomenon of platelet separation was sometimes observed at the top of their filaments.

From above-mentioned findings, the followings could be considered.

The mechanism of the platelet separation from the megakaryocytes was almost clarified, and the splenectomy on the patients with chronic I. T. P. resulted in disappearance of the splenogenic toxic substance and immediate recovery of both function and number of the megakaryocytes by elimination of the disturbance of their development and production.

It is concluded that the genesis of chronic idiopathic thrombocytopenic purpura mainly exists in the hypofunction of the megakaryocytes.