Blood-Fluid level: A Sign of Oral Anticoagulant-Associated Intracerebral Hemorrhage in a Patient with Ischemic Stroke

Ismail Ibrahim Ismail*
Department of Neurology, IbnSina Hospital, Sulaibikhat Area, Shuwaikh Industrial Area, 90003, Kuwait

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Description

A 65 year-old female patient with past medical history of diabetes mellitus, hypertension and ischemic heart disease presented with acute onset of right hemiplegia, right facial palsy and dysphasia. Urgent CT brain showed hypodensity involving the territory of the lower division of left middle cerebral artery denoting recent infarction. On admission the patient was found to have Atrial Fibrillation (AF) and LMW Heparin was started followed by Warfarin. One week after initiation of Warfarin the patient started to have headache, increased weakness with deterioration of the level of consciousness. Urgent NECT brain was done and showed hemorrhagic transformation, mass effect and “Blood-fluid level” (Figure 1). Her international normalized ratio (INR) was 3.5.

“Blood/fluid level” or “Sedimentation level” represents interface between the plasma and sedimented blood and is defined radiologically as presence of area of low computed tomography (CT) attenuation above and high CT attenuation below a discrete line of separation in an area of intraparenchymal hemorrhage. It is a rare and unique finding denoting coagulopathy secondary to oral anticoagulant use. It has 59% sensitivity and 98% specificity for indicating underlying coagulopathy. It could be seen within the first 12 hours [1].

The incidence of oral anticoagulant-associated intracerebral hemorrhage (OAC-ICH) is increasing probably due to increased Warfarin use for the treatment of atrial fibrillation. Anticoagulant-associated intracerebral hemorrhage now accounts for nearly 20% of all intracranial hemorrhage. Among patients using warfarin for atrial fibrillation, the annual risk of ICH in trials is 0.3 to 1.0%. Predictors of potential anticoagulant-associated hemorrhage are increasing age, prior ischemic stroke, hypertension, leukoaraiosis, the early period of Warfarin use, higher intensity anticoagulation, and antiplatelet use in addition to anticoagulation [2].

Compared with other intracranial hemorrhage patients, anticoagulated patients have a greater risk of hematoma expansion, subsequent clinical deterioration and may be fatal in at least 50% of cases if untreated necessitating vigorous reversal of their coagulopathy. Most frequently, OAC-ICH are located in deep or lobar regions of the brain, although it may also occur in the cerebellum and brainstem. Early diagnosis and treatment of this condition may improve the 50% mortality in these patients [3].

*Corresponding author: Ismail II, Neurology Specialist, Department of Neurology, Alexandria University, IbnSina Hospital, Sulaibikhat Area, Shuwaikh Industrial Area, Kuwait, 90003, Tel: 96598871710; E-mail: dr.ismail.ibrahim2012@gmail.com

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