E-ISSN 2148-9211

CVSI

CARDIOVASCULAR SURGERY *and* INTERVENTIONS

Official Electronic Journal of the Turkish Society of Cardiovascular Surgery

Turkish Society of Cardiovascular Surgery 1988

Volume: 6 / Number: 3 / November 2019

E-ISSN 2148-9211

Cardiovascular Surgery and Interventions

Volume 6 - Number 3 - November 2019

Cardiovascular Surgery and Interventions is the official and periodical journal of the Turkish Society of Cardiovascular Surgery.
It is published three times a year.

Executive office:
Türk Kalp ve Damar Cerrahisi Derneği
Ataşehir Mah., Ataşehir Bulvarı, 48 Ada,
Mimoza 2/2, K: 2, D: 6,
34758 Ataşehir, İstanbul, Turkey
Tel: +90 216 - 456 14 54
Fax: +90 216 - 456 14 54
e-mail: info@tkdcd.org
URL: http://www.tkdcd.org

Editorial Contact Person
Bilgin Emrecan, MD.
e-mail: bilginemrecan@yahoo.com

Publisher
Bayçınar Tıbbi Yayıncılık ve Reklam Hiz. Tic. Ltd. Şti.
Örnek Mah., Dr. Suphi Ezgi Sok., Saray Apt., No: 11, D: 6,
34704 Ataşehir, İstanbul, Turkey
Tel: +90 216 - 317 41 14
Fax: +90 216 - 317 63 68
e-mail: info@baycinartibbiyayincilik.com

Type of publication: Periodical
Publication date: March 20, 2020

The control of conformity with the journal standards and the typesetting of the articles in this journal, the control of the English abstracts and references and the preparation of the journal for publishing were performed by Bayçınar Medical Publishing.

Contents

CASE REPORTS

Cardiovasc Surg Int 2019;6(3):35-37
http://dx.doi.org/DOI: 10.5606/e-cvsi.2019.748
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Case Report Open Access

Acute acalculous cholecystitis in a patient undergoing coronary artery bypass surgery

İhsan Alur[1], Mehmet Şirin Yıldırım[2], Özlem Uygun[3], Ali Fedakar[1]

[1]Department of Cardiovascular Surgery, Private Hisar Hospital Intercontinental, Istanbul, Turkey
[2]Department of Anesthesiology and Reanimation, Private Hisar Hospital Intercontinental, Istanbul, Turkey
[3]Department of Radiology, Private Hisar Hospital Intercontinental, Istanbul, Turkey

Received: October 03, 2019 ***Accepted:*** November 26, 2019 ***Published online:*** March 16, 2020

ABSTRACT

Acute acalculous cholecystitis is a very rare complication of gastrointestinal system that can be seen after coronary artery bypass surgery. Early recognition of this clinical-pathological condition and initiation of treatment are very important and vital to prevent serious complications. In this article, we present a 67-year-old male patient of acalculous cholecystitis developing after coronary artery bypass grafting.

Keywords: Acalculous cholecystitis, coronary bypass, surgery.

Acute acalculous cholecystitis (AAC) is an acute inflammation of the gallbladder that develops without gallstones and has a multifactorial pathogenesis.[1] It generally occurs in male patients with an approximate male/female ratio of 2 to 3:1 and 60 years of age.[1] In the etiology, many factors such as major surgery procedures, traumas, bacterial sepsis, viral infection, weakening/suppression of the immune system, collagen tissue diseases, and hyperalimentation have been charged while the cause has not been fully elucidated.[1] Acute acalculous cholecystitis is most commonly associated with right upper quadrant pain, fever, and leukocytosis.[1] Liver function tests, serum bilirubin and alkaline phosphatase levels increase.[1] The most sensitive, specific, and low-cost imaging modality in the diagnosis is hepatobiliary ultrasonography (US). Dilatation of the gallbladder, presence of echogenic bile sludge, and thickening of the gallbladder wall of more than 3 mm are important sonographic findings.[1,2] In this article, we report a case of AAC, which developed after coronary artery bypass grafting (CABG) and was successfully treated.

CASE REPORT

A 67-year-old male patient presented with atypical chest pain. He had a history of diabetes mellitus (DM), dyslipidemia, and hypertension (HT). On physical examination, his pulse rate was 84/min, he had a blood pressure of 90/60 mmHg, and peripheral pulses were palpable. The electrocardiogram showed sinus rhythm. The patient's family history revealed that his father had died at the age of 48 due to myocardial infarction. Multiple coronary artery disease was diagnosed in coronary angiography. Subsequently, the patient underwent five-vessel CABG. The operation was routinely completed without complications. After the operation, the patient was admitted to the intensive care unit. On postoperative day three, the general condition of patient deteriorated, oxygen saturation decreased, and anxiety developed. Transthoracic echocardiography showed that left ventricular ejection fraction was 50%, while pericardial effusion was 27 mm in the posterior wall and 25 mm in the right lateral wall. White blood cell count was 20,870 10^3/μL (normal range: 4.16 10^3/μL-10.20 10^3/μL). Aspartate aminotransferase was 144 IU/L (normal range: 0-37), gamma-glutamyl transpeptidase was 190 IU/L (normal range: 7-60), alanine aminotransferase was 136 IU/L (normal range: 0-41), alkaline phosphatase was 74 IU/L (normal range: 40-129), and C-reactive protein was 1.79 mg/dL (normal range: 0-0.5). Because the liver enzymes of the patient were high,

Corresponding author: İhsan Alur, MD. Hisar Hospital Intercontinental, Kalp ve Damar Cerrahisi Bölümü, 34768 Ümraniye, İstanbul, Turkey.
Tel: +90 216 - 524 13 00 e-mail: alur_i@hotmail.com

Citation:
Alur İ, Yıldırım MŞ, Uygun Ö, Fedakar A. Acute acalculous cholecystitis in a patient undergoing coronary artery bypass surgery. Cardiovasc Surg Int 2019;6(3):35-37.

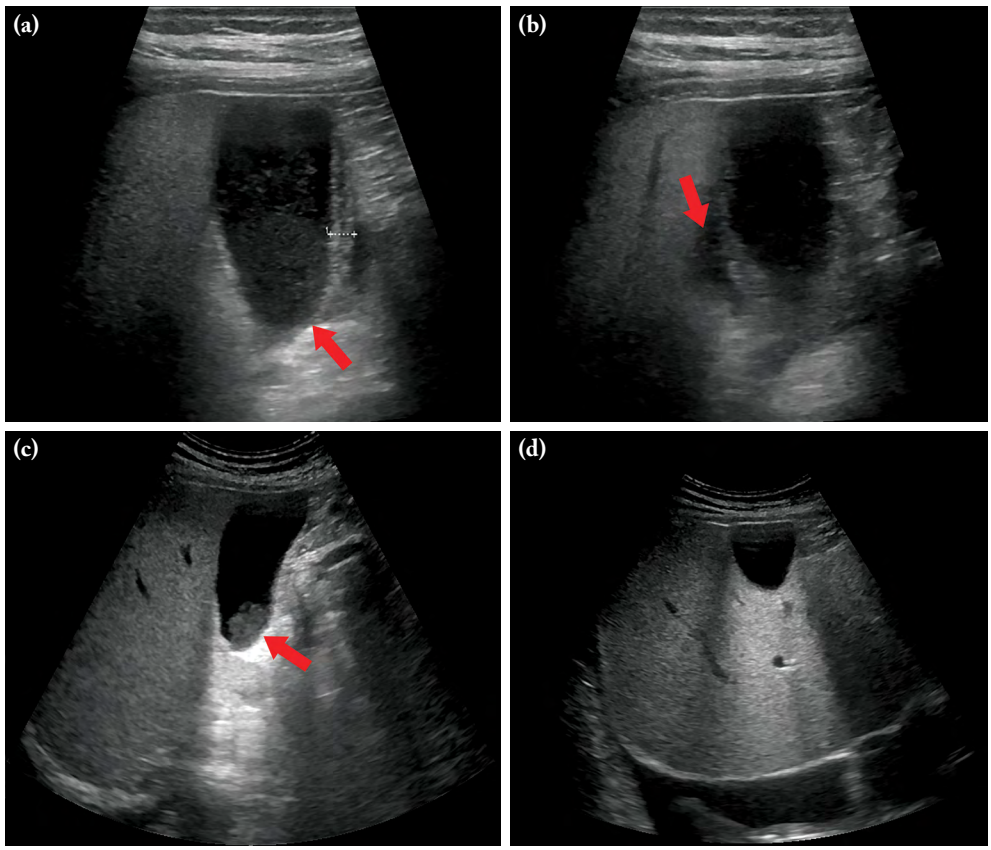

Figure 1. **(a)** Dense biliary sludge filling lumen of gallbladder (red arrow). **(b)** Fluid accumulation was observed in perihepatic area (red arrow). **(c)** Decreased biliary sludge filling lumen of gallbladder (red arrow). **(d)** Decreased perihepatic fluid and volume of gallbladder.

viral hepatitis markers were studied and found to be normal. Acute viral hepatitis was excluded in the differential diagnosis. Hepatobiliary US showed that liver was in normal shape/localization. Borders were regular. Parenchyma echogenicity was rough and Grade I hepatosteatosis was present. There was a slight increase in periportal echogenicity. There was no lesion that showed significant contour demarcation in the parenchyma. The width of the hepatic and portal veins was normal and the lumen was anechoic. The localization, morphology, and dimensions of the gallbladder were natural; the wall thickness was 6 mm and layered. Dense biliary sludge filling the lumen of the gallbladder was observed. Increased intensive internal echo and pericholecystic fluid were observed. There was no dilatation in intra-extrahepatic biliary tract and common bile duct. Fluid accumulation was observed in the perihepatic area (Figure 1a, b). A written informed consent was obtained from the patient.

Since there was no serious complication in our case, we followed the patient with medical treatment. Oral feeding was stopped for three days. The patient was fed with liver protective feeding solution. Liver enzymes returned to normal. Nausea-vomiting symptoms disappeared. One week later, the US findings were normal (Figure 1c, d). The patient was discharged on the postoperative 14th day.

DISCUSSION

Acute acalculous cholecystitis is a rare but serious complication after CABG. Mostly, it is difficult to distinguish from AAC. This disease can progress rapidly. Early recognition of AAC and initiation of treatment are very important and crucial to prevent serious complications. Clinical symptoms and findings include right subcostal pain sensitivity, fever, leukocytosis, excessive sweating, weakness, loss of appetite, nausea, vomiting, and increase in liver

function tests and bilirubin levels.[1] In our case, most of these findings were present. Our patient did not develop fever due to use of antibiotics. There was no right subcostal pain due to postoperative analgesic. The absence of abdominal pain or lack of right subcostal sensitivity may be due to diabetic neuropathy. Acute acalculous cholecystitis has some lethal complications reported in the literature such as gangrene, perforation, peritonitis, pericholecystic abscess, empyema, and cholecystobiliary fistula.[2] The most commonly used imaging modalities for diagnosis are hepatobiliary US or computed tomography (CT). These findings were reported in both hepatobiliary US and CT in our case.

The most commonly proposed mechanisms include bile stasis or ischemia. Bile stasis may occur after fasting, obstruction, chronic irritation or ileus (prolonged total parenteral nutrition), which may cause bile condensation. This may lead to a direct toxic effect on the gallbladder epithelium.[1] The other mechanism is the development of organ ischemia due to many risks such as atherosclerosis, DM, HT and vasculitis syndromes. In one study, leukocyte migration and accumulation due to ischemia and reperfusion in the gallbladder wall, increased focal lymphatic permeability and dilatation due to local microvascular blockage, and gallbladder epithelial damage due to increased bile duct permeability have been reported.[3] In our case, ischemia-related cholecystitis may have developed since DM, HT, and dyslipidemia (preoperative low-density lipoprotein cholesterol: 133 mg/dL and triglyceride: 364 mg/dL) risk factors were present. In addition, the patient was a 67-year-old male and was consistent with the literature. Therapeutic options of the AAC include medical conservative treatment, percutaneous transhepatic gallbladder drainage or surgical interventions such as cholecystectomy and cholecystostomy.[1] The mortality rate was reported as 23-40% in patients with postoperative AAC. The risk of mortality is higher in patients older than 75 years and in diabetic patients.[2]

In conclusion, acute acalculous cholecystitis has a high mortality rate after coronary bypass surgery. Early intervention and treatment are life saving. We believe that this clinical entity may appear silent in diabetic postoperative patients and it should not be overlooked. We also believe that this phenomenon is stimulative and remarkable for cardiovascular surgery specialists.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Huffman JL, Schenker S. Acute acalculous cholecystitis: a review. Clin Gastroenterol Hepatol 2010;8:15-22.
2. Girgin S, Gedik E, Aldemir M, Yağmur Y. Current treatment and surgery of acute cholecystitis. Turkiye Klinikleri J Surg Med Sci 2007;3:44-9.
3. Laurila JJ, Ala-Kokko TI, Laurila PA, Saarnio J, Koivukangas V, Syrjälä H, et al. Histopathology of acute acalculous cholecystitis in critically ill patients. Histopathology 2005;47:485-92.

Cardiovasc Surg Int 2019;6(3):38-40
http://dx.doi.org/DOI: 10.5606/e-cvsi.2019.739
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Case Report Open Access

Intraplaque hemorrhage causing recurrent stroke treated by carotid endarterectomy

Serkan Ertugay[1], Cenk Eraslan[2], Hakan Posacıoğlu[1]

[1]Department of Cardiovascular Surgery, Ege University Faculty of Medicine, Izmir, Turkey
[2]Department of Radiology, Ege University Faculty of Medicine, Izmir, Turkey

Received: March 06, 2019 ***Accepted:*** May 17, 2019 ***Published online:*** March 16, 2020

ABSTRACT

Atherosclerotic plaque characteristics such as intraplaque hemorrhage, thickness of fibrous cap and large lipid-rich necrotic core are the predictors of future cerebrovascular events. Those features are examined by vessel wall magnetic resonance imaging. In this article, we present a 60-year-old male patient who underwent carotid endarterectomy because of intraplaque hemorrhage causing recurrent transient ischemic attacks. Because of the recurrent ischemic attacks, carotid endarterectomy was performed emergently. Patient was discharged after uneventful postoperative course. Vessel wall magnetic resonance imaging is the advanced evaluation of carotid artery disease which determines high-risk plaque according to intraplaque hemorrhage and thin fibrous cap. In case of recurrent symptoms and presence of high-risk plaque, carotid intervention should be considered regardless of the degree of stenosis.

Keywords: Atherosclerosis, carotid artery disease, intraplaque hemorrhage, magnetic resonance imaging.

According to guidelines, the recommendation of carotid endarterectomy (CEA) or carotid artery stenting for carotid artery disease (CAD) is based on the degree of stenosis, presence of symptoms and the risk of CEA.[1] However, plaque characteristics of vulnerability may also predict future cerebrovascular events.[2,3] Doppler ultrasonography (US) and computed tomography (CT) are used commonly at first step for the diagnosis of CAD. Besides, vessel wall magnetic resonance imaging (MRI) has the advantages to demonstrate high-risk carotid plaque characteristics. In this article, we present a patient who underwent CEA because of intraplaque hemorrhage (IPH) causing recurrent transient ischemic attacks.

CASE REPORT

A 60-year-old male patient, without history of risk factor for atherosclerosis, presented with transient ischemic attack causing dysarthria and weakness on right side of the body. After the onset of the symptoms, diffusion weighted MRI study (Siemens Verio 3 Tesla System, Erlangen, Germany, Diffusion B1000 images/apparent diffusion coefficient maps) showed multiple acute infarct areas on right cerebral hemisphere. The infarct areas were located within bilateral centrum semiovale. With the intent of etiologic evaluation, carotid Doppler US revealed atherosclerotic carotid plaque with non-significant stenosis on the left side. Computed tomography angiography showed atherosclerotic plaque without significant narrowing in the left carotid artery (Figure 1). Transesophageal echocardiography showed small plaque on arcus aorta which was not ulcerated. Patient was treated with acetylsalicylic acid and unfractionated heparin. He was discharged with optimal medical treatment including dual antiplatelet therapy, lipid lowering and antihypertensive drug. Two days later, patient was readmitted with recurrent ischemic attack. Vessel wall imaging (T2 axial fat saturated turbo spin-echo, T1 pre-contrast fat saturated axial, T1 post-contrast fat saturated images; diffusion B200, B400 images) was performed. Intraplaque hemorrhage was detected into the atherosclerotic plaque located in the left carotid bifurcation (Figure 2). Because of recurrent attacks, CEA was performed urgently. As surgical approach, endarterectomy was performed by conventional technique without using patch or shunt. Macroscopic view of the plaque confirmed large area of plaque

Corresponding author: Serkan Ertugay, MD. Ege Üniversitesi Tıp Fakültesi Kalp ve Damar Cerrahisi Anabilim Dalı, 35100 Bornova, İzmir, Türkiye.
Tel: +90 232 - 390 40 49 e-mail: serkanertugay80@gmail.com

Citation:
Ertugay S, Eraslan C, Posacıoğlu H. Intraplaque hemorrhage causing recurrent stroke treated by carotid endarterectomy. Cardiovasc Surg Int 2019;6(3):38-40.

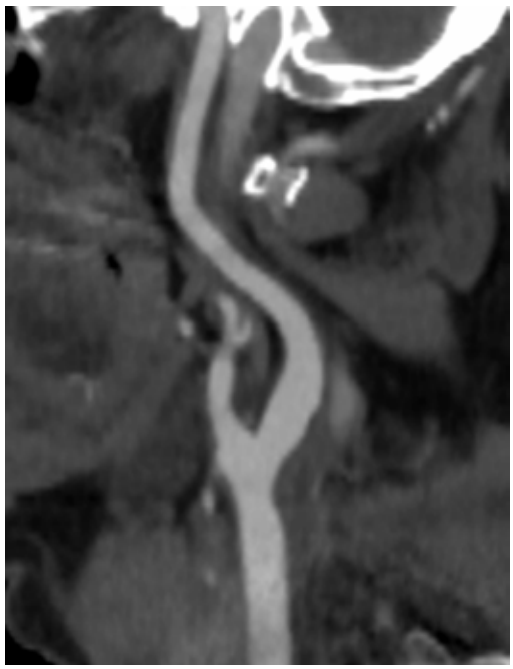

Figure 1. Reconstructed minimum intensity projection computed tomography angiography image showing plaque without significant stenosis.

hemorrhage (Figure 3). Time interval between the onset of symptoms and surgery was four days. Patient was discharged with dual antiplatelet, statin and antihypertensive drugs after uneventful postoperative course. During follow-up, he was asymptomatic which confirmed that ischemic attacks were caused by the IPH. A written informed consent was obtained from the patient.

DISCUSSION

Doppler scanning is usually the first step of the diagnostic algorithm of ischemic stroke. Angiographic (CT or digital subtraction) evaluation of carotid plaque is needed to determine the severity of stenosis and to plan any intervention. However, MRI has the advantages to detect plaque morphology and composition in addition to stenosis. Diffusion weighted MRI is the main imaging modality to diagnose infarct after ischemic stroke. Further evaluation of plaque characteristics by vessel wall MRI such as IPH, lipid-rich necrotic core, and thickness of fibrous cap determines its vulnerability. Zhao et al.[2] found

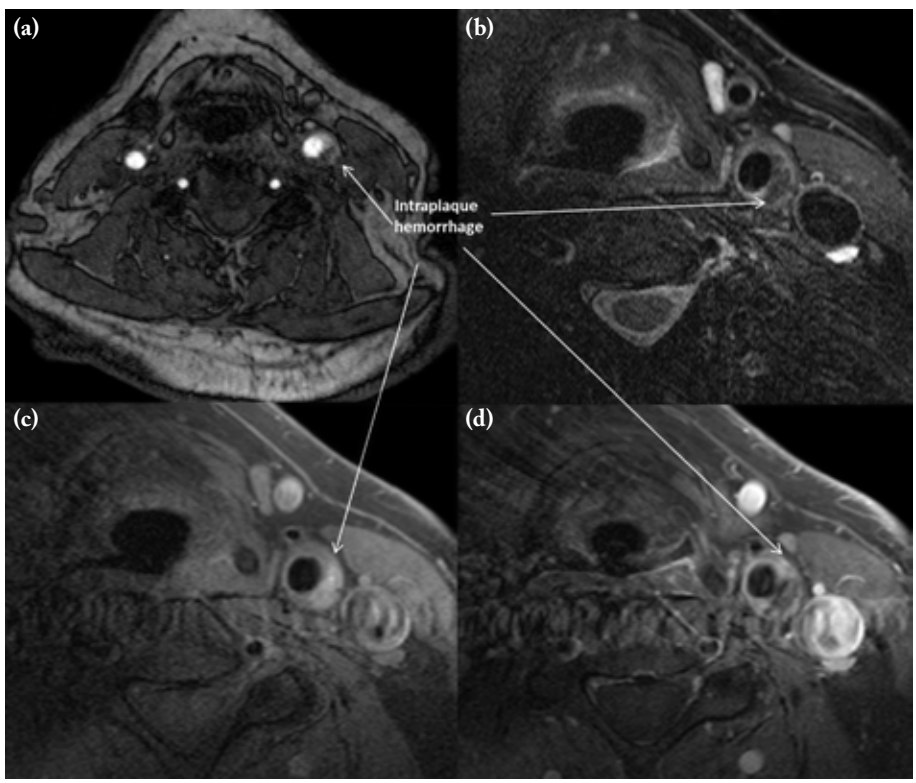

Figure 2. **(a)** Time-of-flight magnetic resonance angiography image. **(b)** T2 axial fat saturated image. **(c)** T1 axial fat saturated image. **(d)** T1 axial post-contrast fat saturated image at level of hemorrhagic plaque.

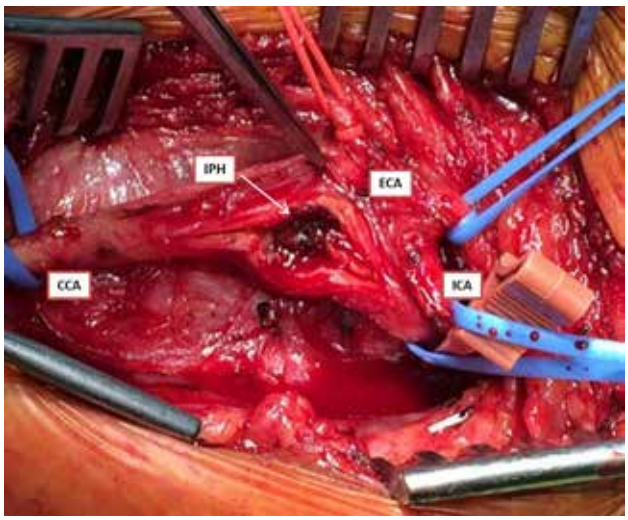

Figure 3. Intraoperative view of intraplaque hemorrhage located in carotid bifurcation.

CCA: Common carotid artery; ECA: External carotid artery; ICA: Internal carotid artery; IPH: Intraplaque hemorrhage.

that presence of those features is commonly related to the degree of stenosis. Furthermore, IPH was not detected in patients with carotid stenosis of less than 30% in this study. Unlikely, in our case, despite non-significant stenosis, IPH was large and the major cause of recurrent stroke attacks.

These features were analyzed to predict future ischemic events in patients with CAD. Virmani et al.[4] described that atherosclerotic plaque with thin fibrous cap (<0.2 mm) and distinct lipid-rich necrotic core carries a high risk of rupture in coronary artery disease. In line with this, in their study based on MRI, Gijsen et al.[5] have analyzed plaque composition which creates peak cap stress and found a significant association between thin fibrous cap atheroma and high peak stress. Intraplaque hemorrhage is the other common feature of carotid plaque which may cause stroke. Intraplaque hemorrhage is the result of ruptured neovascularization and commonly cause fibrous cap disruption.[6] However, to date, there is still no evidence-based recommendation suggesting carotid intervention according to plaque composition in patients with low degree of stenosis. In this case, recurrent ischemic stroke was the main indication for intervention despite optimal medical therapy. Vessel wall MRI revealed IPH causing recurrent events which required urgent CEA.

In conclusion, features of vulnerable carotid plaque, such as intraplaque hemorrhage and thin fibrous cap, are predictors of future stroke and can be identified by magnetic resonance imaging. In case of recurrent symptoms caused by high-risk plaque despite optimal medical therapy, carotid endarterectomy might be considered regardless of the degree of stenosis.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Abbott AL, Paraskevas KI, Kakkos SK, Golledge J, Eckstein HH, Diaz-Sandoval LJ, et al. Systematic Review of Guidelines for the Management of Asymptomatic and Symptomatic Carotid Stenosis. Stroke 2015;46:3288-301.
2. Zhao H, Zhao X, Liu X, Cao Y, Hippe DS, Sun J, et al. Association of carotid atherosclerotic plaque features with acute ischemic stroke: a magnetic resonance imaging study. Eur J Radiol 2013;82:e465-70.
3. Gupta A, Baradaran H, Schweitzer AD, Kamel H, Pandya A, Delgado D, et al. Carotid plaque MRI and stroke risk: a systematic review and meta-analysis. Stroke 2013;44:3071-7.
4. Virmani R, Kolodgie FD, Burke AP, Farb A, Schwartz SM. Lessons from sudden coronary death: a comprehensive morphological classification scheme for atherosclerotic lesions. Arterioscler Thromb Vasc Biol 2000;20:1262-75.
5. Gijsen FJ, Nieuwstadt HA, Wentzel JJ, Verhagen HJ, van der Lugt A, van der Steen AF. Carotid plaque morphological classification compared with biomechanical cap stress: Implications for a magnetic resonance imaging-based assessment. Stroke 2015;46:2124-8.
6. Kawahara I, Nakamoto M, Hayashi K, Kitagawa N, Tsutsumi K, Nagata I, et al. The detection of carotid plaque rupture caused by intraplaque hemorrhage by serial high-resolution magnetic resonance imaging: a case report. Surg Neurol 2008;70:634-9.