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## **Embolic Stroke of Undetermined Source (ESUS) in Obese Patients**

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Article Info	A B S T R A C T
Article type: Review article	<b>Background and Aim:</b> One of the most important contributing factor in stroke is embolic stroke of undetermined source (ESUS), it also named cryptogenic strokes, and occurs frequently despite advances in diagnostic techniques. There are several risk factors and underlying stroke mechanisms associated to ESUS (1).
Article History: Received: 20 January 2020 Revised: 04 March 2020 Accepted: 14 May 2020	Materials and Methods: In this review, we done a general search in Medline, Science Direct and Springer databases during years of 2010 to 2019 and the results discussed here. Antti T. Muuronen, et al suggests that VAT may play an important role in the pathogenesis of thromboembolism (2). Robert G. Hart, et al assessed rivaroxaban for Stroke Prevention after ESUS (3).  Results: In the comparison of all reference populations mean VAT (Visceral Adipose Tissue) area was dramatically higher in stroke patients. An increased in VAT area was seen in 50% of male and 57% of female patients (2). In the prevention of recurrent stroke after an initial ESUS, rivaroxaban was not superior to aspirin. Higher risk of bleeding was seen in rivaroxaban group (3).  Conclusion: The increased visceral fat type in particular was associated with an increase in the risk of stroke and other cardiovascular diseases. Compared between adults with high visceral adiposity and obese patients with lower level of visceral fat, the first group had an insulin resistance, hypertension and dyslipidemia more often. The mentioned metabolic changes lead to plaque formation in both arterial and venous system and, consequently can cause stroke. Visceral fat can be measured carefully with computed tomography (CT) (2). Experiments have shown that the rate of response to insulin and other hormones in visceral adipose tissue is different from subcutaneous adipose tissue. VAT is much more metabolically active than subcutaneous adipose tissue. It damages the endothelium by secreting proinflammatory and anti-inflammatory factors. Also it increased release of procoagulant mediators such as plasminogen activator inhibitor-1 (PAI-1), an endogenous inhibitor of fibrinolysis, tissue factor (TF), and platelet activity that can lead to modulation of inflammatory state and procoagulant response (2).  Antiplatelet therapy was the standard antithrombotic therapy for secondary stroke prevention in all global regions (4).
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