IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 6 Ver. 4 (June. 2018), PP 19-21
www.iosrjournals.org

Case Report Fulminant Meningococcal Meningitis without Meningeal Signs in a 22- Year Old Female.

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Abstract: *The case of a 22- year old female student who presented with a one week history of sore throat, joint pain and fever, and a two-day history of skin rash.She had no neck pain or neck stiffness and physical examination revealed no signs of meningeal irritation. She died four hours after presentation. Autopsy findings revealed thick purulent pus covering the meninges in keeping with pyogenic meningitis. Brain swab culture yielded Neisseria meningitidis. She had fulminant meningococcal meningitis with no meningeal signs. Clinicians usually rely on meningeal signs to make a diagnosis of meningitis. Meningeal signs however have been shown to have a low sensitivity in making a diagnosis of meningitis, and may be absent in the presence of meningitis. Clinicians should therefore not only rely on the presence ofthese signs to suspect meningitis; they should have a high index of suspicion, particularly during an ongoing outbreak of meningitis.*

Key words: *Meningococcal meningitis,meningeal signs.*

Date of Submission: 26-05-2018 Date Of Acceptance: 09-06-2018

I. Introduction

Meningococcal meningitis is an inflammation of the meninges caused by Neisseria meningitidis. It is a serious infection that can cause brain damage and is fatal in 50% of cases if untreated [1].Neisseria meningitidis has the potential to cause large epidemics.The extended meningitis belt of sub-Saharan Africa that stretches from Senegal in the West to Ethiopia in the East has the highest rates of the disease.Nigeria is located within this meningitis belt and has been experiencing epidemics of meningococcal meningitis during the dry dusty Harmattan season.

The common clinical features of meningococcal meningitis are fever, headache and neck stiffness and clinicians usually rely on the presence of neck stiffness and other meningeal signs to make the diagnosis.

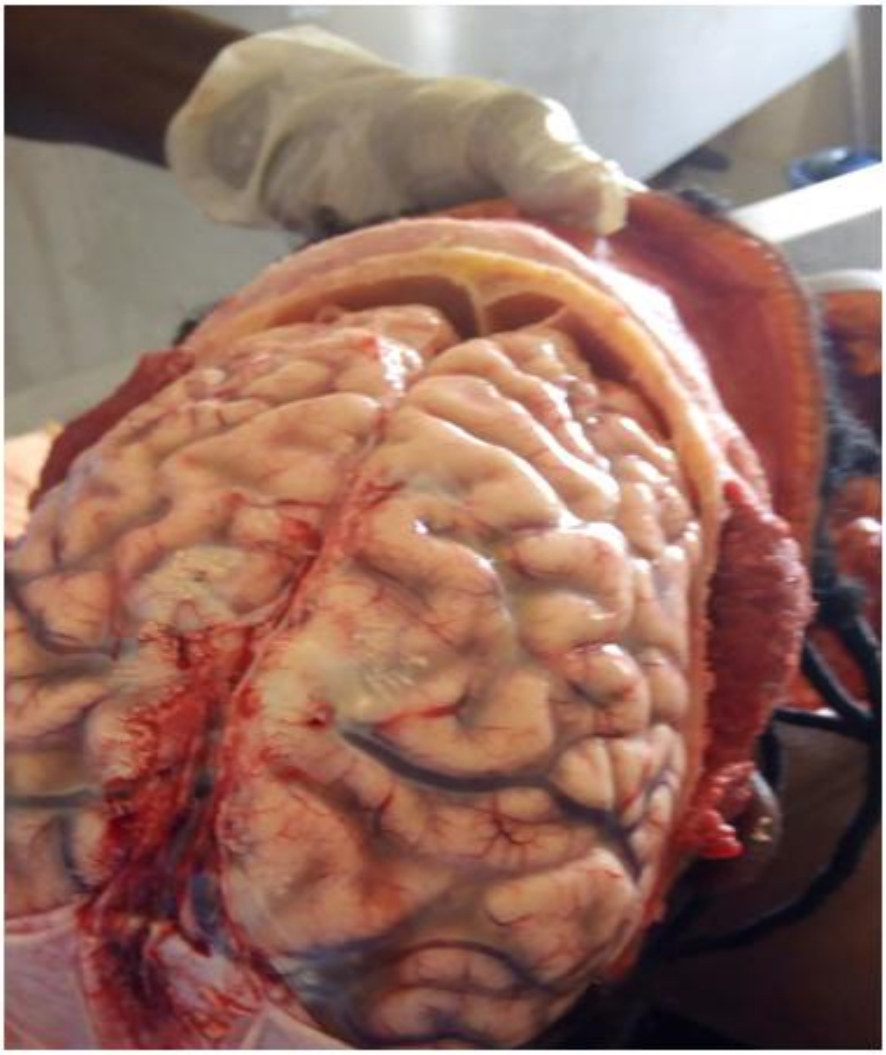
This is a case report ofa 22-year old previously healthy studentwho had fulminant meningococcal meningitis withmeningococcemia but had nomeningeal signs.

II. Case Report

A 22-year old female student was referred tothe University of Abuja Teaching Hospital from a private clinic in the Federal Capital Territory (FCT). She presented with a one week history of sore throat, joint pain, fever, and a two day history of skin rash.The fever was of high grade with no chills or rigors. There was associated headache but no history of neck stiffness, vomiting or photophobia. She had parenteral Chloroquine at the referral hospital with no improvement.The rash appeared a day after she completed the antimalarial and was thought to be an adverse drug reaction, hence the reason for the referral.

On physical examination she was acutely ill-looking, febrile with an axillary temperature of 38.8℃. There were wide spread maculopapular rashes involving the extremities and the trunk with bilateral conjunctival hemorrhages. The pulse rate was 142 beats per minute with a blood pressure of 100/70 mmHg. There was no neck stiffness and Kernig's and Brudzinski's signs were negative. An initial diagnosis of severe adverse drug reaction was made and patient was commenced on intravenous fluids and hydrocortisone. Her clinical condition deteriorated rapidly and the infectious diseases unit was invited to review the patient and made a diagnosis of meningococcal meningitis with Meningococcemia. The patient however died 4 hours after she presented to the casualty unit.

At autopsy, the Central Nervous System examination revealed thick cream colored purulent pus covering the meninges with associated marked cerebral edema evidenced by flattening of the gyri and narrowing of the sulci [figure 1]. Additional findings included bilateral Adrenal hemorrhages, septic spleen and bilateral renal petechial hemorrhages.The cause of death was pyogenic meningitis, septicaemic shock with multiple organ failure. Brain swab obtained during autopsy cultured Neisseria meningitidis.



III. Discussion

Meningococcal meningitis is caused by the bacterium Neisseria meningitidis. It is a Gram-negative diplococcus that may have a polysaccharide capsule or it may be unencapsulated[2]. Based on the biochemical composition of the capsule, meningococci can be divided into 13 serogroups, but only six of these (A, B, C, W-135, Y and X) are currently associated with significant pathogenic potential [3, 4].

Illness believed to be meningococcal disease was first reported in the 16th century. The first definitive description of the disease was by Vieusseux in Switzerland in 1805 [5].Meningococcal disease occurs worldwide as an endemic disease with seasonal variations.The African Meningitis Belt, originally characterized by Lapeysonnie in 1963 has the highest annual incidence of meningococcal disease in the world with superimposed frequent epidemics that constitute a major public health burden [6].It stretches from Senegal in the West to Ethiopia in the Eastand is made up of twenty six countries with a population of over 430 million people.

The current outbreak of meningococcal meningitis in Nigeria started in mid-December 2016 and the Nigerian Center for Disease Control (NCDC) has reported that as of 2nd June 2017, a total of 14,473 suspected cases have been reported from 25 states of the country, with a death toll of 1558 [7]. The case fatality rate of this current outbreak is 8% , which is lower when compared to the case fatality rate of 10.7% recorded in the outbreak that occurred in 1996 [8].

Meningitis is a common life-threatening medical emergency. Rapid and accurate evaluation by history and clinical examination is helpful to guide further specific investigation and treatment. The overall mortality in patients suffering from meningococcal disease is increased from about 10% to more than 50% when the diagnosis is delayed or missed. Kernig's sign, Brudzinski's sign, and nuchal rigidity are bedside diagnostic signs used to evaluate suspected cases of meningitis.These signs are usually present in patients that are awake except infants, the elderly, and the immunosuppressed [9].To elicit the Kernig's sign, the patient is kept in supine position, hip and knee are flexed to a right angle, and the knee is slowly extended by the examiner. The

appearance of resistance or pain during extension of the patient's knees beyond 135 degrees constitutes a positive Kernig's sign. Reflex flexion of the patient's hips and knees after passive flexion of the neck constitutes a positive Brudzinski'ssign. Clinicians rely on these signs to suspect the presence of meningitis. Howeverstudies have shown that they have a low sensitivity and a high specificity in diagnosing meningitis.In 1991, a prospective study by Uchihara and Tsukagoshi reported a sensitivity of 9% and specificity of 100% for Kernig's sign [10]. The sensitivity and specificity was 15% and 100%, respectively, for nuchal rigidity in the same study. Another study by Thomas et al analyzed 297 adults with suspected meningitis and reported a sensitivity of 5% and specificity of 95% for both Kernig's and Brudzinski's signs [11]. These studies show that bothKernig's and Brudzinski's signs are not very sensitive for detecting meningitis and, therefore, when absent, should not be inferred as there is no evidence of meningitis. Their high specificity howeversuggests that if Kernig's or Brudzinski's sign is present, there is a high likelihood for meningitis. This case confirms the low sensitivity of meningeal signs in making a diagnosis of meningitis. Clinicians should therefore have a high index of suspicion, and not only rely on the presence of these signs, particularly during an ongoing outbreak of the disease.

IV. Conclusion

Meningeal signs have a high specificity when present and are reliable in making a diagnosis of meningitis. However, they have a lowsensitivity; they may be absent in the presence of meningitis even in young healthy adults. Clinicians should therefore have a high index of suspicion and not only rely on their presence to make a diagnosis of meningitis.

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