AMNIOTIC FLUID INCREASES PLATELET LEUKOCYTE AGGREGATION AND LEUKOCYTE ACTIVATION

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INTRODUCTION: Amniotic fluid embolism is a rare but devastating condition associated with a very high rate of morbidity and mortality. In a case of amniotic fluid embolism, massive platelet aggregations were confirmed in pulmonary capillaries[1]. In general, the postmortem histological diagnosis of amniotic fluid embolism consisted of demonstrating mucus, squamous, leukocytes, platelets, and fatty cells in the arteries of the lung. Nonetheless, the exact pathogenesis of this syndrome remains unknown and significant controversy exists whether platelet and leukocyte should always be activated. Therefore, we evaluated the effects of amniotic fluid on the interaction between the platelet and leukocyte in whole blood as measured by P selectin, MAC-1 expression and platelet leukocyte aggregation.

METHODS: This study was approved by the institutional review board of our hospital, and informed consent was obtained from ASA I-II full term pregnant women before cesarean section (n =20). Amniotic fluid collected before rupture of amniotic membrane and venous blood samples were collected from an antecubital vein. Amniotic fluid was centrifuged to obtain the upper clear fluid for the experiments. The whole blood, purified leukocyte and platelet rich plasma was preincubated with various concentrations of amniotic fluid (0.1-1.5 mg/mL) in vitro. Samples were stained with a saturating concentration of different fluorochrome-conjugated antibodies(CD62p, CD41a, CD11b), and were analyzed on a flow cytometer to measure platelet P selectin, leukocyte MAC-1 expression and platelet-leukocyte aggregation. Reactive oxygen species (ROS) and p38 activation were also detected.

RESULTS: Amniotic fluid significantly induced platelet-leukocyte aggregation in a concentration dependent manner. Amniotic fluid also induced minimal increase of platelet P selectin expression and significantly increased leukocyte MAC-1 expression at concentration of 0.25-1.5 mg/mL. Amniotic fluid had no effect on ROS production of platelet, but significantly induced ROS production and p38 activation of leukocyte at concentration of 0.25-1.5 mg/mL.

DISCUSSION: Platelet-leukocyte interactions may contribute to the development of several pathological conditions including coronary artery disease and stroke. We first demonstrated that amniotic fluid induced platelet-leukocyte aggregation via activating platelet P selectin expression as well as leukocyte MAC-1 expression. To stimulate leukocytes by amniotic fluid could induce activation of p38 and ROS production.

Platelet-leukocyte aggregates may be an important player in the development of amniotic fluid embolism.

REFERENCES:

1. Furukawa S, Urabe H, Nagai Y, et al. J Obstet Gynaecol Res. 2010;36:397-400.