Carotid Artery Dissection as a Possible Severe Complication of Pertussis in an Adult: Clinical Case Report and Review

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Adults can experience typical symptoms of pertussis. Complications due to severe paroxysmal coughing have been reported in this age group. We report carotid artery dissection as a possible severe complication of pertussis in a previously healthy adult. Adult booster immunization against pertussis should be considered to protect adults themselves from this illness and its potential complications.

Despite the increasing recognition of pertussis as an important cause of prolonged cough illness, it is still underdiagnosed and is perceived as benign in adolescents and adults compared with infants. In fact, both immunized and nonimmunized adolescents and adults can experience typical symptoms of pertussis, including paroxysms, whooping, posttussive vomiting, and apnea [1–3].

Older persons can also experience serious complications of pertussis. These are reported more frequently among adults (28%) than among adolescents (16%) [1]. Case reports have noted seizures and encephalopathy in adults with pertussis, and pneumonia occurs in 5%–9% of patients aged ≥30 years [1, 4, 5]. Urinary incontinence is reported by 4% of adults with pertussis and by 34% of women with pertussis aged >50 years [1]. In addition, rib fractures, pneumothorax, inguinal hernia, aspiration, subconjunctival hemorrhages, hearing loss, herniated lumbar disk, and cough syncope have been reported in adults as mechanical consequences of the severe cough episodes associated with pertussis [1, 2, 6–9]. We report a case of carotid artery dissection that occurred during the course of pertussis in an adult man who ultimately required 6 weeks of acute care hospitalization followed by 3.5 months of inpatient and ongoing outpatient rehabilitation.

Case report. A 44-year-old health care worker had onset of nonproductive cough on 8 December 2000. This cough was described as paroxysmal, worse in the evening, and accompanied by gagging or choking but unaccompanied by vomiting or constitutional signs, such as fever. He commenced azithromycin therapy at the standard dose on 12 December and continued this therapy for 5 days.

On 17 December 2000, the patient’s coughing was noted to be vigorous, and he experienced right posterior retro-orbital headache accompanied by nausea and vomiting and associated with scintillating lines in the left visual field. This headache was unusual for him, and it persisted through the night, but it was not accompanied by other neurological signs. He was able to bike to work the next morning. At work, he experienced sudden onset of left-sided weakness, during which he fell to the floor but retained consciousness.

When examined in the emergency department, the patient was drowsy but alert. He was able to converse, but his speech was slow and he was dysarthric. He had dense left-sided neurological deficits, including hemiplegia, hemisensory abnormality, hyperreflexia, upgoing toe, homonymous hemianopsia, visual field neglect, and body neglect. He had a right gaze preference. Emergency CT scan revealed edema in the distribution of the right middle cerebral artery without evidence of hemorrhage. There was a hyperdensity in the proximal right middle cerebral artery indicative of thrombus. He was given intravenously administered tissue plasminogen activator in accordance with the standard protocol, but this did not improve symptoms.

Transthoracalechocardiography and bilateral leg ultrasonography revealed no abnormalities. A carotid ultrasound study showed normal left carotids but no flow in the right internal carotid artery; there was some thrombus but no significant plaque. A magnetic resonance angiogram showed dissection and occlusion of the right internal carotid artery, but no dysplasia or other abnormalities of the carotid arteries were noted. The patient was admitted to the hospital with the diagnosis of right spontaneous internal carotid artery dissection with resulting complete right middle cerebral artery infarct.

During the next 2 days, the patient became increasingly obtunded. MRI and additional CT scanning confirmed that there
was an increase in the right middle cerebral artery stroke, mass effect, and brain stem distortion. On the third day of hospitalization, a right frontoparietal temporal hemicraniectomy with dural opening and expansion was required. After the operation, the patient again developed signs of transtentorial herniation, and, on the following day, he was taken to the operating room for resection of an infarcted temporal lobe. During the month after surgery, he developed hydrocephalus that was managed with the temporary insertion (8 days) of a lumbar drain.

Six weeks after the acute neurological event, the patient was transferred to another center for inpatient rehabilitation. When the patient was discharged from the hospital 3.5 months later, he retained significant neurological deficits that required continued outpatient rehabilitation and modifications to his home environment to allow mobility at wheelchair level. His higher cognitive function was mostly intact, but he retained significant visuospatial deficits.

The father of 3 daughters (ages, 11, 10, and 6 years), this patient had previously been in good health. He had no preceding risk factors for cerebrovascular or cardiovascular disease. He had no family or personal history of hypertension, diabetes mellitus, high cholesterol level, connective-tissue disorder, or coagulopathy. No other family member had a history of carotid or vertebroarterial dissection. He had never smoked and was taking no medications. He was very active and exercised regularly. He had an episode of transient unilateral body numbness in 1991 that lasted <24 h and fully resolved spontaneously. The findings of MRI performed at the time were normal, and the cause of the numbness was considered to be viral. He had no subsequent recurrence of neurological symptoms. The patient denied having experienced spinal manipulation, blunt trauma, injury, or sporting activity immediately preceding the onset of his most recent neurological event.

Within 8 days after his stroke, numerous other persons from the patient’s household began to experience a similar cough illness, including his 10- and 11-year-old daughters, his brother-in-law, and his wife. Ultimately, all of these persons described a paroxysmal cough that lasted several months. All of the children were up to date with pertussis immunization. The 6-year-old remained asymptomatic before and throughout her father’s illness and had most recently been immunized with an acellular pertussis–containing vaccine in 1999.

The 10-year-old daughter had laboratory-confirmed pertussis diagnosed by means of PCR on 10 January 2001. Among nonhousehold contacts was the 74-year-old mother-in-law, who was noted to have paroxysmal cough that commenced on 6 January 2001 and who had laboratory-confirmed pertussis diagnosed by means of PCR on 15 January 2001.

Although the patient’s cough persisted for several months, the diagnosis of pertussis was not sought during his prolonged and complicated hospital stay. Interest in assessing a link with his carotid artery dissection led to confirmatory testing during his convalescence. No serum samples remained from his original hospital stay, but paired serum samples were obtained 3 and 4 months after the onset of his illness, and tests of these samples supported a diagnosis of pertussis (table 1). Tests revealed a high titer IgG to pertussis antigens in both paired serum specimens, with decreasing titers in the month that intervened between specimen collections. The patient had not been immunized against pertussis in nearly 3 decades.

Discussion. This patient, who had spontaneous internal carotid artery dissection and massive right hemisphere infarct, had no predisposing risk factors for his neurological event. His cerebrovascular accident was preceded by clinical pertussis that included a paroxysmal cough that had commenced 10 days earlier, was noted to be vigorous on the first day of neurological symptoms, and extended several months into his hospital stay.

This patient had an epidemiologically confirmed case of pertussis. Several of his closest contacts, who had a similar cough illness, had PCR-confirmed pertussis. The results of the patient’s serological tests corroborated this diagnosis. His IgG values for the first specimen for pertussis toxin (PT) and fimbriae were 3 standard deviations higher than are those generally observed in healthy Canadian adults [10]. In fact, the PT titers for this patient 3 months after the onset of his cough illness were higher even than those observed 1 month after booster immunization against pertussis in healthy adults [11]. He appears to have been the primary case within his household. The original source of his infection remains unknown. Health care workers are noted to be at increased occupational risk

<table>
<thead>
<tr>
<th>No. of days after cough onset</th>
<th>IgG Pertussis toxin</th>
<th>FHA</th>
<th>FIM</th>
<th>PRN</th>
<th>IgA Pertussis toxin</th>
<th>FHA</th>
<th>FIM</th>
<th>PRN</th>
</tr>
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<tr>
<td>84</td>
<td>387</td>
<td>28</td>
<td>373</td>
<td>55</td>
<td>15</td>
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<td>34</td>
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<td>122</td>
<td>160</td>
<td>17</td>
<td>211</td>
<td>26</td>
<td>13</td>
<td>16</td>
<td>33</td>
<td>10</td>
</tr>
</tbody>
</table>

NOTE. FHA, filamentous hemagglutinin; FIM, fimbriae; PRN, pertactin.
for developing pertussis, and an outbreak of pertussis had been occurring in his urban center for several months before he became ill [1, 12].

The causal link between his preceding pertussis illness and subsequent carotid artery dissection remains uncertain. Patients with spontaneous dissection of the carotid artery are thought to have an underlying structural defect of the arterial wall, although the precise nature of arteriopathy remains unknown in most cases [13]. In 1%–5% of patients, a well-characterized and heritable connective-tissue disorder is identifiable, and 5% have ≥1 family member who has had spontaneous dissection of the aorta or its main branches [14, 15]. The patient we describe had none of these traits in his personal or family history, and investigations identified no other vascular abnormalities.

Minor precipitating events previously identified as causally related to spontaneous dissection of the carotid artery have involved hyperextension or rotation of the neck, such as that caused by using hairdressers’ basins, practicing yoga, painting a ceiling, coughing, vomiting, or sneezing. Such neck movements, particularly if they are forceful and sudden, may injure the artery as a result of mechanical stretching [16–18]. A recent history of respiratory tract infection has also been identified as a risk factor for carotid dissection [19, 20]. The role of an infectious trigger has been supported by the finding of seasonal variation in the incidence of spontaneous dissection of the carotid artery, with a peak incidence in the fall [21].

Neurological events have been reported elsewhere to occur with pertussis in adults. These include encephalopathy, seizures, and cough syncope [1, 5, 8]. In children, cerebral complications have included hemiplegia, diplegia, and paraplegia, and, pathologically, cerebral hemorrhages have been observed [22]. Intracranial bleeding related to symptomatic pertussis has previously been cited as a cause of death among elderly persons during an institutional outbreak [23]. We report an association between carotid artery dissection, stroke, and pertussis illness in a previously healthy adult. This report highlights the potential for serious complications and the devastating impact of pertussis even in young adults.

This case occurred during a substantial outbreak of pertussis in British Columbia, Canada, during 2000. This outbreak has been described elsewhere [12]. Compared with previous outbreaks, this outbreak was distinguished by an increased proportion of cases in adolescents and adults and a peak incidence in preteens and teens aged 10–14 years. This increased incidence was accompanied by an increase in the number of hospitalizations due to pertussis in older persons. During the 1996 outbreak of pertussis in British Columbia, 2% of all hospitalizations involved persons aged ≥10 years, but this rate increased to 14% in 2000. For persons aged ≥20 years, the rate of hospitalization per 100,000 persons in British Columbia increased >5-fold over successive outbreaks between 1990 (0.04 hospitalizations) and 2000 (0.23 hospitalizations).

Although 5 months of inpatient care may be the exception for treatment of adults with pertussis, the presently described case is noteworthy for its considerable medical and social costs in addition to the personal suffering and professional loss inflicted. The case we describe and recent epidemiological trends witnessed in British Columbia add weight to the argument for providing adults with booster immunization against pertussis.

References