Muslinoma and muslin-induced foreign body inflammatory reactions after surgical clipping and wrapping for intracranial aneurysms: imaging findings and clinical features

Clinical article

MIN A. YOON, M.D.,1 EUNHEE KIM, M.D.,1 BAE-JU KWON, M.D.,1 JEEONG EUN KIM, M.D.,2 HYUN-SEUNG KANG, M.D.,2 JAE HYO PARK, M.D.,3 CHUL-HO SOHN, M.D.,1 JI-HOON KIM, M.D.,1 and DONG HOON LEE, M.D.1

Departments of 1Radiology and 2Neurosurgery, Seoul National University College of Medicine, Seoul; and 3Department of Neurosurgery, Kangwon National University College of Medicine, Kangwondo, Korea

Object. Reinforcement of aneurysms with additional wrapping is an alternative procedure if the aneurysm cannot be completely clipped. Wrapping with muslin (cotton gauze) rarely incites foreign body inflammatory reactions. In this study, the authors describe the clinical and radiological features of muslinomas or muslin-induced foreign body reactions that can develop after treatment of intracranial aneurysms.

Methods. Over a 3-year period, 5 patients with muslinomas underwent treatment at the authors’ institution. All patients underwent aneurysm clipping and wrapping, and were subsequently readmitted with acute or subacute neurological symptoms. Clinical and imaging features on diffusion weighted MR images and cerebral angiography images were retrospectively reviewed. The patients’ clinical course and follow-up imaging studies were also evaluated.

Results. In all 5 cases, muslinomas were seen as rim-enhancing inflammatory masses around the clipped aneurysms with perilesional edema visible on MR images at the time of clinical deterioration. The MR images also demonstrated adhesive arachnoiditis with a sterile intracranial abscess in 3 patients, optic neuropathy in 2, parent artery narrowing in 2, and a resultant acute ischemic infarction in 1 patient. Follow-up imaging revealed resolution of both the perilesional edema and adhesive arachnoiditis but no significant changes in the muslinomas. All patients underwent conservative management and fully recovered, but during the follow-up period, 2 patients experienced clinical and radiological relapses.

Conclusions. When a patient with a history of wrapping of an aneurysm presents with acute neurological symptoms and an enhancing intracranial mass in the region of the surgical site on MR imaging, a muslin-induced foreign body inflammatory reaction should be considered in the differential diagnosis, and careful clinical and radiological follow-up is advised. (DOI: 10.3171/2009.7.JNS081625)

KEY WORDS • muslinoma • aneurysm • wrapping • magnetic resonance imaging

Therapeutic approaches to intracranial aneurysms include surgical clipping as well as less invasive neurointerventional coil embolization, depending on the size, shape, and location of the aneurysm. When an aneurysm is unfavorable for clipping alone or its neck cannot be clipped completely, reinforcement with circumferential wrapping using muslin gauze is a well-established neurosurgical procedure.1,11,15,21 Muslin or cotton gauze induces the generation of a fibrotic scar tissue that stabilizes the vascular wall and adheres to surrounding tissue.19 This fibrotic reaction remains restricted to a small area around the aneurysm, but occasionally the inflammatory response can expand to adjacent structures. Consequently, depending on the anatomical site of the wrapped aneurysm, various neurological complications can occur. A spectrum of manifestations of foreign body inflammatory reactions has been illustrated in previous reports.2,5,5–10,12–14,17 When delayed foreign body reactions result in mass formation, the mass is described as a muslinoma, gauzoma, or gauze granuloma. The occurrence of optic neuropathy or chiasmal syndrome in this setting, a term for muslin-induced optochiasmatic arachnoiditis and muslin-induced optic neuropathy, is relatively well-known among neurosurgeons.2,5,10,12,14,17,18 The vessel wall of adjacent arteries can be infiltrated likewise and occluded,2 and there is 1 report in the literature of delayed parent artery narrowing with brain infarction after aneurysm surgery.13 In addition, a muslin-induced intracranial sterile abscess has also been reported,3,18 and there have been several case reports about MR imaging...
Muslin-induced foreign body inflammatory reactions

features of muslinoma and muslin-induced optic arachnoiditis. However, extensive studies of the radiological findings in patients with muslin-induced complications including parent artery narrowing or intracranial abscess have not been performed. Many clinicians and radiologists are unfamiliar with these rare complications after neurosurgical wrapping of intracranial aneurysms and it is crucial that the imaging features of a muslinoma and its induced complications be recognized so that the patient can receive a proper diagnosis and adequate treatment.

In the present study, we describe radiological features in 5 patients who received diagnoses of muslinoma and muslin-induced foreign body inflammatory reactions on the basis of MR imaging and cerebral angiography. The clinical course, outcome, and follow-up in these patients is also described.

Methods

Patient Selection and Imaging Analysis

This study included 5 consecutive patients who received a final clinical and imaging diagnosis of a muslinoma between June 2005 and May 2008. Medical records were reviewed for laboratory findings, treatment, and response to the treatment, and the MR imaging features in the patients were retrospectively analyzed for the presence of the following: muslinomas, brain parenchymal edema, arachnoiditis or an intracranial abscess, optic neuropathy, and acute cerebral infarction. The presence of a regrowth or residual aneurysm, and any postoperative changes in the parent arteries were analyzed on cerebral angiography images. We also reviewed follow-up MR images after clinical stabilization.

Our institutional review board approved this retrospective evaluation of patient images and medical records and did not require informed consent.

Magnetic Resonance Imaging Protocol

All patients underwent MR imaging with a 1.5-T MR imaging system (Sonata, Siemens; Signa HDx and Signa Excite, GE Medical Systems), a 3-T MR imaging system (Signa Excite, GE Medical Systems), and a 1-T system (Magnetom, Siemens). Spin echo T1-weighted, T2-weighted fast spin echo, and fast FLAIR images were obtained. Enhanced T1-weighted images with the administration of 0.1 mmol/kg gadopentetate dimeglumine (Magnevist, Schering) were also obtained in all patients. Echo planar DW images were available from the initial MR imaging examinations in all 5 patients. The MR imaging parameters were as follows: 450–466/12/2 (TR/TE/number of excitations) for spin-echo T1-weighted images; 3666–4000/96–104/1–2/7 (TR/TE/number of excitations/echo train) for fast spin-echo T2-weighted images; and 5000–10,000/110–155/2000/1/7 (TR/TE/TI/number of excitations/echo train) for fast FLAIR images. Other parameters included a section thickness of 5–6 mm with a 1.5-mm gap, 240-mm field of view, and a 256 x 192 matrix. Diffusion weighted images were obtained with a single-shot, spin-echo echo-planar pulse sequence with 2 diffusion sensitivity values of 0 and 1000 second/mm² along all 3 orthogonal axes.

Results

Patients, Clinical Findings, and Outcomes

During a 3-year period, we performed a total of 147 aneurysm operations in 141 patients. These operations included 90 clipping procedures, 54 clipping and wrapping, and 3 wrapping only. Fibrin adhesives were applied onto the wrapped aneurysm in 41 of 57 patients. Of 57 patients who underwent aneurysm wrapping with muslin gauze, muslinomas developed in 5 patients (8.8%). Table 1 shows the clinical history and findings in these 5 patients with muslinomas. There were 3 men and 2 women with a mean age of 56 years (range 51–64 years). Two aneurysms were located in the anterior communicating artery, 2 at the MCA bifurcation, and 1 at the M segment. One patient (Case 4) presented with subarachnoid hemorrhage caused by a ruptured aneurysm, while the other patients had incidentally detected aneurysms. All patients underwent a pterional craniotomy with gauze reinforcement of the aneurysms after surgical clip placement. In 1 patient (Case 5), coil embolization of the aneurysm was subsequently performed for a residual neck. Aneurysms were covered with papaverine-soaked gauze and human fibrin adhesives were applied. Tisseel Kit (Baxter) was used in the patient in Case 1, and Tissucol Duo Quick (Baxter) was used in all other cases.

All patients were discharged from the hospital with no neurological signs. After discharge, however, all 5 patients experienced various clinical courses including headache, optic neuropathy, and motor weakness which arose 2–16 months postoperatively. On readmission to hospital, MR imaging was immediately performed in all patients, including DW images, and additional cerebral angiography in 4 patients. The mean interval from surgical clipping and wrapping of an aneurysm to the initial presentation of symptoms was 11.2 months (range 2–16 months). Two patients presented with headaches (Cases 2 and 5), 1 patient with a progressive visual field defect over 6 months (Case 1), 1 patient with suddenly decreased visual acuity (Case 4) and 1 patient with sudden right-side weakness (Case 3). No patient was febrile and laboratory data demonstrated no increase in erythrocyte sedimentation rate or C-reactive protein, although 1 patient (Case 3) had mild leukocytosis. A CSF study undertaken in 2 patients (Cases 4 and 5) demonstrated no infectious conditions; an infectious origin was therefore deemed unlikely.

Three patients (Cases 2, 4, and 5) underwent conservative treatment with steroid therapy, and 1 patient (Case 1) did not receive any treatment. No patient was given antibiotic therapy. These patients fully recovered after a few months. Despite combination treatment of dexamethasone, clopidogrel, and heparin, in 1 patient (Case 3) with delayed segmental narrowing of the parent artery an acute infarction developed in the upper basal ganglia area at the time of the initial presentation.

The mean clinical follow-up period was 26 months (range 13–42 months). Two patients (Cases 3 and 4) had reactivated inflammation with evident latent periods of clinical and radiological improvement after the first episode over a 24-month follow-up period. The patient in
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Aneurysm Location</th>
<th>Procedure</th>
<th>Postop Time to Symptom Onset (mos)</th>
<th>Postop Clinical Symptoms</th>
<th>Lab Findings at Symptom Onset</th>
<th>CSF Exam at Symptom Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>WBC Count (4000–10,000/ml)</td>
<td>% of SNC (50–75%)</td>
</tr>
<tr>
<td>1</td>
<td>51, M</td>
<td>ACoA</td>
<td>clip &amp; wrap</td>
<td>15</td>
<td>visual field defect</td>
<td>8500</td>
<td>50.20</td>
</tr>
<tr>
<td>2</td>
<td>53, M</td>
<td>rt MCA bifurcation</td>
<td>clip &amp; wrap</td>
<td>16</td>
<td>headache</td>
<td>7900</td>
<td>57.80</td>
</tr>
<tr>
<td>3†</td>
<td>64, M</td>
<td>lt MCA, M1</td>
<td>clip &amp; wrap</td>
<td>2; 22</td>
<td>rt side weakness; headache</td>
<td>10,500; NA</td>
<td>67; NA</td>
</tr>
<tr>
<td>4†</td>
<td>52, F</td>
<td>ACoA (ruptured)</td>
<td>clip &amp; wrap</td>
<td>13; 24</td>
<td>decreased visual acuity (both times)</td>
<td>5390; NA</td>
<td>63.3; NA</td>
</tr>
<tr>
<td>5</td>
<td>58, F</td>
<td>rt MCA bifurcation</td>
<td>clip &amp; wrap, then CE for residual neck</td>
<td>10</td>
<td>headache</td>
<td>5000</td>
<td>72; NA</td>
</tr>
</tbody>
</table>

* All patients experienced improvement of symptoms. Values in parentheses represent normal ranges. Abbreviations: ACoA = anterior communicating artery; CE = coil embolization; CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; lympho = lymphocyte; NA = not assessed; SNC = segmented neutrophil count; WBC = white blood cell.
† Cases 3 and 4 each had 2 episodes of clinical and radiological deterioration. First episode data are separated from second episode by a semicolon.
Muslin-induced foreign body inflammatory reactions

Case 3 had 2 episodes of clinical deterioration: the first episode with right side weakness at 2 months after surgery, and the second episode with headaches at 22 months postoperatively. The patient in Case 4 showed the same pattern with 2 episodes manifesting as optic neuropathy at 13 and 24 months after surgery.

Imaging Findings in Muslinomas and Muslin-Induced Foreign Body Inflammatory Reactions

In all patients, MR images showed the muslinomas as perianeurysmal enhancing masses; these were fairly well-circumscribed and had lobulated contours. The masses closely abutted the clipped aneurysms and showed central hypointensity with a susceptibility artifact on T2-weighted images and peripheral rim enhancement on contrast-enhanced T1-weighted images. The central hypointense area on T2-weighted images did not show high signal intensity on DW images, as would be expected with an abscess (Figs. 1A–C, 2C, F, and I, and 3A, C, and E). The mean maximum diameter of the muslinomas was 2.53 cm (range 1.4–3.2 cm). All muslinomas were surrounded by parenchymal vasogenic edema during the symptomatic period (Figs. 1A and 2D).

In 3 patients (Cases 1–3), the CSF spaces adjacent to the muslinoma and vasogenic edema were entrapped and seen with rim enhancement and central hyperintensities on T2-weighted and DW images, which is consistent with adhesive arachnoiditis or a sterile intracranial abscess (Fig. 1A–C). In the patient in Case 3 with recurrent inflammation, arachnoiditis with a sterile abscess around the muslinoma was seen on MR images obtained during the second episode, but not on the MR images from the first episode (Fig. 2G, I, and K). Two patients with the anterior communicating artery aneurysms had optic neuropathy (Cases 1 and 4), which was seen with localized hyperintensity along the optic chiasm or optic tract on T2-weighted images (Fig. 3B and F).

Four of 5 patients underwent postoperative cerebral angiography on the day of readmission because of their symptoms. All 4 patients who underwent cerebral angiography had completely obliterated aneurysms with no evidence of a residual aneurysmal neck or vasospasm. However, on cerebral angiography, 2 patients (Cases 2 and 3) were noted to have severe stenosis or segmental luminal narrowing of the parent vessels of the wrapped aneurysm (Fig. 2B). In the patient in Case 3, delayed segmental MCA narrowing resulted in an acute infarction in the upper basal ganglia area as seen on DW images at the first episode with clinically apparent weakness on the right side (Fig. 2E). A summary of characteristics of the muslinomas and induced lesions on MR images is shown in Table 2.

Fig. 1. Case 1. A–D: Images obtained at presentation in a 51-year-old man with a visual field defect after anterior communicating artery aneurysm surgery; T2-weighted (A), contrast-enhanced T1-weighted (B), DW (C), and angiographic (D) images are shown. The muslinoma (white arrow) is seen as a rim-enhancing conglomerated mass with central hypointensity or a susceptibility artifact on the T2-weighted and DW images. Adhesive arachnoiditis or intracranial abscess (open arrows) is seen with rim enhancement, but also with central hyperintensity on both T2-weighted and DW images. Note the extensive surrounding brain parenchymal edema in the left inferior frontal area. No residual sac or parent artery narrowing is demonstrated on angiography. E and F: Follow-up images obtained 1 month postoperatively (T2-weighted [E] and contrast-enhanced T1-weighted [F] MR images) reveal that the muslinoma (white arrows) remains, but that the adhesive arachnoiditis has nearly disappeared and the adjacent parenchymal edema has decreased compared with the previous images.
Fig. 2. Case 3. Images obtained in a 64-year-old man with sudden-onset right-side weakness at the first episode, and severe headache at the second episode. A: Preoperative angiogram shows a small aneurysm of the left MCA (black arrow). White arrow indicates the frontosylvian branch of the left MCA. B: Two-month postoperative angiogram demonstrates segmental narrowing (white arrow) at the frontosylvian branch of the left MCA. C–E: Contrast-enhanced T1-weighted image (C), T2-weighted image (D), and DW image (E) obtained 2 months after surgery. An inflammatory enhancing mass is present (C, open arrow), with surrounding vasogenic edema (visible in panel D) and acute ischemic infarction (see panel E). F–H: Images acquired 4 months after the first MR images were obtained, demonstrating persistent muslinoma (F, open arrow). Contrast-enhanced T1-weighted image (F) and T2-weighted images (G and H). The parenchymal edema has disappeared (G), and focal tissue loss due to infarction is newly found (H). I–K: Images obtained 17 months after the second set of MR images (22 months postoperatively). Contrast-enhanced T1-weighted (I), T2-weighted (J), and DW images (K) demonstrate that the muslinoma has not significantly changed (I, open arrow), and the presence of adhesive arachnoiditis or an intracranial abscess (white arrow) and parenchymal edema is again demonstrated.
Muslin-induced foreign body inflammatory reactions

Follow-Up Images

Follow-up MR images were obtained in all patients. The average imaging follow-up period was 6.8 months after symptom onset (range 1–24 months). Follow-up MR images revealed a markedly decreased extent or absence of vasogenic edema, adhesive arachnoiditis, sterile intracranial abscess, and optic neuropathy in all patients. The muslinomas were seen with a decreased size or thinner peripheral enhancement, but persisted on follow-up images even when the patient showed clinical improvement. The patient in Case 4 experienced reactivated inflammation, and on follow-up imaging the muslinomas increased in size and showed more prominent peripheral enhancement (Fig. 3A, C, and E). In the patient in Case 3, who experienced an acute infarction during the first episode, focal tissue loss at the upper basal ganglia area was seen on follow-up images (Fig. 2H). Follow-up cerebral angiography was not performed after symptom improvement, and changes in parent arteries with narrowing could not be assessed.

Discussion

In the present study, we have demonstrated clinical and radiological features of muslin induced foreign-body reactions in patients with history of surgical clipping and wrapping of intracranial aneurysms.

Muslin (cotton gauze) is commonly used as a surgical material for the wrapping of aneurysms. It is known to produce a dense fibrotic reaction, thus strengthening fragile vessel walls. However, like other surgical materials, muslin can cause various inflammatory reactions. In addition to muslin gauze, coating adhesives are applied to adhere the gauze to the vessel and prevent regrowth or rupture of clipped aneurysms. Like the muslin gauze itself, however, these adhesives may induce an excessive foreign body response such as intimal proliferation. Yasuda et al. reported that when cellulose cotton sheets and fibrin adhesives were used in rabbits to wrap artificially induced intracerebral aneurysms, intimal thickening was observed after 8 weeks, resulting in luminal stenosis after 12 weeks. Therefore, combined use of wrapping and coating adhesives may carry the potential to further drive granuloma formation and intimal thickening due to inflammatory reactions.

In this retrospective review, we found unique imaging features in patients who presented with delayed neurological deterioration after craniotomy and aneurysm clipping and wrapping. Magnetic resonance images revealed multiple conglomerated rim-enhancing masses around the surgical clips with relative hypointensity on T2-weighted images and no hyperintensity on DW images. Bhatti et al. also suggested that solid hypointensity on T2-weighted images was a feature that distinguishes muslinomas from abscesses, but the authors did not obtain DW images.

In our series of muslinomas, all patients underwent MR imaging with the acquisition of DW sequences, which provided additional information on muslin-induced complications. We also obtained follow-up images, from which we learned more about the natural course of muslin-induced foreign body reactions.

A muslinoma may be accompanied by adhesive arachnoiditis or an intracranial abscess. This finding manifests as entrapped CSF spaces with thin rim enhancement and high signal intensity on DW images. To the best of our
knowledge, ours is the first report of muslinomas with adjacent adhesive arachnoiditis or intracranial abscess on DW images. Our cases of adhesive arachnoiditis or intracranial abscess were conservatively managed without antibiotic therapy or surgical intervention, but the patients recovered, from which we conclude that the muslinomas and abscesses were sterile. No microbiological infection was demonstrated in previous reports, except for a single case in which a microbiological examination revealed the presence of Staphylococcus epidermidis.

In the present study, muslin-induced optic neuropathy occurred in parasellar aneurysms located near the optic nerve or other cranial nerves. Repka et al. have suggested that the causes of optic neuropathy are various, including ischemia, compression, and inflammation. More than 30 cases of muslin-induced optic neuropathy have been reported, and the authors of a few reports have demonstrated MR imaging findings of optic neuropathy. In the 2 cases of optic neuropathy in our study, MR images demonstrated edematous changes in the optic chiasm or optic tract that were distinguishable from the muslinoma itself. On the basis of our study and retrospective review of previous reports, we believe that muslin-induced optic neuropathy may occur in parasellar aneurysms near the optic nerve or chiasm in anterior or posterior communicating arteries or the distal internal carotid artery.

Authors of previous reports on muslin-induced optic neuropathy after parasellar aneurysm clip placement have described coexisting parent artery narrowing seen on angiography. However, despite its clinical significance, parent artery narrowing has received little clinical attention. Kurita and associates observed delayed parent artery narrowing in 2 patients who underwent circumferential wrapping and coating of aneurysms, and suggested that this rare phenomenon caused acute ischemic infarction 3 and 6 months after surgery. Severe symptomatic parent artery narrowing that may cause ischemic infarction is rare but possible. In our series, delayed luminal narrowing (demonstrated on cerebral angiography) developed at the wrapped portion of the parent artery in 2 patients with aneurysms at a bifurcation or M1 segment of the MCA. One patient with delayed segmental stenosis of the parent artery had an acute cerebral infarction in the region of the wrapped aneurysm.

In our review of the literature we found that 24 of 30 reported cases of muslin-induced foreign body inflammatory reactions were in women; however, there was no female predominance in the present study. The time from surgical repair of the aneurysm to initial presentation has been previously reported as 1–54 months, and was 2–16 months postoperatively in the present study. Two patients in our study showed clinical and radiological waxing and waning inflammation during the follow-up period of 22–24 months. To the best of our knowledge, reactivated inflammation in patients with muslinomas or muslin-induced foreign body reactions has not been reported. Muslin-induced optic neuropathy and adhesive arachnoiditis was reversible, but the rim-enhancing muslinomas remained persistent on follow-up images. Ischemic infarction resulting from parent artery narrowing was by definition irreversible, but reversibility of the parent artery narrowing could not be assessed. Muslinoma-induced lesions show a relatively self-limiting course, and some patients showed relapsed inflammation, on the basis of which we believe an autoimmune pathophysiology may play a role.

Questions concerning the efficacy of specific therapies for muslinomas have not been completely answered. Many therapeutic approaches have been tried, including surgical exploration, corticosteroid therapy, immunosuppressants, antibiotic therapy, and observation, but the efficacy of any specific treatment still remains controversial. There have been reports of spontaneous improvement, as well as cases in which the condition was unresponsive to any of the aforementioned treatments. In our series, the muslinomas were conservatively managed, and the patients temporarily or finally recovered. One patient (Case 1), who did not receive any treatment, showed spontaneous clinical improvement and partial MR imaging improvement, and 4 patients who received corticosteroid therapy showed dramatic symptomatic improvement. Despite the limited number of cases in the present study, we believe that surgical exploration or adhesiolysis should be considered only in cases unresponsive to conservative treatment.

In consideration of the various treatment options for intracranial aneurysms, wrapping with muslin should be considered with greater caution because of the rare but serious possible complication of muslinoma or a muslin-induced foreign body reaction. Alternative endovascular or microsurgical treatment options should be considered.

### Table 2: Summary of muslinoma and muslin-induced foreign body reactions as depicted on MR images and angiography

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Muslinoma</th>
<th>Adjacent Brain Parenchymal Edema</th>
<th>AA or ICA</th>
<th>Optic Neuropathy</th>
<th>PA Narrowing on Angiography</th>
<th>Acute IF of PA Territory</th>
<th>FU Interval Post-Tx or MCD (mos)</th>
<th>Imaging Improvement Demonstrated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>1</td>
<td>yes</td>
</tr>
<tr>
<td>2</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>24</td>
<td>yes</td>
</tr>
<tr>
<td>3</td>
<td>yes; yes</td>
<td>yes; yes</td>
<td>no; yes</td>
<td>no</td>
<td>yes; NA</td>
<td>yes; no</td>
<td>4; 3</td>
<td>yes</td>
</tr>
<tr>
<td>4</td>
<td>yes; yes</td>
<td>yes; yes</td>
<td>no; yes</td>
<td>no</td>
<td>NA; NA</td>
<td>no; no</td>
<td>1; NA</td>
<td>yes</td>
</tr>
<tr>
<td>5</td>
<td>yes</td>
<td>yes</td>
<td>no; no</td>
<td>no</td>
<td>no</td>
<td>no</td>
<td>1</td>
<td>yes</td>
</tr>
</tbody>
</table>

* Cases 3 and 4 each had 2 episodes of clinical and radiological deterioration. First episode data are separated from second episode by a semicolon. The patient in Case 4 did not undergo follow-up imaging after the second episode. Abbreviations: AA = adhesive arachnoiditis; IF = infarction; ICA = intracranial abscess; MCD = maximal clinical deterioration; PA = parent artery.
Muslin-induced foreign body inflammatory reactions

Conclusions

In conclusion, despite the paucity of published medical reports on the subject, muslin-induced foreign body reactions should be considered in the differential diagnosis when a patient with a history of aneurysm wrapping presents with new neurological symptoms and the characteristic MR imaging findings. Clinicians and radiologists should be aware that muslin wrapping of intracranial aneurysms may induce rare but serious complications including muslinomas, muslin-induced optic neuropathy, adhesive arachnoiditis, or a sterile intracranial abscess and parent artery narrowing and resultant acute ischemic infarction. In patients with a muslinoma or muslin-induced complications, careful clinical and neuroimaging follow-up is required for proper treatment and understanding the natural course of the disease.

Disclaimer

The authors do not report any conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

References


Current affiliations for Dr. Eunhee Kim: Seoul National University College of Medicine, and Samsung Medical Center, Sungkyunkwan University College of Medicine, Seoul, Korea.

Please include this information when citing this paper: published online September 4, 2009; DOI: 10.3171/2009.7.JNS081625.

Address correspondence to: Eunhee Kim, M.D., Department of Radiology, Seoul National University College of Medicine, 101 Daehangno, Jongno-gu, Seoul, 110-744, Korea. email: kimeunheekh@gmail.com.