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AN UNUSUAL CERVICALGY.

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Case Report

We report the case of a 47-year-old woman admitted in the emergency department with pain localized to the left and anterior side of the neck. This symptom was briefly preceded by dry cough and rhinorrhea, without any fever reported. The patient has previously been treated with oral amoxicillin-clavulanate for 3 days without improvement. Physical examination at admission revealed an isolated left tenderness overlying the left carotid artery, exacerbated by the touch. Laboratory findings demonstrated normal C-reactive protein level and peripheral blood cell count. An ultrasound investigation revealed a hypoechoic thickening of the carotid bulb wall, with no haemodynamic disturbance in Doppler study. Outcome was favourable under nonsteroidal anti-inflammatory drugs. One-month follow-up revealed an uneventful clinical course with complete ultrasound remission

Discussion

Fay et al first used the term "carotidynia" in 1927 to describe an unilateral neck pain syndrome associated with tenderness over the carotid artery, exacerbated by digital compression, lateral head movement and, sometimes, chewing, swallowing, yawning, sneezing and coughing[1]. The International Headache Society initially classified this clinical entity as an idiopathic neck pain syndrome, but further considered it as a symptom-only condition. Ultrasonographic exam reveals a hypoechoic wall thickening of the artery without haemodynamic changes in Doppler velocity. MR imaging may confirm these anomalies by showing contrast-enhanced tissue surrounding the carotid artery and increased signal on T2-weighted images, corresponding to focal glucose hypermetabolism.

Several publications have studied the structural changes characterised by a focal eccentric thickening of the carotid wall. Indeed, structural modifications seem to be limited to the adventitia. Conventional laboratory tests are usually normal.

Conclusions

The aetiology of carotidynia remains unknown although some authors have suggested that carotidynia may develop after upper airway infections. The treatment remains largely symptomatic with anti-inflammatory drugs and eventually additional oral steroids.