Gauze-induced granuloma ("gauzoma"): an uncommon complication of gauze reinforcement of berry aneurysms

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Incompletely and even adequately clipped berry aneurysms are often reinforced with finely shredded gauze. In seven female patients this practice led to a series of events including headache, pyrexia, seizures, cranial nerve deficits, endocrinopathy, cerebrospinal fluid pleocytosis, and an enhancing mass demonstrated by computerized tomography at the aneurysm site. One patient with blindness, hydrocephalus, and panhypopituitarism died and was examined at autopsy.

Three additional female patients have been identified in the literature with similar case histories. It is suggested that in these patients the gauze induced a foreign-body granuloma, accompanied by progressive occlusion of neighboring small arteries. It would seem prudent to reserve gauze reinforcement for aneurysms that cannot be securely obliterated surgically.

KEY WORDS • foreign-body granuloma • gauze • aneurysm, berry • wrapping

The consequences of postoperative rupture of an insecurely obliterated berry aneurysm have encouraged surgeons to use every means at their disposal to secure the fundus. One popular strategy consists of cutting a piece of gauze sponge into approximately 1-mm fragments with scissors, suspending it in papaverine hydrochloride injection solution and then applying a thin layer to cover either the neck alone or both the neck and fundus of the aneurysm after clipping. This practice is well established. In 1933, Dott, in performing the first successful operation on a ruptured aneurysm of the middle cerebral artery (MCA), reported wrapping the aneurysm with muscle hammered into gauze. The patient did well and died from myocardial infarction 12 years later. After introducing routine surgery for the obliteration of berry aneurysms in the 1950's, Botterell, et al., often used either fine strips of gauze or fragments of finely shredded gauze to reinforce clipped aneurysms. Drake and Vanderlinden used gauze reinforcement when an aneurysm was incompletely clipped. Gillingham proposed wrapping all MCA aneurysms after completely dissecting the fundus.

Sachs showed that wrapping of aneurysms with muscle afforded little reinforcement but that the addition of cotton was effective. Taylor and Choudhury recommended wrapping aneurysms with postage stamp-sized pieces of gauze on the basis of a 2- to 15-year follow-up study of 35 consecutive patients. One of these patients died of unrelated intracerebral hemorrhage 1 year after wrapping; autopsy revealed the wrapped aneurysm encased in a "thick fibrotic wall." At the 1986 meeting of the Ontario Medical Association Section of Neurosurgery, where these findings were reviewed, the majority of those present considered such gauze reinforcement routine. However, recurrent bleeding has been reported even when an aneurysm is reinforced with gauze.

This report describes six female patients and alludes to one more, also female. All underwent gauze reinforcement of berry aneurysms after surgical clipping, and all followed an often stuttering clinical course including pyrexia, headache, cranial nerve deficits, endocrinopathy, or epilepsy. One patient died 4 years postoperatively with blindness, hydrocephalus, and panhypopituitarism; the autopsy findings are reported. Three additional cases, also all female, are identified in the literature. The pathogenesis and implications are reviewed.
Case Reports

Case 1

This 63-year-old woman with a long history of migraine was admitted to the Toronto General Hospital on July 26, 1982, with the sudden onset of occipital headache, a 15-minute episode of blurred vision, and meningismus, but no other abnormal neurological symptoms or signs. The cerebrospinal fluid (CSF) was bloody. A computerized tomography (CT) scan revealed blood in the left sylvian fissure and around the left temporal lobe. Four-vessel angiography showed an irregular 15-mm right ophthalmic artery aneurysm and an 8-mm right MCA aneurysm.

At surgery 11 days after admission, the right ophthalmic artery aneurysm, which was adherent to the undersurface of the optic chiasm, was visualized through a right frontal craniotomy after removal of the anterior clinoid process. Just as the aneurysm was being isolated, it ruptured. Bleeding was controlled with two temporary small straight Sugita clips until a large permanent fenestrated Sugita clip could be applied, encircling and flush with the internal carotid artery. The aneurysm was surrounded with shredded gauze which had been suspended in papaverine solution. Immediately postoperatively, the patient complained of an unusually severe headache which persisted, nausea, and vomiting. She developed progressive right ptosis, myosis, and lateral strabismus. A CT scan showed no new abnormality and she was discharged home.

On August 25, 1982, the patient was readmitted and uneventful elective clipping of the unruptured MCA aneurysm was carried out through a pterional approach. Again the aneurysm was surrounded with shredded gauze. By December 14, 1982, although she still had headache and felt tired, the right eye signs had lessened and vision was normal. On January 18, 1983, she had a severe left-sided headache. When examined on June 9, 1983, her headache persisted; her vision was normal, and, although she had stable exotropia, she could fuse the images of the two eyes. On June 14, 1983, she had a 2-hour episode of blurred vision in the right eye following which she was found to have a right superior quadrantanopsia. Repeat CT was unaltered. In July, she experienced an attack of severe right supraorbital pain with bilateral blindness lasting several hours. Afterwards, she was able to perceive only light with the right eye and had the illusion of looking through a veil with the left. Four-vessel angiography on November 25 showed narrowing of the A1 segment of the right anterior cerebral artery, without evidence of residual aneurysm.

On January 20, 1984, right optic atrophy was noted and, by May 22, vision in the right eye had deteriorated further but the right lateral strabismus and ptosis had not changed. On June 26, after the patient had experienced sudden loss of vision in the left eye while bowling, left optic atrophy and a left temporal field defect were noted. On March 13, 1985, the right eye was found to be completely blind; there was a temporal hemianopsia on the left. On the same day a CT scan showed an enhancing mass (Fig. 1) surrounding the chiasmatic region, and an arteriogram on April 15 showed elevation of the further narrowed right A1 segment. On May 9, the optic chiasm was reexplored, revealing a mass of adherent fibrous material which elevated the right optic nerve and chiasm and separated them from the internal carotid artery; it extended posteriorly beyond the chiasm. The mass was partially excised, sacrificing the encased frontopolar artery. The right optic nerve was so distorted and incorporated into the mass that only its inferior border could be identified, whereas the left optic nerve could be partially freed (Fig. 2). The excised material consisted of vascular hyalinized connective

FIG. 1. Case 1. Computerized tomography scan showing enhancing presellar mass (arrow).

FIG. 2. Case 1. Operative diagram showing marked distortion revealed at surgery of the optic chiasm and nerves by gauze-induced granuloma.
Granuloma from gauze reinforcement of aneurysms

tissue, containing birefringent filamentous foreign material and acute and chronic inflammatory cell infiltrates, with lymphocytes, plasma cells, neutrophils, foamy macrophages, and numerous multinucleated foreign-body giant cells (Fig. 3).

When examined on June 25, 1985, the patient complained of burning dysesthetic pain below both knees. She exhibited excessive weight gain and was found to have distal hypegesia in all four limbs. She was admitted to a hospital in Kingston, Ontario, in December, 1985, with a 4-month history of confusion, nausea, ataxia, leg weakness, urinary incontinence, and increasing blindness in the left eye in addition to the previous problems. A CT scan revealed hydrocephalus, and a ventriculoperitoneal shunt was inserted on February 19, 1986. Her confusion and ataxia improved but, on May 1, 1986, while awaiting discharge she suddenly lost consciousness for 1 minute, showing fixed dilated pupils, hypotension, and tachycardia. She regained consciousness and became oriented but remained hypoxic. She then had a second similar episode with a cardiac arrest and remained hypotensive and bradycardiac. She regained the ability to move all limbs but was hyperreflexic with upgoing toes. She developed polyuria with a serum osmolality of 320 mOsm/kg and a urine osmolality of 323 mOsm/kg; she was treated with vasopressin. On May 6, 1986, she developed apneic spells and gradually deteriorated, dying on May 12, 1986.

At autopsy, an ovoid mass of dense fibrous tissue measuring 2 cm in greatest dimension surrounded the clip on the right MCA aneurysm. A similar densely adherent mass encircled the right ophthalmic and internal carotid arteries, optic nerve, third nerve, and optic chiasm, and extended to the hypothalamus adjacent to the pituitary stalk. The right MCA and ophthalmic artery masses contained dense fibrous tissue, foci of foreign-body granuloma, and abundant refractile material. The optic chiasm, nerves, and tracts were severely gliotic, fibrotic, and demyelinated. Fibrosis extended into the hypothalamus and pituitary stalk.

Case 2

This 39-year-old woman suddenly developed an occipital headache on December 17, 1984, and was treated with analgesics. Because of persistent headache, she was admitted to the Toronto General Hospital where neurological examination and a CT scan were normal. The CSF was bloody and four-vessel angiography revealed a 1-cm bilobed anterior communicating artery aneurysm without vasospasm. On December 26, 1984, the aneurysm was clipped with a large and a small straight Sugita clip, and its neck was reinforced with finely shredded gauze which had been suspended in papaverine solution.

Postoperatively, the patient complained of generalized headache, attributed to a right-sided 3-mm subdural hematoma demonstrated by CT scan, without mass effect. In July, 1985, she complained of low-grade fever, fuzzy peripheral vision, and "waves of light" in the right eye, worse at night; examination revealed a left upper temporal quadrantanopsia. A CT scan showed an enhancing right suprasellar mass in addition to the small subdural effusion. By January, 1986, although she still complained of hazy vision in the right eye, the visual field defect and other symptoms had resolved.

Case 3

This 71-year-old woman presented to the Toronto General Hospital on May 16, 1982, with sudden fronto-occipital headache, vomiting, drowsiness, hypertension (190/100 mm Hg), and meningismus, but no other neurological deficit. She became fully alert over 48 hours, and four-vessel angiography on the 3rd day showed a 9-mm right posterior communicating artery aneurysm without vasospasm. On May 20, 1982, this was clipped with a straight Sugita clip applied flush with and parallel to the internal carotid artery. Shredded papaverine-soaked gauze was placed around the neck.

In September, 1983, the patient returned with a 2-month history of bifrontal headache and low-grade fever. A CT scan showed an enhancing right suprasellar mass but arteriography revealed no aneurysm. In December, 1983, she began to notice painless right ptosis which progressed over the next 5 months to complete oculomotor palsy and then improved again over the next year until she could partially open her right eye. Paresis of the superior, medial, and inferior rectus muscles persisted. In March, 1985, while brushing her teeth, she lost consciousness briefly without residual neurological or CT changes. A right anterior temporal electroencephalographic (EEG) focus was identified but, under treatment with diphenylhydantoin sodium, she had no further seizures; the third nerve palsy still persisted at her latest follow-up examination in March, 1987.
Case 4

This 31-year-old woman experienced a sudden excruciating fronto-occipital headache and transient loss of consciousness on March 10, 1986. Her CSF was bloody, a CT scan was normal, and angiography was equivocal. Repeated angiography on March 15, 1986, showed a 16-mm left MCA aneurysm. She had persisting headache without other abnormalities. The same day the aneurysm was partially clipped with a curved Sugita clip; two residual small blebs of aneurysm were obliterated with a right-angled fenestrated clip, sparing all parent vessels. The clipping was reinforced with finely shredded papaverine-soaked cefotaxime gauze.

On April 15, 1986, the patient suffered the first of six epileptic events, each lasting less than a minute, during which she smelled “rotten eggs.” In May, 1986, she noted a pounding occipitotemporal headache, worse when upright, and intermittent low-grade fever. On June 19, 1986, she was readmitted with a temperature of 38.8°C, but no neurological deficit; a CT scan showed a multiloculated 1 × 2-cm enhancing mass in the left Sylvian fissure just above the clips with surrounding edema (Fig. 4). There was a left temporal sharp-wave EEG focus. She was treated with intravenous cloxacillin and cefotaxime, although no infectious organisms were ever identified. Further generalized seizures were controlled, first with diphenylhydantoin sodium, then (because of a skin rash) with carbamazepine. Serial CT scans demonstrated diminution in size of both the edema and the mass. Three months later she was symptom-free.

Case 5

This 65-year-old woman presented to the Toronto General Hospital on March 30, 1986, with sudden, intense occipital headache, meningismus, drowsiness, and hypertension (210/100 mm Hg) without other neurological abnormality. A CT scan demonstrated blood in the Sylvian fissures, worse on the right, with mild enlargement of the lateral and third ventricles; four-vessel angiography revealed a 20-mm right MCA bifurcation aneurysm. The next day the aneurysm was exposed and dissected through a right superior temporal cortical incision with the help of a temporary clip on the M1 segment. The neck and proximal portion of the aneurysm were calcified, broad, and ill-defined, so that a clip could not be placed across the neck without compromising the parent vessels. The aneurysm was therefore clipped distally with right-angled fenestrated Sugita clips, sparing all vessels, and the neck was surrounded with shredded papaverine-soaked cefazidime gauze.

On August 31, 1987, the patient presented with lethargy, confusion, a fever of 38.7°C, malaise, chills, fatigue, headache, photophobia, blurred vision, and urinary frequency, but no focal neurological signs. A CT scan with no contrast enhancement demonstrated moderate hydrocephalus and a low-density right frontotemporal lesion compressing the right frontal horn. Lumbar puncture revealed an opening pressure of 13 mm H2O and slightly turbid CSF, with 4150 white cells (80% of which were neutrophils), 160 red cells, no organisms, a glucose level of 2.2 mg/dl, and a protein level of 1.36 gm/dl. The patient was treated with intravenous cefazidime, vancomycin, and tobramycin; her mentation improved within 24 hours and her fever disappeared after 2 days. A follow-up CT scan showed a 2.5 × 1.5-cm enhancing mass above the clip, elevating the right frontal horn and associated with right frontal lobe edema extending into the superior right temporal lobe (Fig. 5). Abscess was suspected so that intravenous Flagyl (metronidazole) with prophylactic diphenylhydantoin sodium was added. Blood cultures showed no growth although urine cultures grew coliform bacteria and Streptococcus. In October, 1987, and October, 1988, CT scans showed no change but the patient remained asymptomatic.

Case 6

This 35-year-old woman with three previous bouts of severe headache was admitted to the Wellesley Hospital on May 14, 1987, with photophobia and meningismus. Shortly before, she had suffered the onset of severe generalized headache while playing baseball, when her legs collapsed beneath her. A CT scan showed moderate cisternal and interhemispheric subarachnoid blood, and arteriography demonstrated an unusual aneurysm arising from a wide neck at the origin of an anomalous accessory recurrent MCA branch from the proximal A1. There was a fusiform change in the left
mirror position. On the 10th day of an intended 14-
day presurgery delay, during which she remained in
neurological grade 1 and was treated with Amicar (ami-
nocaproic acid, 1 gm/hr), her headache recurred, her
temperature and white blood cell count rose, while her
blood pressure remained normal. Arteriography on Day
11 demonstrated enlargement of the aneurysm, the
appearance of distinct bilobularity, but no spasm. The
aneurysm was exposed, dissected, and secured with a
curved side-angled Sugita clip through a right pterional
craniotomy on the same day. Since it proved impossible
to safely secure a residual small thin-walled pulsatile
segment 1 mm in length, the latter was covered with
papaverine-soaked shredded gauze, and the patient was
discharged without neurological deficit 12 days post-
operatively. Within 10 days she began to complain of
repeated olfactory hallucinations which were controlled
with 300 mg diphenylhydantoin sodium.

By August, 1987, the patient gradually became aware
of a left-sided defect of the visual field and visual
hallucinations in the hemianoptic field in the form of
elemental shapeless forms and also highly organized
visual material described as “an extension of reality.”
A smooth concrete walk appeared to her as a distorted
surface extending into the hemianoptic field while a
lawn appeared like rolled sod in the same area. Exam-
ination revealed an almost completely congruous left
homonymous hemianopsia; a CT scan was normal. In
October, 1987, she complained of further failure of
vision in the right eye where she was found to be capable
only of finger-counting; vision was 20/30 on the left,
associated with a complete left homonymous hemi-
anopsia and a very large right central scotoma. Oligo-
menorrhea supervened and neuroendocrine investiga-
tion revealed a follicle-stimulating hormone (FSH) level
of 5 IU/liter, a luteinizing hormone (LH) level of less
than 3 IU/liter, but normal FSH and LH responses
after administration of LH-releasing hormone. Subse-
quently, normal menses resumed. A CT scan on May
20, 1988, demonstrated a large enhancing right para
sellar mass, while right carotid arteriography showed
satisfactory clipping of the aneurysm and preservation
of the parent vessel. The patient’s visual status has
remained stable over the subsequent year.

Comment

One further patient, also female, has been brought to
our attention. She developed symptoms and a mass was
visualized on CT at the site of gauze application to a
clipped, ruptured berry aneurysm in another Toronto
hospital.

Discussion

Within 17 months after reinforcement of aneurysmal
clipping with shredded gauze, seven female patients
developed some or all of the following symptoms: head-
ache, low-grade fever, epilepsy, endocrinopathy, CSF
pleocytosis, cranial nerve deficit, and a localized en-
hancing mass on CT. In one patient such a mass was
partially removed, although she subsequently died with
headache, blindness, hydrocephalus, and panhypopitui-
tarism. Pathological examination of both the surgical
and autopsy specimens revealed a dense fibrotic gran-
ulomatous foreign-body reaction containing birefrin-
gent material at the sites of the gauze implantation.

The only relevant publication appears to be a report
by Carney and Oatley of three women who developed
partial blindness 1, 7, and 11 months, respectively, after
unruptured aneurysms at three different sites had been
clipped and wrapped with gauze. One patient experi-
cenced the sudden onset of symptoms. In two cases the
deficits improved, one had fever and CSF pleocytosis,
and none had masses visible with CT. The authors
incriminated an underlying ischemic process with gauze
as a possible culprit.

Table 1 summarizes the clinical features of the six
patients reported here fully and the three patients of
Carney and Oatley (Cases 7, 8, and 9). The significance
of the fact that all were women is unclear. No other
risk factor was identified and none of the patients was
proved to have infection. The aneurysm site and occur-
rence of subarachnoid hemorrhage appear irrelevant.
The delayed onset of symptoms (up to 17 months) sug-
gests a slowly progressive process. Headache in seven
patients, pyrexia in six, CSF pleocytosis in three of the
four whose CSF was studied, and an enhancing CT
mass in six suggest an inflammatory process with event-
tual apparent stabilization within the limited follow-up
period in eight cases.

Epilepsy in five patients was compatible with a focus
near the aneurysm site, while neurological signs could
be attributed to involvement of adjacent structures: the
optic nerves in six, oculomotor nerves in two, and the

Fig. 5. Case 5. Computerized tomography scan showing
an enhancing mass in the right sylvian fissure with surround-
ing edema.
TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age (yrs)</th>
<th>Postop Onset of Symptoms (mos)</th>
<th>Size &amp; Site of Aneurysm</th>
<th>Sudden Onset</th>
<th>Febrile</th>
<th>Cranial Nerve Signs</th>
<th>Epilepsy</th>
<th>CSF Pleocytosis</th>
<th>Headaches</th>
<th>Other</th>
<th>CT Scan Appearance</th>
<th>Apparent Stabilization</th>
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<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>63</td>
<td>0</td>
<td>ruptured 15 mm, rt ophthalmic; unruptured 8 mm, rt MCA</td>
<td>yes</td>
<td>yes</td>
<td>III, II</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>enhancing mass</td>
<td>no, step-wise progression of symptoms; died 45 mos postop</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>39</td>
<td>6</td>
<td>ruptured 10 mm, anterior communicating</td>
<td>no</td>
<td>yes</td>
<td>II</td>
<td>no</td>
<td>?</td>
<td>yes</td>
<td>no</td>
<td>enhancing mass</td>
<td>yes</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>71</td>
<td>16</td>
<td>ruptured 9 mm, rt posterior communicating</td>
<td>no</td>
<td>yes</td>
<td>III</td>
<td>yes</td>
<td>rt temporal lobe focus</td>
<td>yes</td>
<td>no</td>
<td>enhancing mass</td>
<td>yes</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>31</td>
<td>1</td>
<td>ruptured 16 mm, lt MCA</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>enhancing mass</td>
<td>yes</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>65</td>
<td>17</td>
<td>ruptured 20 mm, rt MCA</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>hydrocephalus</td>
<td>enhancing mass yes</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>35</td>
<td>0.3</td>
<td>ruptured, rt anterior cerebral</td>
<td>no</td>
<td>no</td>
<td>II</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>yes</td>
<td>endocrinopathy</td>
<td>enhancing mass yes</td>
</tr>
<tr>
<td>7†</td>
<td>F</td>
<td>37</td>
<td>7</td>
<td>15 mm, ICA (no gauze); 4 mm anterior communicating (gauze); both unruptured</td>
<td>no</td>
<td>no</td>
<td>II, lt eye temporal field defect lobe</td>
<td>no</td>
<td>yes</td>
<td>no</td>
<td>no</td>
<td>negative mass</td>
<td>yes</td>
</tr>
<tr>
<td>8†</td>
<td>F</td>
<td>47</td>
<td>1</td>
<td>3-cm subfrontal meningioma; unruptured 10 mm, rt ICA, not clipped</td>
<td>yes, sudden blindness in rt eye</td>
<td>yes</td>
<td>III, lt eye inferior temporal defect</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>negative mass</td>
<td>yes</td>
</tr>
<tr>
<td>9†</td>
<td>F</td>
<td>37</td>
<td>11</td>
<td>8 × 5 × 7 mm, lt posterior communicating</td>
<td>no</td>
<td>no</td>
<td>II, lt eye nasal field defect</td>
<td>no</td>
<td>?</td>
<td>no</td>
<td>lt MCA infarct</td>
<td>infarct yes</td>
<td></td>
</tr>
</tbody>
</table>

* CSF = cerebrospinal fluid; CT = computerized tomography; MCA = middle cerebral artery; ICA = internal carotid artery.
† Cases reported by Carney and Oatley.²
‡ Partial recovery was obtained in all patients except Case 9.

Related to retained sponges after four instances of abdominal surgery, noting the CT appearance and describing the presence of cotton fibers and/or birefringent fragments in the lesions. Sturdy, et al.,¹³ studied foreign-body granulomas after 32 intraperitoneal, 23 subcutaneous, and six uterine cervical operations and one intra-articular procedure, comparing results with experimental studies in which lint from dry packs, laparotomy sponge, gauze sponge, and starch were placed in the peritoneal cavities of rats. Gross granulomas developed in 52%, 33%, 33%, and 67%, and microscopic granulomas in 72%, 33%, 33%, and 83% of these groups of rats, respectively. They noted typical “Maltese cross” crystals under polarized light in the starch group and variable birefringent patterns in the gauze groups. A similar granuloma was seen after cotton had been inadvertently left, presumably in the epidural space, after discomoty. The observations of Sturdy, et al., suggest that bleached cotton, either in

hypothalamus-pituitary axis in two. Pathological examination in one case revealed a granulomatous foreign-body reaction containing birefringent material at the site of gauze implantation.

Onset of symptoms was abrupt in two patients and progressed stepwise in five. At least partial recovery was documented in eight cases, in keeping with an associated vascular pathophysiology. It seems reasonable to conclude that the application of gauze in these nine patients initiated a foreign-body granulomatous reaction, regardless of site or occurrence of subarachnoid hemorrhage. The reaction was self-limiting in all but the fatal case, yet was capable of damaging adjacent structures to produce epilepsy or cranial nerve palsies. Although concomitant bacterial infection might be suspected of being the critical factor initiating the granulomatous response, none was ever proven in the cases described, the reaction appearing to be a sterile one. Choi, et al.,³ described masses with similar features related to retained sponges after four instances of abdominal surgery, noting the CT appearance and describing the presence of cotton fibers and/or birefringent fragments in the lesions. Sturdy, et al.,¹³ studied foreign-body granulomas after 32 intraperitoneal, 23 subcutaneous, and six uterine cervical operations and one intra-articular procedure, comparing results with experimental studies in which lint from dry packs, laparotomy sponge, gauze sponge, and starch were placed in the peritoneal cavities of rats. Gross granulomas developed in 52%, 33%, 33%, and 67%, and microscopic granulomas in 72%, 33%, 33%, and 83% of these groups of rats, respectively. They noted typical “Maltese cross” crystals under polarized light in the starch group and variable birefringent patterns in the gauze groups. A similar granuloma was seen after cotton had been inadvertently left, presumably in the epidural space, after discomoty. The observations of Sturdy, et al., suggest that bleached cotton, either in
the form of gauze, cottonoid, or laparotomy sponge, can cause foreign-body granulomas containing birefringent material, particularly if lint is plentiful; the failure of granulomas to form in every rat suggests an idiosyncratic reaction.

The incidence of this granulomatous reaction after aneurysm surgery, for which radiologists have coined the word “gauzoma,” is unknown. From 1982 to 1986, during which time the five cases occurred at the Toronto General Hospital, 218 aneurysms were clipped by the five staff neurosurgeons, virtually all with gauze reinforcement. However, CT scans were not performed routinely after surgery, so some cases might have been missed. Before a high-quality scanner became available during 1983, CT scans might not have revealed “gauzomas” even in symptomatic cases; thus, asymptomatic lesions would certainly have been missed. On the other hand, enhanced CT scans, carried out for other reasons in patients whose aneurysms are known to have been reinforced with gauze, have thus far not revealed any asymptomatic “gauzomas.”

There is no reason to believe that there was anything exceptional about the gauze used in patients who developed granulomas, nor any common factor between the Toronto and Australian cases. The gauze used in Toronto was usually radiomarked and 4 × 4-in. in size, taken off the operating room table. Occasionally, the wrapping material was similarly prepared in advance from 4 × 4-in. unmarked gauze. Both types, obtained from two suppliers, consisted of 100% cotton thread and were stored unsterile in bulk to be counted, packaged, and sterilized as needed. Any unused material was discarded after expiry of sterilization date.

In Toronto, gauze wrapping has been carried out since the early 1950’s. Originally, strips of gauze were wrapped around aneurysms and secured with silver clips. The appearance of “gauzomas” only in the 1980’s is perhaps explained by the fact that, if this apparently idiosyncratic self-limiting process had appeared before adequate CT scanning, it might either have been overlooked or else attributed to other causes. It remained for our exceptional Case 1 to draw attention to this problem. Neither the composition of the gauze nor the strategy of its use has changed with time. The implications for the surgeon are difficult to define. Although the incidence of “gauzomas” may be low, and the consequences nearly always reversible or self-limiting, nevertheless, wrapping of aneurysms with gauze can lead to remarkable complications, and should likely be avoided when an aneurysm has been satisfactorily obliterated, particularly near cranial nerves. The risk of using gauze for wrapping in other situations should be weighed against the dangers of aneurysmal rebleeding.

Alternatives are elusive. Dutton41 introduced the coating of aneurysms with acrylic, and reported an 8% mortality in 106 patients, none of whom rebled. Hoppenstein, et al.,9 however, noted thrombosis of canine arteries 2 mm or less in diameter when methyl-2-cyanoacrylate (commonly used to coat aneurysms) was used for their anastomosis. Coe and Bondurant4 re-ported a patient who suddenly developed hemiparesis and aphasia 5 weeks after a cerebral aneurysm had been coated with fascia and cyanoacrylate resulting in thrombosis of both the aneurysm and its parent artery. Sachs, et al.,12 reported acute necrosis of the aneurysmal wall leading to a massive hemorrhage 3 days after coating with methyl-2-cyanoacrylate. Yodh and Wright17 and Handa, et al.,9 have reported neurotoxic effects from similar compounds. A recent review by Weir16 has raised not only concern about toxicity but also doubts about long-term effectiveness when plastic adhesive agents are used in an attempt to prevent fatal aneurysmal rebleeding. As an alternative, Sugita14 has proposed the use of thin layers of Bemsheets with fibrin glue to protect unclippable aneurysms. It will require time and experience to be certain that alternative substances do not produce the same type of delayed inflammatory response that we have identified after use of gauze.

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References


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