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Symptomatic Pulmonary Complications from Liquid Acrylate Embolization of Brain Arteriovenous Malformations

David M. Pelz, Stephen P. Lownie, Allan J. Fox, and Linda C. Hutton

PURPOSE: To describe symptomatic pulmonary emboli from brain arteriovenous malformation embolization with liquid acrylates and to analyze the reasons for these complications and describe preventive techniques. **METHODS:** The clinical records of 182 patients embolized with acrylate glue since 1978 for treatment of brain AVMs were searched for evidence of symptomatic pulmonary complications. Originally iso-butyl-2-cyanoacrylate and more recently n-butyl-2-cyanoacrylate were used in all patients. Arteriovenous malformation morphology, amounts and techniques of glue injection, and clinical and radiologic investigations in the symptomatic patients were recorded. **RESULTS:** Three patients had pulmonary symptoms within 48 hours of glue injection. One patient with a left frontal arteriovenous malformation had embolization with an isobutyl-2-cyanoacrylate/pantopaque/acetic acid mixture; severe pleuritic chest pain developed 2 days later. One patient with a left temporal and one with a left cerebellar arteriovenous malformation had embolization with n-butyl-2-cyanoacrylate/lipiodol mixtures; a cough, pleuritic chest pain, and bloody sputum developed in both within 24 hours. Two patients experienced a significant drop in Po_2. No flow-arrest techniques were used for any of the injections in these three patients. All patients demonstrated significant changes on chest x-ray and CT chest examinations. All were treated conservatively and recovered spontaneously. **CONCLUSIONS:** Symptomatic pulmonary complications can occur after acrylate glue injection, particularly when delivery systems without flow arrest are used in high-flow vascular brain lesions. Techniques using acetic acid to delay polymerization time and "sandwich" techniques in which glue is pushed with dextrose are also more susceptible to this complication.

Index terms: Interventional neuroradiology, complications of; Interventional materials, embolic agents; Arteriovenous malformations, embolization; Iatrogenic disease or disorder

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Embolization therapy of brain vascular malformations with the cyanoacrylate glues isobutyl-2-cyanoacrylate (IBCA) and more recently n-butyl-2-cyanoacrylate (NBCA) has been performed for two decades (1–3). Various complications resulting from this therapy have been described, usually related to the central nervous system (4–7). An infrequently documented complication involves embolization of cyanoacrylate to the lungs. Asymptomatic embolization of IBCA to the lungs has been described (8), and there has been a report of a death from respiratory failure caused by pulmonary emboli after IBCA embolization of a nasopharyngeal arteriovenous malformation (AVM) (9). This report reviews three patients who suffered symptomatic pulmonary complications from embolization of brain AVMs with cyanoacrylate glues. Each case was confirmed by chest x-ray and chest computed tomography (CT) examinations.

Materials and Methods

Of 182 patients who underwent embolization of brain AVMs with cyanoacrylate glue at our institution since 1978, pulmonary symptoms were known to have developed in three patients after embolization. Their clinical and radiologic records were reviewed. Details of AVM angio-

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architecture and the embolization techniques used were recorded. Particular attention was given to the presence or absence of associated arteriovenous fistulas, amounts of glue injected, proportions of glue to polymerization-altering agents such as iophendylate (Pantopaque), lipiodol, and acetic acid, types of delivery systems used, and the techniques of glue injection.

Results

Three patients were identified who clearly suffered symptomatic pulmonary complications related to their embolization procedures. All three complications occurred in the series of 52 patients who had embolizations after the introduction of newer embolic delivery systems in 1986 (7). In this group of cases, Tracker minicatheters (Target Therapeutics, Fremont, Calif) and Balt Magic minicatheters (Balt Extrusion, Montmorency, France) without flow arrest and the cyanoacrylate glues IBCA and NBCA were used for embolization. Acetic acid was used in approximately 15 to 20 cases to prolong polymerization time of the acrylic glue (10). In the previous 115 embolization cases calibrated leak balloons with flow arrest had been used for injection of IBCA.

Patient One

The first patient is a 37-year-old woman who presented with left parietal headaches and one grand mal seizure. Investigations revealed a large left frontal AVM supplied primarily by branches of the left anterior and middle cerebral arteries (Fig 1A, B). Embolization was performed as a preoperative procedure. During the first embolization session, a Tracker 18 minicatheter was navigated into the left callosomarginal artery, and three injections of 0.4 mL of IBCA diluted to 50% with iophendylate were made. Tantalum powder was used as the opacifying agent. The glue was pushed with a bolus of dextrose each time, the so-called sandwich or push technique (11). The patient experienced a transient mild left arm weakness lasting approximately 3 hours but recovered spontaneously.

The second session was performed 8 days later. A Balt Magic catheter was positioned in the left middle cerebral artery. A single injection of 0.4 mL of IBCA was made. The glue mixture was 1.0 mL of IBCA to 0.6 mL of iophendylate with addition of 10 μL of acetic acid. The acetic acid was used to delay the polymerization time because of a relatively proximal catheter position (10). The glue again was pushed with a bolus of dextrose.

During the injection, some glue was seen to traverse the AVM and enter the right transverse sinus and torcular. The patient was initially well after the procedure, but within 48 hours she began experiencing left-sided posterior pleuritic chest pain. Initially this was not severe, and the patient was discharged from hospital only to return 3 days later with much more severe pain.

The chest x-ray showed peripheral, segmental areas of consolidation in the middle and lower lung zones bilaterally, with multiple small radiodensities in both lungs (Fig 1C, D). A CT scan of the chest showed multiple peripheral wedge-shaped areas of consolidation. Metallic densities, consistent with tantalum-cyanoacrylate emboli, were seen at the apexes of these consolidations, which were considered to represent infarcts (Fig 1E). The patient was treated conservatively because no significant drop in Po_2 was observed. She recovered spontaneously, and her AVM was completely resected 10 days later.

Patient Two

The second patient is a 23-year-old man who presented with ataxia and progressive blindness caused by a large left cerebellar AVM (Fig 2A, B). Arterial supply was primarily from large left superior cerebellar, anterior inferior, and posterior inferior cerebellar arteries. There was a rapid intense nidus blush observed with rapid filling of draining veins, indicating the likely presence of multiple arteriovenous fistulas within the nidus. The straight sinus was occluded, and there was retrograde filling of deep and medullary cortical veins. The patient's visual loss was best explained by venous hypertension in ophthalmic veins that filled from the AVM. Embolization was planned to lower the venous pressure and possibly precede radiation therapy.

During the first embolization session, a Tracker 18 catheter was navigated into the left occipital artery and provided transosseous supply to the AVM. One injection of 0.3 mL of NBCA diluted to 50% with lipiodol was made, and this was pushed with dextrose. Tungsten powder was used as the opacifying agent. A second occipital artery branch was embolized with 0.45 mL of this mixture, this time without a

Fig 1. Patient 1. *A,* Anteroposterior and *B,* lateral projections from a left internal carotid arteriogram. There is a large left frontal AVM supplied by branches of the left anterior and middle cerebral arteries.

C, Posteroanterior and *D,* lateral chest films on the day after embolization. There are moderate-size areas of consolidation in the middle and lower lung zones bilaterally (*arrows*).

E, CT chest scan performed 3 days after the embolization. There are multiple peripheral wedge-shaped areas of consolidation in the right middle lung zone with punctate radiodensities, representing IBCA-tantalum emboli, at the apexes of these lesions (*arrows*).

dextrose push. It was thought that all of the glue was deposited in the AVM nidus.

The second session, performed 6 days later, began with superselective catherization of the left superior cerebellar artery using a Balt Magic 1.8 catheter. A continuous-column technique was used for injection of approximately 1.0 mL of NBCA diluted to 50% with lipiodol (11). During the initial stages of this injection, small amounts of glue were seen to pass through the nidus into three draining veins. Most of the NBCA then was deposited within the AVM nidus. The patient was neurologically unchanged, but almost immediately severe left-sided pleuritic chest pain developed and required narcotic analgesia. An immediate postembolization chest x-ray showed areas of subsegmental atelectasis at the right base (Fig 2C, D). The Po_2 had dropped to 62 mm Hg. Initially there was clinical concern that the patient may have had a pulmonary embolus, and a ventilation-perfusion nuclear medicine lung scan was compatible with this diagnosis. The patient was therefore treated with intravenous heparin, oxygen, and analgesia. A CT scan of the chest done the next day showed multiple tiny radiopacities in both lower lung zones and the right apex consistent with NBCA emboli. Multi-

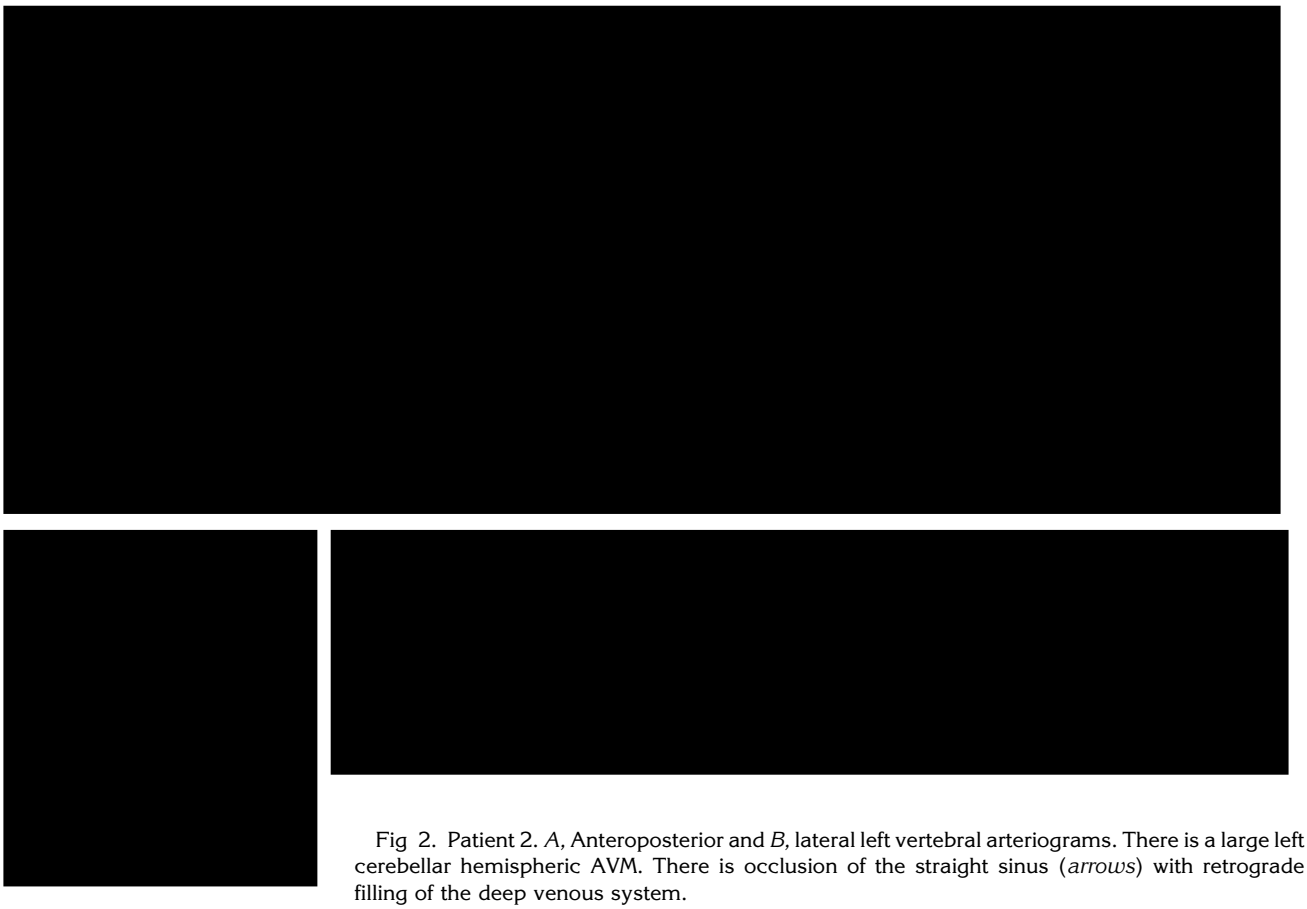

Fig 2. Patient 2. *A,* Anteroposterior and *B,* lateral left vertebral arteriograms. There is a large left cerebellar hemispheric AVM. There is occlusion of the straight sinus (*arrows*) with retrograde filling of the deep venous system.

C, Posteroanterior and *D,* lateral chest films immediately after the embolization. There is ill-defined consolidation with volume loss in the right lower lobe (*arrows*).

E, F, Chest CT performed the same day. There are multiple small densities, consistent with NBCA-tungsten emboli in both lung bases (*E, arrows*). Triangular pleura-based opacities beyond these emboli represent pulmonary infarcts (*F, arrows*).

ple pleura-based opacities were observed extending from these emboli, representing pulmonary infarcts (Fig 2E, F). The heparin was stopped after 5 days, and the patient slowly recovered.

Patient Three

The final patient is a 35-year-old man who presented with temporal lobe seizures originating from a moderate-size left temporal lobe AVM. The AVM was being supplied by several large anterior temporal branches of the left middle cerebral artery (Fig 3A, B). There was moderate arteriovenous shunting through the lesion but no fistulas were identified. Preoperative embolization was requested.

A Balt Magic Olive 1.8 catheter was used to navigate selectively into the largest middle cerebral artery feeder. The first glue injection consisted of 0.3 mL of NBCA, diluted to 50% with lipiodol, and pushed with dextrose. Tungsten powder was again used as the opacifying agent. This material passed entirely through the AVM, and no glue was identified in the draining veins. It was thought that most of the glue must have passed into the lungs. The second injection of 0.5 mL of the same NBCA mixture, this time injected as a continuous column, was then made through the same catheter. This glue appeared to deposit within the AVM nidus. A final injection of 0.4 mL of this mixture, using the continuous-column method, was made into another middle cerebral artery feeder using a new Magic catheter, and this material also remained within the AVM nidus.

Initially the patient was well, but within 24 hours he began to have moderate left-sided pleuritic chest pain. A chest x-ray performed 3 days after embolization showed bilateral pleural effusions with subsegmental atelectasis at the left base (Fig 3C, D). Multiple tiny radiodensi-

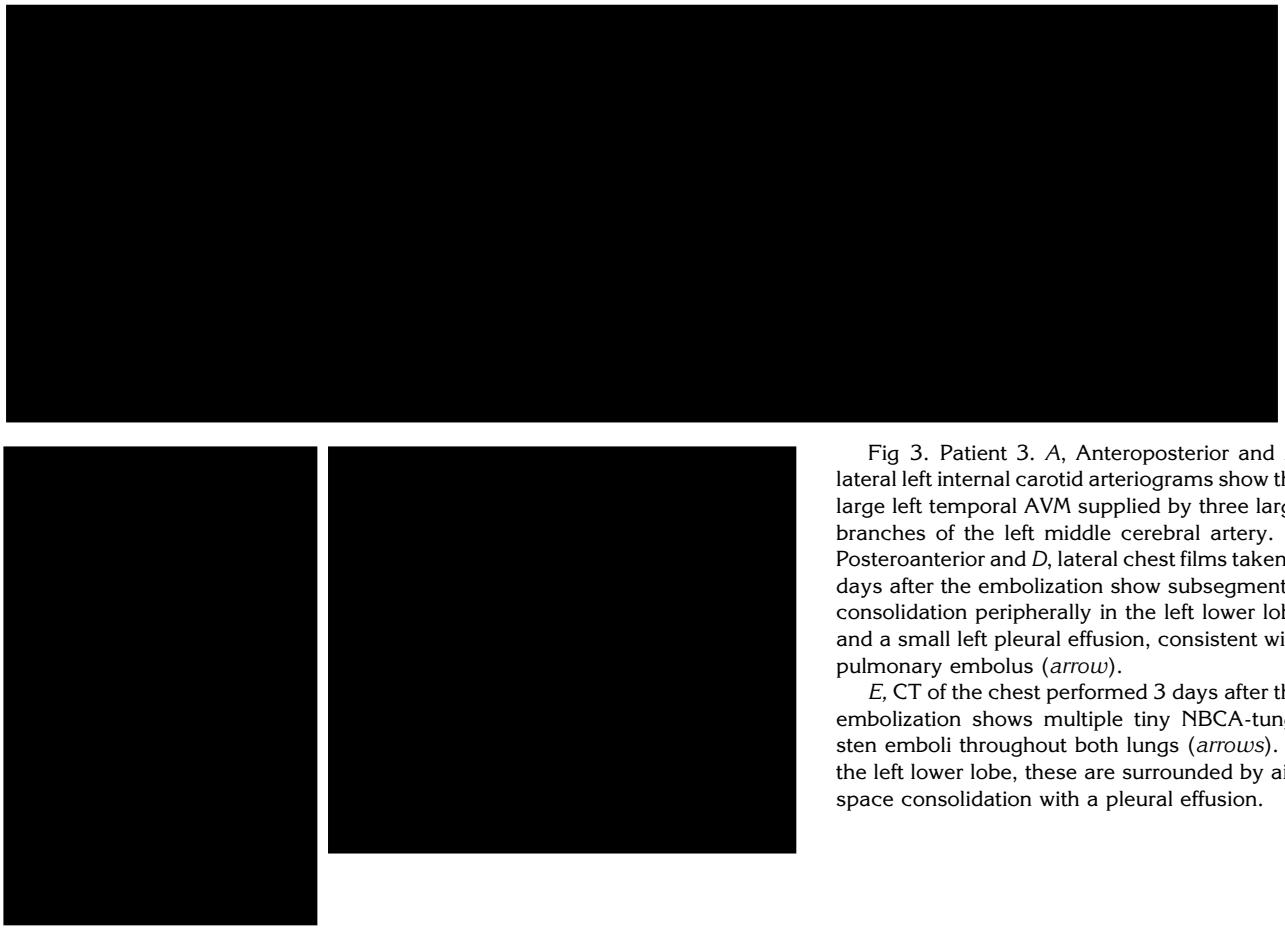

Fig 3. Patient 3. *A*, Anteroposterior and *B*, lateral left internal carotid arteriograms show the large left temporal AVM supplied by three large branches of the left middle cerebral artery. *C*, Posteroanterior and *D*, lateral chest films taken 4 days after the embolization show subsegmental consolidation peripherally in the left lower lobe and a small left pleural effusion, consistent with pulmonary embolus (*arrow*).

E, CT of the chest performed 3 days after the embolization shows multiple tiny NBCA-tungsten emboli throughout both lungs (*arrows*). In the left lower lobe, these are surrounded by airspace consolidation with a pleural effusion.

ties representing the NBCA-tungsten mixture were evident in both upper lobes. A CT of the chest performed on the same day also showed these peripheral radiopacities, consistent with NBCA-tungsten, surrounded by patchy air space consolidation representing infarcts (Fig 3E). The patient was in some distress from his pleuritic chest pain. He had shortness of breath and was coughing up bloody sputum. The Po_2 dropped to 65 mm Hg. He gradually recovered over the next week with oxygen and analgesia.

A second embolization session was performed 12 days after the first session in which two more branches of the left middle cerebral artery were embolized with a 50% NBCA/lipiodol mixture using the continuous column technique. The AVM was completely resected 1 week later.

Discussion

Many embolic agents have been used to treat brain AVMs since the original report by Luessenhop (12). These include microspheres of various types (12), silk thread (13), polyvinyl alcohol (14), avitene (15), and the liquid cyanoacrylates including IBCA and NBCA (1–3, 16). Most complications of these treatments relate to the central nervous system; complications involving other organ systems are uncommonly observed and rarely reported. Pulmonary complications have however been described after embolization of vascular lesions elsewhere in the body.

McCarthy et al (17) described two patients with symptomatic pulmonary emboli in a series of 137 patients embolized for treatment of peripheral AVMs with gelfoam, polyvinyl alcohol, and hypertonic dextrose. In a smaller, prospective series of 10 patients, they describe 2 patients who showed perfusion defects on postembolization nuclear medicine perfusion lung scans, consistent with pulmonary emboli, although neither patient was symptomatic. The authors believe that subclinical pulmonary em-

boli occur in a significant proportion of patients undergoing embolization of these lesions.

In a series of 336 patients who underwent transarterial chemoembolization of hepatic carcinomas with lipiodol-adriamycin mixtures, Chung et al (18) describe 6 patients who developed respiratory symptoms 2 to 5 days after embolization. These symptoms included cough, dyspnea, and hemoptysis with arterial Po_2 ranging from 39 to 60 mm Hg. The chest x-rays showed diffuse bilateral pulmonary infiltrates, and 1 patient died of a respiratory arrest. The authors believe that the likely cause of symptoms was lipiodol embolization to the lungs with a subsequent chemical injury resulting from breakdown of the lipiodol to free fatty acids.

There has been a single report of symptomatic IBCA embolization to the lungs. In a review of 100 patients with a variety of lesions embolized with IBCA, Goldman et al (9) described a 12-year-old girl who underwent IBCA–ethiodized oil embolization of a large nasopharyngeal AVM. The patient was initially well after the procedure, but respiratory difficulties developed approximately 3½ hours later. A chest x-ray showed high-density glue and patchy pulmonary consolidations throughout both lungs, and the patient died of a respiratory arrest. Two other patients who manifested no pulmonary symptoms after embolization were found to have IBCA in their lungs at postmortem exam.

Asymptomatic pulmonary migration of embolic material likely occurs more often than is appreciated. Takasugi et al (8) described the preoperative chest radiographic findings in an asymptomatic patient who had undergone IBCA embolization many years previously. Multiple small radiopacities were seen throughout both lungs and a ventilation-perfusion nuclear medicine scan showed multiple peripheral subsegmental defects consistent with pulmonary emboli.

Hutton et al (19) described the chest findings in six patients who underwent embolization of neurovascular lesions. In two patients with arteriovenous fistulas, striated muscle and tantalum mesh in one patient and a detachable latex balloon in another patient were seen on subsequent chest x-rays in the lungs. The second patient briefly became hypotensive and tachycardiac but recovered spontaneously. Four patients had brain AVMs embolized with polyurethane and silastic spheres, silicone beads, and IBCA. The beads and microspheres were detected on follow-up chest x-rays, and the IBCA-tantalum was found in the lungs at postmortem exam. None of these patients demonstrated any pulmonary symptoms.

A series of three postmortem cases was reported by Coard et al (20). Three patients who had undergone IBCA embolization for cerebral AVMs died, one from surgical complications and two from subsequent cerebral hemorrhages. Calibrated leak balloons with temporary flow arrest had been used for all glue injections, and none of the patients had shown any pulmonary symptoms. Postmortem examination of the lungs in all cases showed IBCA and tantalum powder in the lumina of numerous pulmonary arteries and arterioles with thrombi, chronic inflammatory cells and lymphocytes, plasma cells, and macrophages in the vessel walls with extentions into the perivascular connective tissue. The authors found a rough correlation between the total amount of IBCA injected and the number of pulmonary emboli detected. It is likely, therefore, that asymptomatic embolization of cyanoacrylate occurs more often than is appreciated, even when flow-arrest techniques are used.

IBCA is known to cause an intense inflammatory reaction in brain (21) and nonneural tissue (22). Cromwell et al (22) described acute and chronic inflammatory changes in canine kidneys up to 4 weeks after IBCA embolization. These changes included perivascular inflammation and vessel wall necrosis with chronic foreign body reactions seen 4 to 5 weeks after the procedure. Similar findings were described by Vinters et al (21) in their series of 17 surgically resected brain AVMs. An acute polymorphonuclear infiltrate was seen in the first 48 hours, with an intense foreign body reaction and necrosis of vessel walls noted in the first week. Necrosis of parenchyma adjacent to embolized vessels also was observed with IBCA actually migrating into adjacent neuroglial cells. This could be seen up to 6 weeks after the procedure. The authors theorized that this intense reaction could be attributable to slight heating of the tissues as the IBCA polymerizes or toxin release as IBCA degrades. IBCA clearly has the potential to cause significant tissue damage in the lungs. Brothers et al (15) found the tissue responses of IBCA and NBCA to be virtually indistinguishable in the pig rete. NBCA is therefore likely to be no less irritating to lung parenchyma.

Our three patients became symptomatic within 48 hours of their procedures, one almost immediately. The initial symptoms may relate to mechanical obstruction of pulmonary vasculature with an acute inflammatory reaction leading to pleuritic chest pain. This may have been exacerbated by the chemotoxic effects of the oily contrast agents iophendylate and lipiodol. The cause of chest pain observed in the first patient 3 weeks after her embolization is uncertain but may relate to breakdown products of IBCA.

In all three patients, glue was injected using minicatheters without flow arrest. This lack of flow arrest, particularly in high-flow lesions such as in case 2, may have contributed to the pulmonary migration of glue. In the series of 115 cases performed using flow arrest with calibrated leak balloons, no symptomatic pulmonary complications were observed, although IBCA was seen in the lungs in three patients at postmortem exam (20). Clearly, flow arrest does not prevent pulmonary migration of acrylic glue, but it may reduce the amount to subclinical levels in high-flow lesions. Calibrated leak systems are awkward to use and generally have been replaced by new minicatheters; however, temporary balloon occlusion or use of double-lumen catheters could be considered in these cases. The risk of vessel rupture by these flow-arrest techniques must be acknowledged and may outweigh the lower risk of pulmonary glue embolization. In our series of 115 cases treated with the calibrated leak balloon system, four vessel ruptures occurred with one associated death.

In two of our symptomatic patients, the "sandwich," or "push," technique was used for injecting glue (11). This technique has been used at our institution since 1980. Small amounts of cyanoacrylate (0.1 to 0.4 mL) are injected into the catheter and are then pushed with a bolus of dextrose. This technique is useful to permit more than one injection through the delivery catheter and is less likely to result in catheter adhesion to the feeding vessel. It is, however, more likely to result in fragmentation of the glue bolus with delayed polymerization. Therefore, there is a higher risk of distal glue migration into venous channels and the pulmonary circulation. In case 3, distal embolization of glue ceased when a continuous column injection technique was used (11), thus removing arterial inflow as the NBCA polymerized in the feeding vessel. In case 2, distal glue migration was observed even though a continuous-column technique was used, caused by the presence of multiple high-flow arteriovenous fistulas within the AVM nidus. Faster glue polymerization time and flow-arrest techniques likely would have prevented complications in this case.

In case 1, the use of acetic acid to delay glue polymerization time also likely contributed to the pulmonary migration of IBCA. We have used acetic acid with IBCA in several other cases with no pulmonary symptoms observed subsequently, although asymptomatic emboli cannot be excluded. Careful calibration of polymerization time is necessary in all embolization cases, but this is a very inexact science with many variables to consider (5, 6, 10). NBCA, however, has a slightly longer setting time in vivo than IBCA (10). The risk of gluing the catheter into the feeding vessel is therefore decreased, and the use of acetic acid to prolong polymerization time is now likely rarely indicated.

Treatment guidelines for symptomatic pulmonary complications of glue embolization are primarily conservative, using oxygen and analgesia as required. Heparin was used in case 2 because of initial uncertainty about the cause of the patient's chest pain. When it became clear that the cause was NBCA emboli to the lungs, there was no rationale for continuation of heparin therapy. Use of antiinflammatory medication to decrease the tissue response to NBCA may be useful for pain control in these patients. Ventilation-perfusion scans were performed in two patients and both confirmed the clinical suspicion of multiple pulmonary emboli and were therefore clinically useful.

In summary, we have presented three cases in which cyanoacrylate glues have traversed brain AVMs during embolization, resulting in symptomatic pulmonary emboli. We have identified three possible factors leading to this complication in our patients, including lack of flow arrest during glue injection in high-flow lesions, the use of the sandwich or push injection technique, and the use of acetic acid to delay glue polymerization time. Although asymptomatic pulmonary emboli are known to occur even when flow-arrest techniques and continuous-column injections are performed, these events are unlikely to result in clinical symptoms requiring treatment, as shown in our group of 115

patients in which the calibrated leak system was used and no symptomatic pulmonary complications were observed. Careful assessment of AVM angioarchitecture for precise calibration of transit time is essential to determine polymerization mixtures. This calibration is still the most important parameter for optimal placement of cyanoacrylate glues. The continuous-column technique of injection or flow-arrest techniques should be considered when high-flow fistulas are known to be present. Both these techniques increase the risk of proximal feeder occlusion rather than deposition in the AVM nidus, and polymerization times must be adjusted accordingly. The use of acetic acid to delay polymerization time rarely is indicated with the current use of NBCA. These precautions will help prevent migration of glue to the pulmonary circulation, as well as deposition in cerebral venous structures with associated complications.

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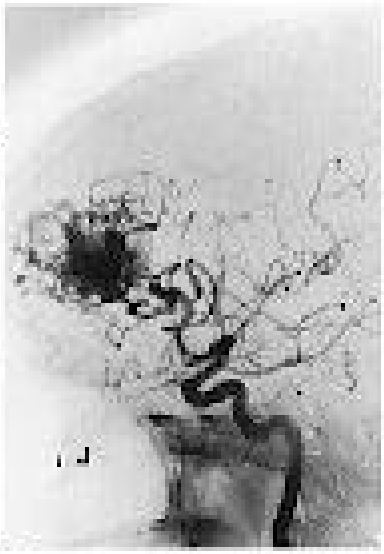

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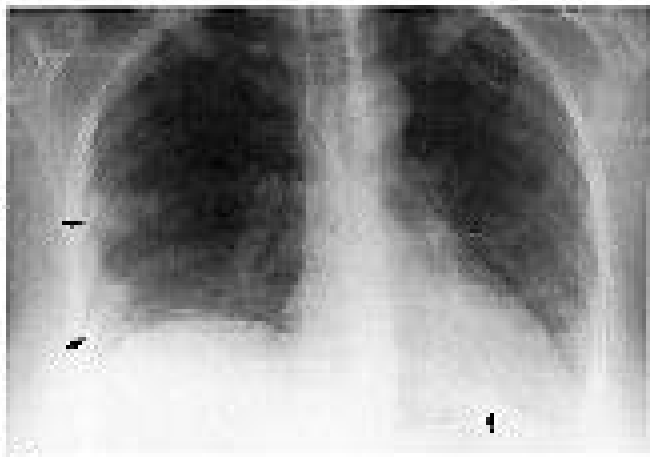

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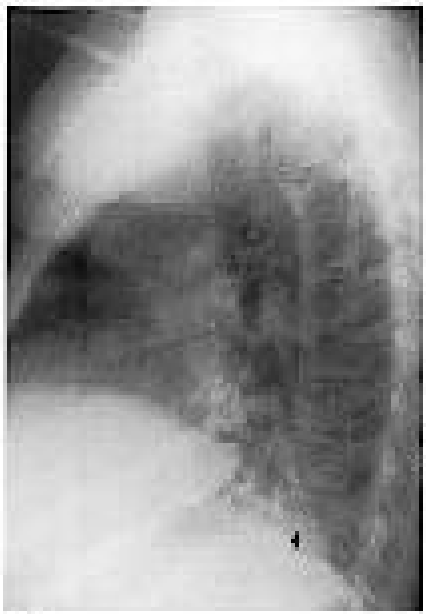

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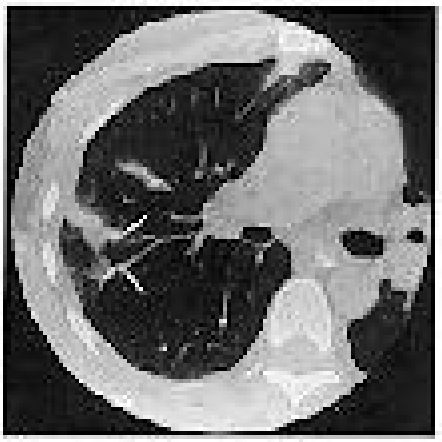

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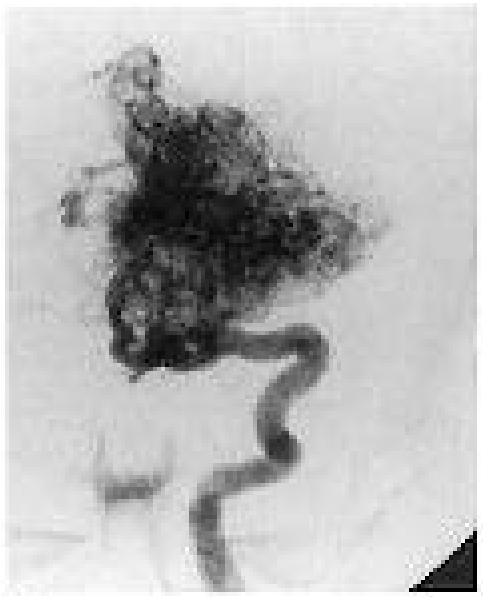

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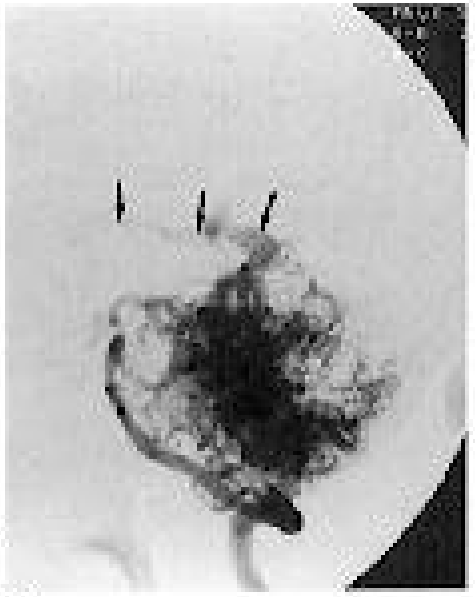

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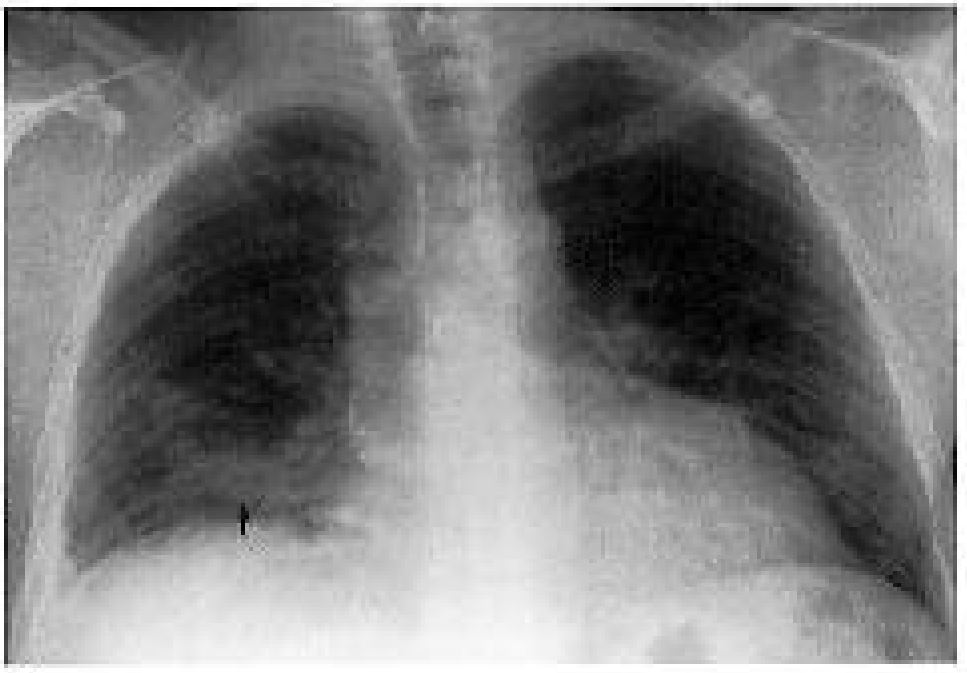

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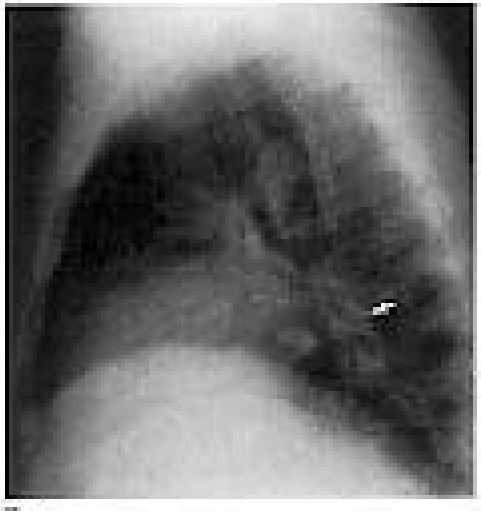

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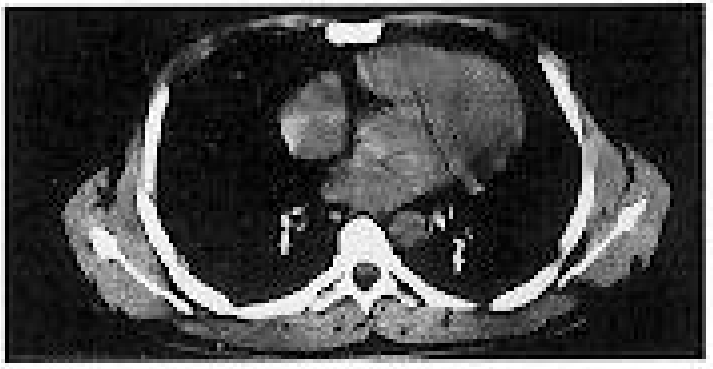

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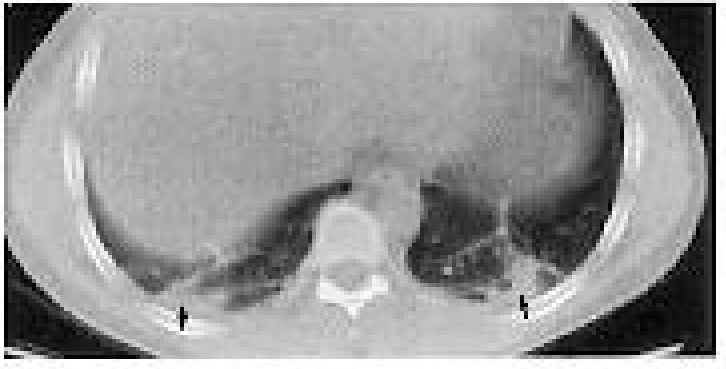

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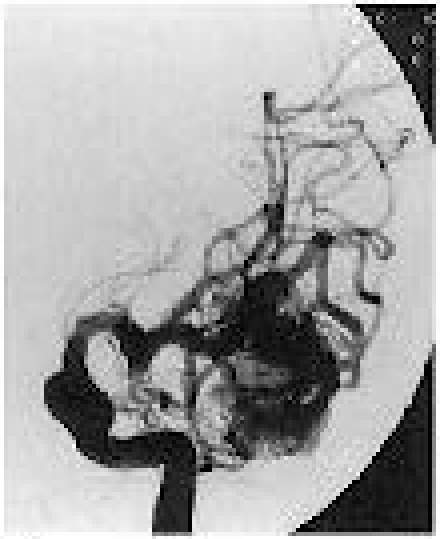

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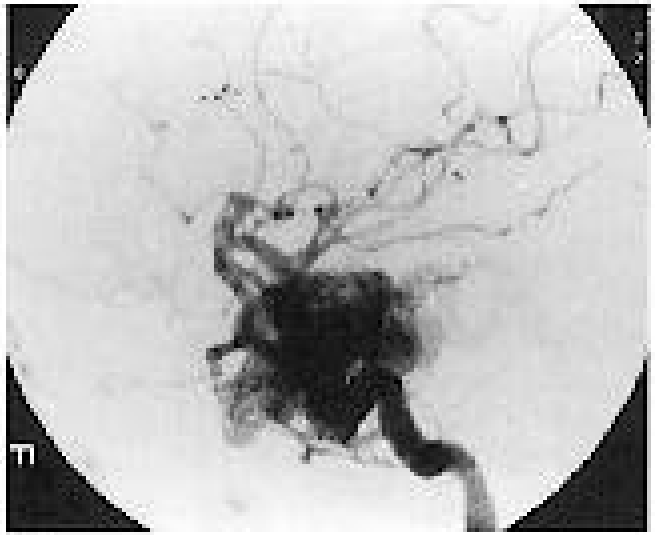

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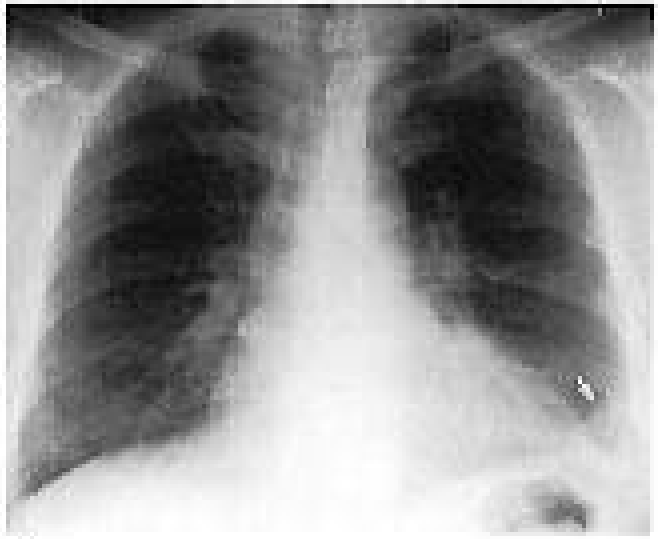

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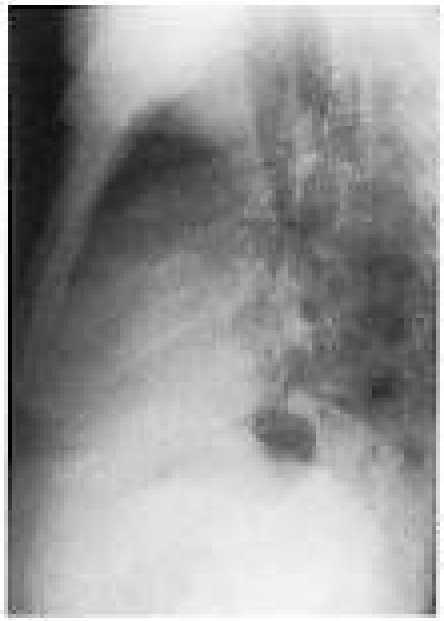

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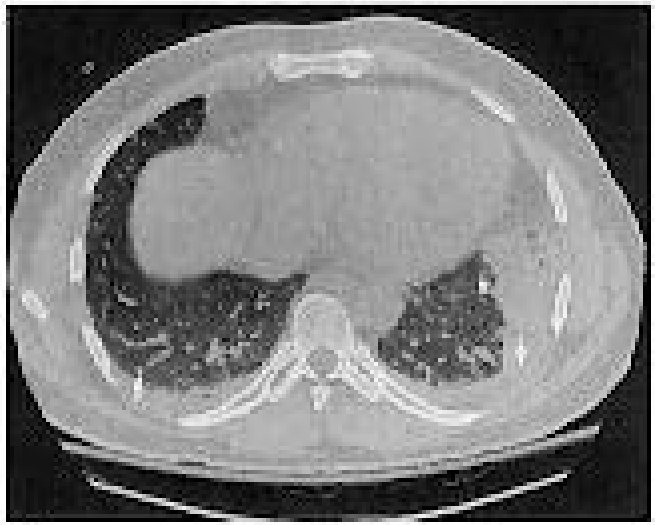

E