Background
The current stroke literature places increased emphasis on carotid imaging especially plaque imaging. Mobile/ ulcerated soft atheromatous plaques affecting large arteries are a major risk factor for embolic strokes.

Method
We report a 67-year-old female who had embolic right acute cortical infarction with a floating (unstable) plaque in the right internal carotid artery but couldn’t have surgery due to severity of the stroke.

Case report
A 67-year-old lady was admitted to our stroke unit with significant left-sided weakness and left facial palsy. She didn’t have a significant medical history, but she was a heavy smoker and drank an excess of alcohol. Her initial NIHSS score was 21.

Her initial CT brain showed an acute cortical infarct in the right frontal lobe. A carotid duplex was done, and it revealed right internal carotid artery (ICA) mixed plaques and thrombus causing 80-89% stenosis. Her weakness got worse, and she developed new dysphagia, so an MRI brain was done and showed extensive acute infarction in the right hemisphere. A Computed Tomography Angiogram (CTA) showed a soft tissue ulcerated plaque at the right ICA bulb with stenosis of less than 30%.

As she gave a history of recent significant weight loss, CT thorax, abdomen and pelvis was done. A urinary bladder mass was found which was confirmed malignant on cystoscopy examination. She received palliative treatment for her tumour because of her significant disability after the stroke.

Discussion:
Atherosclerosis is a systemic disease. The atherosclerotic plaque is composed of a lipid core and a covering fibrous cap (FC). The plaque will be symptomatic once the changes lead to significant vascular stenosis or occlusion. Vulnerable plaques are still able to cause damage apart from the degree of stenosis. A cascade of changes converts the stable plaque into a vulnerable plaque. Histologically, the vulnerable plaque has a large lipid core and a thin fibrous cap. The high lipid content decreases the load-bearing capability of the plaque and increases the stress in the overlying fibrous cap.

Under certain extrinsic factors, like high blood pressure and vasospasm, the plaque can easily rupture. Other features of plaques vulnerability are neovascularization and intraplaque haemorrhage. There are controversies regarding the effect of arterial calcification on plaque nature. Some studies suggest that calcification gives the plaque more stability while others consider it as a sign of vulnerability, irrespective of the degree of stenosis. The upstream side of the stenosis is considered the oldest part of the plaque. It is proved that it has more neovascularization and haemorrhage with the thinnest FC. This could explain why vulnerable carotid plaque mostly ruptures at this side. Unstable plaques can be symptomatic even in absence of significant carotid stenosis. Hence, early identification of the plaque vulnerability and its medical stabilisation can help to reduce the risk of cerebrovascular insults.

Conclusion:
Our case illustrates the importance of plaque imaging. We recommend that patients with vulnerable soft mobile / ulcerated/unstable plaques on carotid imaging presenting with TIA symptoms/ stroke with NIHSS of less than 5 should be considered for carotid endarterectomy even if the stenosis is less than 50% as per NASCET criteria to prevent further embolic infarctions.

References: