



## CASE REPORT

### PAPILLITIS: A RARE COMPLICATION OF SEVERE SEPSIS

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#### ABSTRACT

A 12-year-old girl was admitted to the emergency room as a result of a vehicle accident with a large tissue defect in the gluteal and perineal region. She was diagnosed as having sepsis and on the 23rd day of her intensive care unit stay she complained of a sudden loss of vision. On physical examination, her pupils were mid-dilated and pupillary reflexes were bilaterally sluggish. The Marcus Gunn sign was negative. Fundoscopic examination revealed bilateral papilledema with peripapillary and periretinal hemorrhage. Cranial diffusion and angiographic magnetic resonance imaging (MRI) studies showed no pathology. MRI examination of the optic nerve was normal. Papillitis was prediagnosed. Although patients in severe sepsis are prone to develop papillitis, the diagnosis of papillitis in these patients is very rare. As many of these patients are sedated, or have an altered mental status, they are not able to express the symptoms of papillitis. In critical care practice, periodic fundoscopic examination should be considered for the early diagnosis and the detection of papillitis in septic patients.

**Keywords:** Sepsis, Papillitis, Loss of vision, Intensive care unit

### PAPİLLİT: AĞIR SEPSİSİN NADİR GÖRÜLEN KOMPLİKASYONU

#### ÖZET

12 yaşında kız çocuğu araç dışı trafik kazası sonucu acil servise gluteal ve perine bölgesinde geniş doku defekti ile başvurdu. Sepsis tanısı konulan hastada yoğun bakımdaki 23. gününde ani görme kaybı gelişti. Fizik muayenede, pupiller middilata, pupil refleksi her iki tarafta zayıflamış olarak değerlendirildi. Marcus Gunn işareti negatifti. Fundoskopik muayenede bilateral papil ödemi, peripapiller ve periretinal kanama saptandı. Kranial difüzyon ve anjiyografik MRI'da patoloji saptanmadı. Detaylı MRI görüntülemeye optik sinir normal değerlendirildi. Papillit öntanısı konuldu. Sepsisli hastalarda papillit tanısı nadirdir. Sepsisli hastalar papillite yatkındırlar. Bu hastaların çoğu sedatize olduğundan ve metntal durumları uygun olmadığından genelde semptom vermezler. Yoğun bakım ünitelerinde, periyodik fundoskopik muayenelerin yapılmasının papillitin hem erken teşhisinde hem de insidansının belirlenmesinde faydalı olacağı kanısındayız.

**Anahtar Kelimeler:** Sepsis, papillit, görme kaybı, yoğun bakım ünitesi

#### INTRODUCTION

In severe or complicated sepsis, there is impaired organ system function or inadequate tissue perfusion, manifested clinically as many morbid states<sup>1</sup>. Over the past two

decades, major advances in the understanding of the pathophysiology of sepsis have resulted in the development of novel therapies, leading to better survival rates<sup>2</sup>. As a result of these improvements in the pathophysiology and the

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outcome of sepsis, many organ dysfunctions are able to be determined<sup>3,4</sup>. We report a case of a 12-year-old girl with severe sepsis complicated by bilateral papillitis.

## CASE REPORT

A 12-year-old girl was admitted to the emergency room after a vehicle accident. On physical examination, she was alert and oriented, with a pulse of 130 beats/min in sinus rhythm. Her blood pressure was 100/70 mmHg. Computerized tomography examination of the cranium, thorax and abdomen demonstrated no pathology, but she had a left tibia fracture, left popliteal artery thrombosis, large tissue defect (affecting the skin, the subcutaneous tissue and the muscle) of the right pelvis including gluteus maximus muscle, right flank and inguinal region (30X30 cm in diameter), the 1/4 distal part of the vagina, the clitoris and right lateral part of the perine. The external urinary meatus was intact. On the same day, she was operated for an internal fixation of the left tibia. Debridement of the gluteus maximus and colostomy were performed to avoid further contamination of the tissue defects. Postoperatively, she was admitted to the surgical intensive care unit. On admission, she was oriented, cooperative, with a pulse of 120 beats/min in sinus rhythm, a blood pressure of 124/78 mmHg, a respiratory rate of 14/min and an axillary body temperature of 37.7°C. Arterial blood gas values, while the patient was breathing 5 L/min oxygen with a face mask, were PaO<sub>2</sub> 163.5 mmHg, PaCO<sub>2</sub> 31.7 mmHg, pH 7.43 and base deficit -2.5 mmol/L. Laboratory investigations were normal other than the followings: sodium 133 mEq/L, potassium 2.99 mEq/L, lactate dehydrogenase 999 IU/L and alkaline phosphatase 405 IU/L. Analgesia was maintained with meperidine i.v. PCA (Patient controlled analgesia). Fluid therapy was administered to achieve a central venous pressure of about 10 mmHg. Total parenteral nutrition was initiated. Empiric i.v. antibiotherapy including clindamycin, ampicillin and gentamicin was begun. The debridement of the tissue defects of the right gluteus maximus, vagina and anus was

performed in every 48 hours, under general anesthesia. On the 3<sup>rd</sup> day of ICU admission, her axillary body temperature increased to 38.8°C. Cultures of the debris materials were positive for pseudomonas aeruginosa and E. Coli. Antibiotherapy was then arranged as gentamicin, ceftriaxone and metronidazol i.v. On the 5<sup>th</sup> day of admission, her blood cultures became positive for pseudomonas aeruginosa and E. Coli, her urine cultures became positive for non-albicans candida. Meropenem, fungisone and tobramycin i.v. were administered. During this period, she was febrile (peak axillary temperature=39.8°C), and unresponsive to antipyretics. Tachycardia was sustained between 130-140 beats/ min after the 5<sup>th</sup> day of admission.

On the 7<sup>th</sup> day of her stay in ICU, morphine HCl i.v. infusion was started for analgesia, instead of meperidine i.v. PCA. The debridement of the tissue defects was performed in every 48 hours, under general anesthesia. On the 16<sup>th</sup> day, cultures from the debris materials became positive for aspergillus and non-albicans candida. Trimethoprim-sulfamethoxazole i.v. was added to the triple antibiotherapy.

She became progressively agitated and 1 mg/day lorazepam p.o. was administered. On the 23<sup>rd</sup> day of her stay in ICU, her body temperature decreased to normal range. Her blood pressure was in normal ranges and the heart rate was 135 beats/min. On the same day, she complained of a sudden loss of vision. On physical examination, pupils were mid-dilated and pupillary reflexes were bilaterally sluggish. The Marcus Gunn sign was negative. Fundoscopic examination revealed bilateral papilledema with peripapillary and periretinal hemorrhage. Visual acuity could not be assessed since the patient was not cooperative. Physical and CSF examination (direct and microbiological examination) revealed no signs of meningitis. Cranial diffusion and angiographic magnetic resonance imaging (MRI) studies showed no pathology. MRI examination of the optic nerve was normal. Papillitis was prediagnosed



and prednol 32 mg i.v. was administered 3 times a day.

On the 25<sup>th</sup> day, her body temperature increased to 40°C, her respiratory rate was 38 / min, hypoxemia and metabolic acidosis requiring bicarbonate therapy developed. She was sedated and intubated endotracheally. Mechanical ventilatory support with SIMV mode was initiated. The day after, she became hypotensive (60/30 mmHg) and unresponsive to inotropic administration. She died the same day.

## DISCUSSION

Papillitis is a general term implying inflammation, degeneration or demyelination of the optic nerve<sup>5</sup>. It is indistinguishable ophthalmoscopically from papilledema but papillitis is accompanied by a dramatic decrease in visual acuity and an afferent pupillary defect is readily apparent. The causes of papillitis include demyelinating diseases (e.g., multiple sclerosis, postinfectious encephalomyelitis), systemic infections (viral - polio, flu, mumps, measles - and bacterial ), nutritional and metabolic diseases (diabetes, pernicious anemia, hyperthyroidism), Leber's Disease, secondary complications of inflammatory diseases (e.g., sinusitis, meningitis, tuberculosis, syphilis, chorioretinitis, orbital inflammation), toxic reactions (to tobacco, methanol, quinine, arsenic, salicylates, lead)<sup>5</sup>.

Our patient was diagnosed as having bilateral papillitis. In the literature, this is the only case report about papillitis in a septic patient. There is one case report suggesting that in patients with systemic *Bartonella henselae* infection, optic disk edema was as an early sign<sup>6</sup>. The most probable cause of papillitis in our case report was sepsis. Bilateral inflammatory involvement of the papilla indicated that this was a complication of a systemic disease. We excluded the presence of meningitis on physical and CSF examination, and the presence of sinusitis on CT scan. She had no known nutritional or metabolic diseases and no history of exposure to toxic agents. The incidence of papillitis in septic patients is not known. Most of the

patients in severe sepsis are sedated and mechanically ventilated as a result of many organ system dysfunctions. In these patients, the diagnosis of papillitis may be difficult because the sedated patient cannot express the loss of vision. As a result, the incidence of papillitis may be higher than expected in septic patients.

The exact mechanism of papillitis in septic patients is not clear. It may be due to the direct invasion and infection of the papilla with a microorganism. Other effects of sepsis such as the decrease in the perfusion of papilla due to arterial hypotension; or the development of a cellular stunning due to the decrease in either the uptake and the utilization of oxygen by the papilla would result in ischemic optic neuropathy rather than papillitis.

The causative agents of sepsis in our patient were aspergillus, non-albicans candida, pseudomonas aeruginosa and E. Coli. In this case, it is not easy to determine the causative agent of papillitis because positive blood cultures are present only in 40 % of patients with sepsis<sup>7</sup>.

In view of this case, it should be emphasized that patients in severe sepsis are prone to develop papillitis and since many of these patients are sedated and mechanically ventilated, they are not able to express the symptoms of papillitis. In critical care practice, periodic fundoscopic examination should be considered for the early diagnosis and the detection of papillitis in septic patients.

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