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Vascular

Effects of arterial and venous wall homogenates, arterial and venous blood, and different combinations to the cerebral vasospasm in an experimental model

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Abstract

Background: Risks related to rebleeding of a ruptured intracranial aneurysm have decreased. However, ischemic neurologic deficits related to vasospasm are still the leading causes of mortality and morbidity. It is well known that vasospasm is a dynamic process affected by various factors. The severity of vasospasm in animal models and clinical observations differ from each other. This variability has not been completely explained by blood and blood degradation products. Therefore, metabolites released from the damaged vessel wall during the bleeding are thought to play an important role in vasospasm.

Method: To test this hypothesis, we used 46 male Wistar rats that were divided into 7 groups and administered one of the following to cisterna magna: venous blood, arterial blood, arterial wall homogenate, venous wall homogenate, combined mixture of arterial blood and artery wall homogenate, or combined mixture of venous blood and venous wall homogenate. Brainstems of the rats were excised, and the basilar arteries were harvested for morphometric measurements.

Result: There were significant differences between the degree of vasospasm caused by arterial and venous blood ($P < .0001$). The intraluminal area of the basilar artery was significantly narrower after application of arterial blood, artery wall homogenate, or their combination (49% ± 1%) than after venous groups (30% ± 1.9%) ($P < .0001$).

Conclusion: The results of this experiment demonstrated that metabolites from vessel walls play as important roles in the pathophysiology of vasospasm as blood and blood degradation products. Further investigation of these metabolites will improve our understanding of vasospasm, pathophysiology, and its treatment.

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Keywords: Vessel wall; Metabolite; Vasospasm; Subarachnoid hemorrhage; Blood product

1. Introduction

Subarachnoid hemorrhage is a type of bleeding into the subarachnoid space surrounding the brain. The most common cause is trauma resulting in rupture of an intracranial aneurysm. An estimated 30 000 people have SAH each year [8]. Today, risks related to rebleeding of a ruptured intracranial aneurysm have declined to an acceptable level because of the use of new diagnostic techniques and early surgical interventions; however, DIND related to vasospasm

Abbreviations: AB, arterial blood; AVM, arteriovenous malformation; AW, arterial wall; BA, basilar artery; C, control; CSF, cerebrospinal fluid; DSA, digital subtracted angiography; EDCF, endothelium-derived constrictor factor; EDRF, endothelium-derived relaxing factor; ET-1, endothelin 1; DIND, delayed ischemic neurologic deficits; NO, nitric oxide; OxyHb, oxyhemoglobin; SAH, subarachnoid hemorrhage; VB, venous blood; VW, venous wall.

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after SAH remains the leading cause of mortality and morbidity [8]. Unfortunately, experimental and clinical studies on vasospasm have not clearly identified the factors underlying vasospasm and its' treatment.

In vivo and in vitro studies have demonstrated growth factors, extracellular matrix molecules, vasoconstrictor immunomodulators, and various other mediators released after bleeding to be important factors in the pathogenesis of vasospasm [8].

Vascular tonus is the result of the balance between EDCF (endothelin) and EDRF (NO) [1]. A number of studies have demonstrated that OxyHb in the arterial blood—where it is found in higher amounts than in venous blood—together with ET-1 have important roles in vasospasm [1,19,20].

According to clinical observations, the vasospasm observed after aneurysmal SAH is more prominent than that seen after AVM, trauma, or perimesencephalic SAH. This observation has led to the hypothesis that there should be a difference in the degree of vasospasm caused by arterial blood containing more OxyHb and metabolites from the arterial wall than that caused by venous blood and venous wall metabolites. To test this hypothesis, we investigated the effects of arterial blood, venous blood, arterial wall homogenates, and venous wall homogenates, and their different combinations on the arterial wall using morphometric analyses.

2. Method

We used 46 male Wistar rats weighing between 225 g and 250 g (mean, 235 ± 15 g) according to the guidelines of American National Institutes of Health. Rats were fed with a standard rat diet and tap water.

Rats were randomly divided into 7 groups as follows:

For control (n = 6), 0.2 mL of 0.9% NaCl was injected into the subarachnoid space via puncture of the cisterna magna.

For the arterial blood group (n = 6), 0.2 mL of autologous arterial blood was injected into the subarachnoid space via puncture of the cisterna magna.

For venous blood group (n = 6), 0.2 mL of autologous venous blood was injected into the subarachnoid space via puncture of the cisterna magna.

For the arterial wall homogenate group (n = 6), a mixture of 0.15 mL of 0.9% NaCl and 0.05 mL of autologous homogenate of the femoral artery wall was injected into the subarachnoid space via puncture of the cisterna magna.

For the venous wall homogenate group (n = 6), a mixture of 0.15 mL of 0.9% NaCl and 0.05 mL of autologous homogenate of femoral vein wall was injected into the subarachnoid space via puncture of the cisterna magna.

For the arterial blood and wall homogenate group (n = 6), a mixture of 0.15 mL of autologous arterial blood and 0.05 mL of autologous homogenate of femoral artery wall was injected into the subarachnoid space via puncture of the cisterna magna.

For venous blood and wall homogenate group (n = 6), a mixture of 0.15 mL of autologous venous blood and 0.05 mL of autologous homogenate of the femoral vein wall was injected into the subarachnoid space via puncture of the cisterna magna.

2.1. Experimental SAH model and the study protocol

Animals were anesthetized with intraperitoneal injection of 60 mg/kg ketamine (Ketalar; Pfizer, Istanbul, Turkey) and 6 mg/kg xylazine (Rompun; Bayer, Germany).

Preparation of femoral artery and vein was performed under sterile conditions. Animals were laid in the supine position, and inguinal canals were exposed after a 3-cm skin incision at the femoral region. One centimeter of femoral artery from the animals in the arterial wall homogenate and arterial blood and wall homogenate groups and 1 cm of femoral vein from the animals in the venous wall homogenate and venous blood and wall homogenate groups were harvested. Skin incisions were then sutured.

Tissue samples (1-cm-long femoral arteries from the rats in the arterial wall homogenate and arterial blood and wall homogenate groups and 1-cm-long femoral veins from the rats in the venous wall homogenate and venous blood and wall homogenate groups) were homogenized inside a tube containing 1 mL of 0.9% NaCl solution, in the cold chain, with a microtome (Ultra-turraks T8; IKA Labortechnik, Germany) at 12500 G revolutions per minute, for 0.5 minutes.

For preparation of cisterna magna puncture, rats were positioned into the prone position with the head in overflexion. Using a microscope, under sterile conditions, a median subcutaneous incision was made, starting from the midportion of the calvarium and extending to the lower cervical region. The atlantooccipital membrane was exposed after subperiostal dissection of the paravertebral muscles and muscles overlying the occipital bone.

After exposing the contralateral inguinal canals of the animals in the arterial blood, venous blood, arterial blood and wall homogenate, and venous blood and wall homogenate groups, 0.2 mL of blood was collected with a 26-gauge needle from the femoral arteries of the animals in the arterial blood and arterial blood and wall homogenate groups and from the femoral veins of the animals in the venous blood and venous blood and wall homogenate groups.

A 26-gauge needle was inserted into the cisterna magna of each animal, and 0.1 mL of CSF was collected. Each of the following was then administered through the needle for a 15-second interval:

- 0.2 mL of 0.9% NaCl to the animals in the control group,
- 0.2 mL of autologous arterial blood collected after femoral artery puncture to the animals in the arterial blood group,
- 0.2 mL of autologous venous blood collected after femoral vein puncture to the animals in the venous blood group,

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