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Case Report

Bilateral Amaurosis Caused by *Salmonella enteritidis* Infection

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A B S T R A C T

The aim of this paper was to show the potential of Salmonella enteritidis infection to eventually result in visual impairment. A case of salmonellosis in a 6-year-old boy, caused by intake of a cake made from eggs infected with Salmonella enteritidis, is presented. Prolonged duration of the disease was followed by complete remission of neurologic complications and persistent amaurosis with bilateral optic nerve atrophy. A severe form of Salmonella enterocolitis with neurologic involvement can lead to optic nerve lesion with consequential loss of vision.

Key words: *amaurosis, Salmonella, infection enteritidis*

Introduction

Ophthalmic complications occur in a small number of patients with Salmonella induced fever. Duke Elder reports on iritis, uveitis, choroditis and panuveitis[1]. Encephalitis and bilateral optic neuritis have been described in patients with typhoid fever, in whom the vision recovered to normal[2]. There is also a report on a case of bilateral optic neuritis in a 3-year-old girl, consequential to enteric typhoid, which entailed transitory loss of vision and restitution of vision within 3 months[3].

Case Report

M.Š., a 6-year-old boy, was admitted as an emergency to a regional hospital several hours after he had eaten a lot of cream-puffs. All family members except the boy's grandfather (who was the only one free from symptoms) ate the cake. Soon after ingestion, the boy developed symptoms of alimentary intoxication including elevated body temperature, abdominal pain, tonoclonic spasms with loss of consciousness, foamy salivation, and bluish discoloration around the mouth and eyes. The seizure lasted for more

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than 15 minutes. Upon admission, the patient received a sedative infusion, but the loss of consciousness persisted for 4 days, with hematemesis and melena, cold extremities and dilated pupils, without pupillary light reflex. Computed tomography (CT) of the brain revealed diffuse brain edema. Ophthalmologic examination showed papilledema bilaterally, more pronounced on the left. The patient's state of consciousness made the measurement of visual acuity impossible.

The patient had been treated with polyvalent antimicrobial therapy (penicillin G potassium and gentamicin) until coproculture positive for *Salmonella enteritidis* was obtained. Antimicrobial therapy for Salmonella according to antibiogram consisted of chloramphenicol, followed by sulfamethoxazole + trimethoprim, administered along with an osmotic diuretic (20% mannitol) and hy- pertonic carbohydrate solution (10% dextrose). Psychoneurologically, after 4-day coma, deep somnolence and sopor persisted for another 5 days. Stools were loose and green, positive for *Salmonella enteritidis*.

Antiedematous therapy was continued, with correction of electrolyte disbalance, seizure prophylaxis and antimicrobial therapy (chloramphenicol, sulfa- methoxazole + trimethoprim). This therapy resulted in slow recovery of the patient's condition, he regained consciousness but was found to be blind. Bilateral mydriasis and lack of photomotor reflexes persisted.

The patient was transferred to the Department of Pediatrics, Zagreb University Hospital Center. On admission, the boy was conscious and communicative, providing appropriate answers to the questions posed, afebrile and of normal vital functions. Ptosis of the right upper lid reached to a half of the pupillary opening. The child looked very exhausted and weak. Active motoricity was generally reduced.

Laboratory findings: ESR 10 mm/h; erythrocytes 3.90 million; hemoglobin 111 g/l; platelets 350 thousand; leukocytes 6.6 thousand. Stool parasitology was negative. *Salmonella enteritidis* (sensitive to chloramphenicol, norfloxacin, sulfamethoxazole + trimethoprim) was bacteriologically verified. The finding of cerebrospinal fluid (CSF) cytology was normal. CSF protein electrophoresis was normal, with the exception of slightly increased prealbumin and α_1-globulin. IgG class was not demonstrated by use of specific antiserum. Contrast CT of the brain showed signs of blood-brain barrier (BBB) impairment, strongly suggestive of encephalitis. Generally, electroencephalogram was diffusely slow and inadequately formed for the boy's age, with generalized slow irritative-dysrhythmic changes, more pronounced on the left. Paranasal sinus x-ray was normal.

Ophthalmologic examination performed one month after Salmonella intoxication showed no visual function improvement. Ophthalmoscopy revealed distinctly delineated papillae bilaterally, temporally paler, grayish in color. The blood vessels, macular area and retinal periphery were normal. Measurement of the visual evoked potentials (VEP) to nonstructured light stimulus of both eyes showed no P100 wave.

On discharge from the hospital, bilateral amaurosis persisted, with otherwise completely normalized neurologic status. On control neuro-ophthalmologic examinations performed at one and two years there was no improvement of visual function.

Discussion

A severe form of *Salmonella* enterocolitis with dehydration, electrolyte disbalance, and probable development of hypovolemic shock occurred in this 6-year-old boy after the intake of a cake made from eggs

infected with *Salmonella enteritidis*. The consequential brain edema was exacerbated by relapsing seizures, while the development of encephalitis and possible spasm of the calcarine branches of the posterior cerebral artery led to a BBB compromise[4]. Hypovolemic shock and relapsing epileptic seizures entailed visual cortex hypoxia[5].

Similar cases of acute encephalopathy apart from visual impairment following *Salmonella enteritidis* intoxication have been described as a separate entity, i.e. nontyphoidal salmonella encephalopathy[6,7]. The development of amaurosis in our patient remained obscure. Brain CT obtained in acute condition indicated no septic metastatic foci that might point to the causative agent dissemination in the central nervous system (CNS). In addition, the CSF finding at the time of acute disease was missing. However, the finding of initial bilateral papillary edema pointed to a direct toxic-infectious effect of *Salmonella enteritidis* on optic nerves which, together with ischemia, led to impairment of the visual function at the peripheral level, with subsequent bilateral optic nerve atrophy.

On the other hand, papilledema may have been caused by the brain edema coupled with the increased intracranial pressure, as laboratory and CSF cytology findings obtained in the subacute course of the disease were normal. Had the amaurosis been caused exclusively be ischemic lesions of the occipital cortex, the optic disk atrophy would have been more difficult to explain. Thus, the question of the possible combined action of the occipital cortex ischemic lesion and optic nerve peripheral damage remains open. On differential diagnosis, the administration of chloramphenicol can be associated with vision impairment in the form of ischemic neuropathy, however, most of these patients were on prolonged therapy for chronic infection and vision impairment developed after 3 to 8 months[8,9]. The ophthalmic complication accompanying severe salmonellosis caused by *Salmonella enteritidis* was additionally exacerbated by the prolonged duration of the severe form of the disease[10]. According to literature data, cases of multiple brain abscesses due to *Salmonella enteritidis* infection have been observed, but thus induced amaurosis, let alone permanent loss of vision, has never been reported. Transitory blepharoptosis during typhoid fever may be caused by thrombotic infarcts that affect the nuclei or fascicles of the oculomotor nerves or both in the brainstem, or (more likely in our case) by cerebral hemispheric dysfunction[11,12].

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OBOSTRANA SLJEPOĆA UZROKOVANA INFEKCIJOM BAKTERIJOM *SALMONELLA ENTERITIDIS*

S A Ž E T A K

Cilj rada bio je prikazati oštećenje vida uzrokovano infekcijom bakterijom *Salmonella enteritidis*. Salmoneloza se razvila u šestogodišnjeg dječaka koji je jeo kolače pripremljene jajima inficiranima salmonelom. Nakon dužeg liječenja došlo je do cjelovitog oporavka neuroloških komplikacija uz obostrano sljepilo s atrofijom oba vidna živca. Težak oblik salmoneloze s neurološkim komplikacijama može zahvatiti vidni živac i vidne putove s posljedičnim gubitkom vida.