Transient cortical blindness: a complication of bronchial artery embolization

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Introduction

Bronchial artery embolization is an effective therapeutic alternative for the treatment of severe haemoptysis, especially when conservative treatments fail or when patients are not good candidates for surgery (1). Some complications of bronchial artery embolization have been reported in the literature including chest pain, ectopic deposition of coil and embolization of other vessels, left main bronchial stenosis or infarction (2), bronchoesophageal fistula (3), spinal cord injury (4), fatal ischaemic colitis (5), and transient pulmonary infarction after complete pulmonary artery and bronchial artery embolization (6). To our knowledge, this is the first report of cortical blindness following bronchial artery embolization in the English literature.

Case Report

CASE 1

A 29-year-old male was admitted to our hospital emergency room because of persistent haemoptysis for 2 days. He had a history of pulmonary tuberculosis and underwent left lower lobe lobectomy for bronchiectasis in 1990, as well as bronchial artery embolization for recurrent haemoptysis in 1991. Because of continued haemoptysis after conservative treatments during this hospitalization, a secondary bronchial artery embolization was performed. Angiogram through bilateral bronchial arteries injection via right femoral artery approach revealed dilated and tortuous hypervascularization over the bilateral lung fields, with blood supplied from the left inferior and right bronchial artery [Plate 1(a) and (b)]. Embolization was performed with ivalon particles (250–590 μm) in 20 ml lipiodol injection in both left inferior artery and right bronchial artery.

Received 26 May 1997 and accepted in revised form 4 August 1997.

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PLATE 1. (a) Left inferior bronchial angiogram showing dilated and tortuous hypervascularity in the left lung field. (b) Right bronchial angiogram showing dilated and tortuous hypervascularity in the right lower lung field.

Stasis of blood flow was obtained [Plates 2(a), (b) and 3]. However, the patient began to suffer from headache, neck stiffness, and bilateral loss of vision approximately 3 h after embolization. The pupillary response to light reflex was normal and there was a normal range of extraocular movements. There were no other abnormal neurological findings except neck stiffness. Brain computed tomography (CT) was done immediately but revealed no abnormalities. Cerebrospinal fluid examination was also normal. Ophthalmoscopy showed no evidence of central retinal artery occlusion. Follow-up brain CT 2 days later was unchanged. He completely recovered 3 days later and he was discharged 10 days after bronchial artery embolization.

CASE 2

A 22-year-old female with pulmonary tuberculosis was admitted via the emergency room with severe haemoptysis during the previous 24 h. Because of uncontrolled haemoptysis following medical treatment, a bronchial artery embolization was performed. A selective right bronchial artery angiogram demonstrated hypervascularity with dilated and tortuous bronchial artery branches. Embolization was performed with ivalon particle (250-590 μm) and 9 ml lipiodol injected slowly via the right bronchial artery. Stasis of right bronchial artery flow was obtained. However, the patient began to complain of severe headache and bilateral loss of vision approximately 3 h after embolization. The findings of neurological examination were normal except for bilateral loss of vision. The findings of brain CT were normal. Ophthalmoscopy showed no evidence of central retinal artery occlusion. Her symptoms and signs
disappeared 2 days later and she finally was discharged in stable condition 7 days after bronchial artery embolization.

Discussion

Several mechanisms have been postulated to be responsible for ischaemia of structures adjacent to bronchial arteries following bronchial artery embolization, including embolization of blood supply to the proximal bronchus, occlusion of the capillary bed by small embolic particles, and reflux of the embolic agent into adjacent vessels (2-6). But these mechanisms cannot completely explain the ischaemia of distant structures following bronchial artery embolization.

Embolic agents, such as gelfoam, absolute alcohol, ivalon, and isobutyl-2-cyanoacrylate have been reported to cause ischaemic complication in adjacent structures. These liquid agents or small particles can cause tissue infarction of adjacent structures due to distal vascular occlusion. When right to left shunting is present, these liquid agents or small particles can also embolize into the systemic circulation to cause distant structure ischaemia. Therefore, embolic particles should be at least 200-300 µm in size and larger (300-500 µm) when shunting is identified on the diagnostic angiogram (8,9). In the two cases, ivalon particle is 250-500 µm in size and no shunting can be visible on the angiogram. The lipiodol may have more potential than ivalon to pass abnormal shunting into the systemic circulation causing ischaemia in distant structures.

The two patients in this report presented with transient bilateral loss of vision and headache approximately 3 h after bronchial artery embolization and recovered 2-3 days later. Because the patients' symptoms were bilateral loss of vision and associated with other cerebral manifestations, and because central retinal artery occlusion was not seen on the ophthalmoscopic examination, the possibility of bilateral central retinal arteries occlusion is unlikely. Although the findings of brain CT were normal in both patients, we believe that the embolic of ivalon/lipiodol are the main causes of the cortical blindness. It is hypothesized that the cortical blindness developed because of embolism of the particles to the occipital cortex in both hemispheres, either through a bronchial artery to pulmonary vein connection or through collaterals between the bronchial and vertebral arteries. The latter is more likely, as the postulated emboli are localized in the same arterial territory in both patients. The process of neovascularity can occur in lung inflammation and the neovascularity may arise from other adjacent collateral pathways or different arterial source vessels. Communication via collaterals to the vertebral arteries can occur and has been demonstrated at the time of angiographic study/embolization by Irvin F. Hawkins. Embolic agents can reflux into these collaterals at the time of embolization, especially with bolus delivery of the embolic agents. Once the agents enter these vertebral artery collaterals, they then enter into the vertebral-basilar system and into the posterior cerebral artery distribution where cortical blindness can result as a sequelae. There is one report in the literature of bilateral transient cortical blindness due to emboli during coronary and graft angiography.

Vertebral angiography was performed in the first patient and the appearances were compatible with multiple emboli in the vertebral-basilar circulation, occurring as a result of manipulation of the guidewire close to the left vertebral artery. The first patient's vision recovered within 72 h and all neurological deficits also resolved later. The second patient's vision recovered fully within 15 minutes (10).

Both patients developed headache with one also having neck stiffness. Such reactions are not typical in the setting of cerebral ischaemia. The embolization may be unlikely to be the sole mechanism of the cerebral manifestations and vasospasm (11), contrast neurotoxicity (12), and toxic tissue reaction of embolic particles (13) have been invoked as possible explanations. Light microscopic and immunohistochemical examination showed distinct patterns of tissue reaction to multiple embolic agents which were used in embolotherapy for arteriovenous malformations of the brain. Aritene produced the mildest tissue response but resulted in relatively early endothelialization and recanalization. Cyanoacrylates were longer-lasting but associated with more acute and chronic inflammation and vessel wall changes. Ivalon/ethanol mixture had intermediate properties (13). Tissue ischaemia within the embolized region, together with toxic effects, may promote necrosis of vessel walls and adventitial tissue, and leakage of embolic into the adventitia. The nature and extent of these changes will vary not only with the with location and amount of embolic injected, but with their type (14). The ivalon/lipiodol used in the two patients do not belong to the same batch and contamination is not likely during the whole course. The emboli of ivalon/lipiodol may produce similar localized or generalized toxic tissue reactions which probably induce headache and neck stiffness.

Although using lipiodol to embolize the bronchial artery is an unsafe manipulation in this report, the cortical blindness is an important and potentially morbid complication following bronchial artery embolization that has not been significantly addressed in the literature to date.

References


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**Pulmonary vasculitis associated with anti-Jo-1 antibodies**

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**Introduction**

Anti-aminoacyl t-RNA antibodies (anti-Jo1, PL-7, PL-12, EJ, OJ and KJ) are evident in over a quarter of patients with polymyositis, dermatomyositis or idiopathic inflammatory myositis (1). Over two thirds of such patients have interstitial lung disease (ILD) (1). The anti-synthetase antibody (ASA) syndrome comprises various combinations of ILD, fever, inflammatory myositis, symmetric inflammatory arthritis, Raynauds phenomenon (RP) and mechanics hands (2,3). These may occur in any temporal sequence and some features may be more prominent than others. Myositis is evident in the majority of patients. However, disease prognosis is determined by the severity and rate of progression of the ILD (3). Pulmonary histology generally confirms an interstitial fibro-inflammatory process (4). While respiratory symptoms often develop over many weeks occasional

patients have presented with rapidly progressive lung disease or the adult respiratory distress syndrome (1). Among these latter patients some have had little obvious myositis (1). We describe a patient with a fatal respiratory illness of rapid onset in whom anti-Jo antibodies were associated with a pulmonary vasculitis but no overt myositis.

**Case Report**

A previously well 60-year-old Caucasian female presented with pleuritic chest pain and a 2 week history of progressive dyspnoea, fever, non-productive cough and arthralgia of the hands and feet. She denied recent overseas travel and occupational exposure to allergens or toxic substances. Her past medical history included hypertension, mild episodic asthma and hypothyroidism. There was no significant family history and the patient was a non-smoker. Her medications were thyroxine, frusemide and atenolol and inhaled salbutamol, ipratropium bromide and betamethasone.

On examination she was pyrexial (38°C), hypotensive (blood pressure 100/65) and hypoxaemic (O₂ saturation 92% on 28% mask oxygen). Bibasal inspiratory crackles