



# Cocaine and Platelets Activation: Is there a Link?



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## Editorial

Increasing evidence supports the relationship between substance abuse and stroke [1-4], Cocaine induces both hemorrhagic and ischemic stroke and the stroke type significantly varied according to type of cocaine formulation as well as contaminants and between current and previous cocaine users [1]. In fact, crack-use is associated with both ischaemic and hemorrhagic strokes while cocaine hydrochloride-use with hemorrhagic strokes [1,2]. Multiple mechanisms may be involved in the genesis of ischemic stroke in cocaine users.

Cocaine can also promote endothelial dysfunction in subjects with significant coronary artery disease and ischemic stroke and facilitate thrombus formation in diseased vessels [1,2]. In patients with cocaine-induced myocardial infarction, platelet-rich coronary thrombi were observed suggesting the activation of platelets [5].

Platelets have also a crucial role on vascular disease which are involved in pathogenesis of ischemic stroke. Platelets play an important part in the formation of cerebral atherothrombotic events and ischemic processes, which encompass adhesion, release reaction, and aggregation of platelets [5,6]. Circulating platelets are heterogeneous with regard to their size, density, and reactivity, and platelet volume indices are biomarkers of degree of platelet activation that are thought to be associated with systemic inflammatory responses. These indices include platelet count (PC), mean platelet volume (MPV), and platelet distribution width (PDW). It is accepted that increased MPV may be an indicator of increased platelet activation, and may thus be related to the severity and prognosis of stroke [6].

Increased MPV has been reported in patients with known coronary artery disease risk factors such as smoking, diabetes mellitus, obesity, hypertension, and increased cholesterol

compared to healthy controls [5,6]. Patients with unstable angina have also been reported to have increased MPV when compared to patients with stable angina [5].

Cocaine can also promote endothelial dysfunction in subjects with significant coronary artery disease and ischemic stroke and facilitate thrombus formation in diseased vessels [1,2,5]. In patients with cocaine-induced myocardial infarction and stroke, platelet-rich coronary thrombi were observed which suggested activation of platelets [5]. A decrease in cerebral blood flow in cocaine-dependent patients has been associated with an increase in platelet aggregation [1]. Acute platelet rich thrombi have been observed in fatal cocaine related infarcts, in both normal and atherosclerotic coronary vessels [5].

In chronic cocaine users, the release of cell growth factors by activated platelets might promote atherosclerosis, predisposing users to thrombosis and ischemia in the absence of acute intoxication, despite a young age [1]. The cause of this condition is supported by the finding that advanced atherosclerosis is observed in the renal arteries and aorta of cocaine users [1]. The arteriosclerotic toxicity of cocaine has also been demonstrated in rabbits [1], in which the same pathophysiological process may also occur in intracerebral vessels, leading to cocaine-induced lacunar infarction. Similarly, cocaine consumption is able to induce platelet activation, granule release, reversible stomatocytosis of red blood cells and an increase in plasma von Willebrand factor, with a decrease in bleeding time, in healthy volunteers [1].

Akkus et al. [5] reported that MPV is not elevated in patients with cocaine use even when they had acute myocardial infarction. MPV values were increased 7 days after stroke in patients with acute ischemic stroke without alteration of platelet counts [7]. Moreover a MPV is involved in the predicts delayed cerebral ischemia after subarachnoid hemorrhage [8]. Lok et al. [6]

showed that MPV was not a significantly associated and reliable marker for the prediction of prognosis or functional outcome of first-ever acute ischemic stroke attack. Despite these findings, the role of platelets activation in patients with ischemic stroke remains unclear.

Could the MPV values involved in cocaine users when they had acute ischemic stroke? Further exploration of this area is warranted, as greater knowledge of the effects of cocaine on platelet activation could reveal the importance of platelets in the pathophysiology of ischemic stroke in cocaine users. In addition, the genetic factors for vessel ischemic disease may influence the high-risk of ischemic stroke in cocaine-users with platelets disorders.

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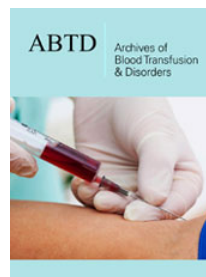
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