CASE REPORT

Optic Atrophy Secondary to Preseptal Cutaneous Anthrax: Case Report

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ABSTRACT

Bacillus anthracis, the agent of anthrax, is a nonmotile, aerobic gram-positive rod that can form very resistant spores in economically poor environments. Anthrax can manifest as cutaneous, gastrointestinal, or inhalational form. Cutaneous anthrax, caused by direct skin contact, presents with eschar, lymphadenopathy, and a febrile illness. The face and eyelids are most commonly involved in cutaneous anthrax. A 45-year-old man was admitted to our clinic with high fever and swelling of the right eyelid. One day later on re-examination, formation of ulcerous lesions in the right medial canthal region was observed, with general oedema in the upper and lower eyelids. The patient was evaluated as having cutaneous anthrax and medical treatment was continued until the 28th day; he was discharged from the hospital with no loss of vision. He returned for a follow-up examination after 2 months, with decreased visual acuity (<logMAR 1.0). In the fundus examination, optic atrophy was observed. The patient was evaluated as having toxic optic neuropathy.

Keywords: Anthrax, optic atrophy, preseptal cellulitis

INTRODUCTION

Anthrax is a zoonotic disease caused by infection with the gram-positive bacterium *Bacillus anthracis*. It is primarily a disease of domesticated and wild herbivores, but can be transmitted to man by contact with infected animals or their products. The human disease continues to be prevalent in developing countries.¹ The forms of the disease in humans include cutaneous, respiratory, meningoencephalitic, and gastrointestinal; the cutaneous disease accounts for most of the cases of anthrax in humans.^{2,3}

Meat and leather industry workers can contract a pulmonary form of anthrax, but cutaneous, gastrointestinal, and meningoencephalitic forms are usually nonindustrial.^{1,4–6} Transmission may result from ingestion of grossly contaminated or undercooked meat or bones of infected animals, contact with hair and wool of infected animals, introduction of the

bacteria by nicks/incisions in open parts of the body, or inhalation of the spores.⁷ The most common method of transmission is infection of the skin because of accumulation of spores under the nails of the hands or feet. It has been suggested that some fly species may act as vectors, transporting the infection from infected animals to humans.⁸ The face and eyelids are most commonly involved.

In this study, we report a case of anthrax that was initially diagnosed as preseptal cellulites, with typical black and necrotic eschar tissue in the medial canthal region, in which optic atrophy developed after successful medical treatment.

CASE REPORT

A 45-year-old man living in a rural region of Turkey was admitted to our clinic with a complaint of high

fever and swelling of the right eyelid. The patient was diagnosed with preseptal cellulitis and amoxicillin + clavulanate 1000 mg tablets 2×1 , topical fusidic acid cream 3×1 , and ofloxacin drops 5×1 treatments were applied. One day later, the patient was admitted to our clinic for re-examination because of non-decreasing fever, increase in the current eyelid swelling, and formation of ulcerous lesions in the right medial canthal region. When the patient was evaluated, the upper and lower eyelids were generally oedematous and there was a 2.5×2 cm patch of black necrotic tissue in the medial canthal region (Figure 1).

The anterior segment structures were evaluated as normal when the eyelids were opened with a lid ecartor during the ocular examination. Pupillary response, tonometry, anterior segment biomicroscopy, and ophthalmoscopy examinations were evaluated as normal. The patient's visual acuity was 10/10 bilaterally. There was no trauma in his history but he had slaughtered an animal with anthrax, and during this slaughter he was bitten by a fly. Findings from the patient were body temperature 39.2°C (normal values $37\text{--}37.4^{\circ}\text{C}$), heart rate 110 beats per minute (bpm) (normal values 60–80 bpm); white blood cell count of 19,700 (normal values 4000–10,000 cells/mcL), and erythrocyte sedimentation rate (ESR) of 36 mm/h (normal values 0–20 mm/h). There were no other pathological findings. Orbital computed tomography (CT) scans and ultrasound revealed no additional pathology. Microscopic examination of the material scraped from beneath the edge of the eschar, without disturbing the remaining eschar, revealed gram-positive rods. *Bacillus anthracis* was found in the culture of the scraped material. The microorganism was sensitive to penicillin, ampicillin, tetracycline, ciprofloxacin, doxycycline, erythromycin, and vancomycin. We used intravenous penicillin G, 13 million units per day, in the treatment of the patient.

The patient was followed in the infectious diseases clinic and medical treatment was continued until the

28th day, when the patient's leukocyte/sedimentation values decreased to a normal level. When the patient was being discharged from the hospital, his visual acuity was 10/10 bilaterally. He was discharged with advice to return for a follow-up examination after a week. He came to the hospital as an outpatient after 2 months. Granulation tissue had developed on the lesion and there was a visual loss in the affected eye (Figure 2). His visual acuity was <0.1 ($<\log\text{MAR } 1.0$); anterior segment structures were evaluated as normal and in the fundus examination optic atrophy was observed (Figure 3). The patient was evaluated as having toxic optic neuropathy and followed up in the oculoplasty unit in our department.

DISCUSSION

In this study, we report for the first time of a case of optic atrophy after anthrax preseptal cellulitis.

Periorbital anthrax primarily involves the upper eyelid. It spreads toward the lower eyelid and the cheek. The first sign of cutaneous anthrax is severe and generalised oedema. Translucent epidermal bullae and vesicles surround the lesion in this early phase. Afterwards, oedema begins to reduce in the characteristic necrotic tissue areas, and pustules develop on the lesion. This structure is known as eschar or anthrax. The granulation tissue, which will heal with scarring, develops on the eschar within 2 to 4 weeks.² If the oedema spreads towards the neck, it may cause an obstruction in the airway that requires tracheotomy. Serious complications such as septicaemia and meningoencephalitis can be observed in untreated patients. Meningoencephalitis may develop with any type of anthrax, including cutaneous, gastrointestinal, and inhaled, but in some cases the portal of entry has not been identified.⁴

Anthrax meningoencephalitis presents with fever, headache, nausea, vomiting, and altered mentation

FIGURE 1 The appearance of black necrotic tissue in the medial canthal region.

FIGURE 2 The appearance of granulation tissue.

FIGURE 3 Optic atrophy in fundus evaluation.

(i.e. confusion or agitation) along with symptoms related to the source of infection (e.g. cutaneous, gastrointestinal, or inhalational). Clinical signs include fever (38.3–41 °C), malaise, meningeal signs (i.e. nuchal rigidity, Kernig and Brudzinski signs), hyperreflexia (often with unilateral or bilateral Babinski sign), and confusion, delirium, stupor, or coma. Other less frequent findings include papilloedema, cranial neuropathies, focal or generalised seizures, myoclonus, fasciculations, generalised rigidity, lateralising long-tract signs, and coma with decerebrate posturing.⁴ In our patient, none of these clinical and radiological signs were observed.

Another clinical condition that can cause optic atrophy is septic thrombosis of the cavernous sinus. Because of the close anatomical relationship, spreading infection in the nose, sinuses, ears, or teeth is a potentially high risk of cavernous sinus thrombosis.^{9,10} The clinical presentation may be varied, but the presence of periorbital oedema, chemosis, proptosis, or ocular motility disorders including ophthalmoplegia is frequently seen in almost all patients.¹¹ Hypoaesthesia in the distribution of the first and second divisions of the trigeminal nerve may

occur.¹² Although uncommon in cavernous sinus thrombosis, loss of vision may develop secondary to optic nerve inflammation and the disturbance of retinal blood supply. The most frequent complication of cavernous sinus thrombosis, despite successful treatment, is ocular motility disorder.¹²

Orbital cellulitis can be contracted from dental abscess, orbital trauma, periocular surgery, panophthalmitis, septicaemia, and immunosuppression, but in the majority of the cases, these are complications of maxillary sinusitis.¹³ According to the Chandler Classification of Orbital Cellulitis,¹⁴ inflammatory oedema of the eyelids, proptosis, chemosis, visual loss, reduced eye movements, complete ophthalmoplegia, cavernous sinus thrombosis, and meningism can be observed in orbital cellulitis. Loss of vision is due to optic atrophy secondary to increased intraorbital pressure and compressive neuropathy. In our patient, none of these clinical and radiological signs were observed.

Human vaccines against anthrax have been developed and are currently given to military personnel and occupational groups at risk of exposure to the pathogen.¹⁵ They can be divided into two

groups: live-attenuated spores and nonliving subunit vaccines. Although in Payne et al.'s study they did not find any association between optic neuritis and anthrax vaccinations in the US military,¹⁶ Kerrison et al. reported two cases with optic neuritis after anthrax vaccination.¹⁷ They thought that this may have been the result of a potential immune-mediated response to optic nerve antigens that was stimulated by the vaccine. In our opinion, based on our case, a potential immune-mediated response to optic nerve antigens after *Bacillus anthracis* infection is responsible for the optic nerve atrophy. Kerrison et al.¹⁷ reported that they had successfully treated one case with intravenous methylprednisolone, whereas the other one needed chronic immunosuppression to maintain his vision. In our case, the patient was referred to our clinic after optic atrophy had developed; no corticosteroid therapy was given to our patient because of the late presentation at the sequel phase of the disease.

One weakness of our report is that we do not have any data regarding the course of the patient's vision change due to the lack of follow-up for 2 months. It is possible that during this period, the patient might have developed ischaemic optic neuropathy.

Periocular localisation of anthrax can result in serious complications such as meningoencephalitis, cavernous sinus thrombosis, orbital cellulites, and postinfection immune-mediated optic neuritis, which would require the clinician's familiarity with the manifestations of this disease in order to recognise it early and treat it successfully.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Note: Figures 1–3 of this article are available in colour online at www.informahealthcare.com/oph.

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