HYPOPYON IN SEVERELY MALNOURISHED CHILDREN WITH MEASLES

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INTRODUCTION

Measles is an acute, communicable, disease characterized by cough, coryza, conjunctivitis, a confluent erythematous maculopapular rash and Koplik's spot. The measles virus is classified as RNA containing paramyxovirus with only one antigenic type known.[1,2] Acute measles infection at an early age is associated with more complications and increased mortality. Complication from measles can occur in almost every organ system. Complication rates are increased by immune deficiency disorders, malnutrition, vitamin A deficiency, intense exposures to measles and the lack of previous measles vaccination.[3]

In developing countries with high birth rates (up to 50 per 1000 persons per year), the measles incidence was between 20 and 30 cases per 1000 persons per year before immunization activities.[4] In Indonesia, measles is the fifth of the ten major disease of infant (0.7%) and the fifth of the ten major disease of 1-4 year old children (0.77%).[5] The common complication in measles is conjunctivitis. Severe conjunctivitis may be accompanied by corneal ulceration, particularly in children with vitamin A deficiency. Ulcerative keratitis and blindness were observed in 0.3% children with measles.[3] In some African countries in development, around 1% of all children with measles will sustain permanent, severe ocular damage of corneal origin.[6]

Measles are associated with reduction in serum retinol concentrations and may induce overt vitamin A deficiency. Measles associated with vitamin A deficiency is one of the most common causes of acquired blindness in children in developing countries.[3] Vitamin A deficiency was the commonest cause of measles associated ulceration. In Africa, 1 to 4% of hospitalized children with acute measles develop true corneal ulceration as distinct from punctuate keratitis. Corneal ulceration induced by vitamin A deficiency usually occurred after measles (76% of cases).[7]

In measles, virus is present in the corneal ephitelium and conjunctiva, there is both a measles keratitis and a conjunctivitis, it also can occurred corneal ulcer. In a severe corneal ulcer, there is pus inside the anterior chamber behind the cornea and in front of iris called hypopyon. Severe conjunctivitis in measles can be worsening become hypopyon and panophtalmitis until blindness too.[8]

The objective of this case was to report case of hypopyon in a child with measles.

CASE REPORT

V, a 2 year 8 month old female patient, came to Emergency Departement of Dr.Soetomo Hospital on October 22nd 2016 with chief complaint of having white spot in the right eye. She was suffered from white spot in the right eye about 4 days before admission. The complaint of white spot also felt in the left eye from 4 days before admission, but it was got better.

There was secretion in both eyes, pus in the right eye and she also felt pain in the right eye. Both eyes look hyperemia. The hyperemia in her eyes became worsening with secretion in both eyes. There was no worsening vision and headache. 15 days before admission she was suffering from measles. She got fever then the rash appeared at the whole body. Rash came firstly at her face on the third day of fever. A day after the rash appeared at her face, the rash spread at her whole body. The conjunctivitis occurred in the fourth day of illness. She also got cough and coryza. She never got medicine when she suffered measles. White spot in the both eye occurred 1 week after the rash disappear.

In her past medical history, she never experienced a complaint like this before. Neither family nor neighbor had the same complaint, no one around the patient got rash or hyperemia at the eyes. When she was one year old, she was diagnosed severe malnutrition. She got formula milk and red rice from public health center for about 1 year. She got vitamin A oral 2 times from public health center.

The patient is the youngest child of eight siblings. During pregnancy, the mother never consumed herbal medicine, didn't routinely control to her midwife. The patient was spontaneously delivered, term, supported by midwife with 3500 gram of birth weight, 50 cm of birth length, clear amniotic fluid, history of premature rupture did not exist, no data of head circumference because her mother did not remember. The immunization history was not complete, she was not immunized eccept for BCG and Polio.

The patient was never breastfed, she had been given formula milk since birth until the age of 2 years. After 1 year old, she had been given sugar water. Semi solids such as fine porridge had been firstly given from the age of 6 months

and followed by a rough porridge and rice at the age of 9 months. Family menu had been given from the age of 1 year.

According to the patient's mother, the growth and development of the patient seemed normal as their peers. Her mother didn't remember when her child firstly lifted her head and crawl. The patient sit at the age of 7 months, walking at the age of 1 year and speak at the age of 1 year and 6 month. The patient is 2 years 8 months old now.

The physical examination revealed an alert girl with body height 71 cm, body weight 7.2 kg, upper arm circumference 11 cm. vital sign showed that the respiratory rate was 22 times/minute, the pulse rate was 100-110 times/minute regularly, the body temperature was 37.1 ℃.

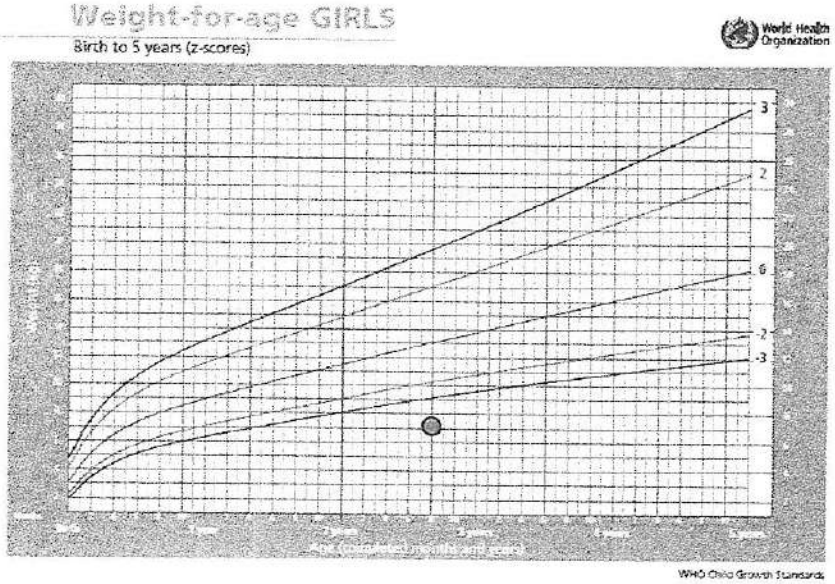

Figure 1. Growth chart showed body weight 7.2 kg, age 2 year 8 month. The percentile below -3.

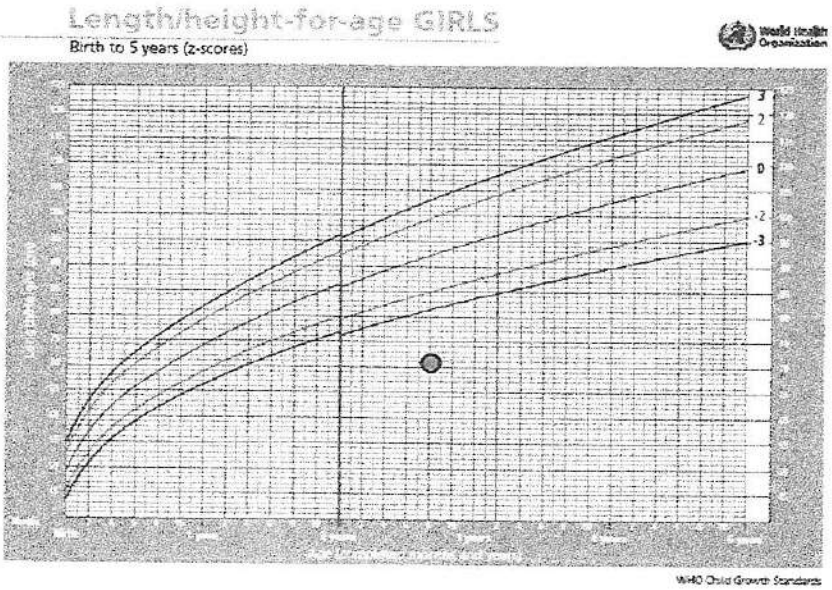

Figure 2. Grow chart showed height 71 cm, age 2 year 8 month. The percentile below -3.

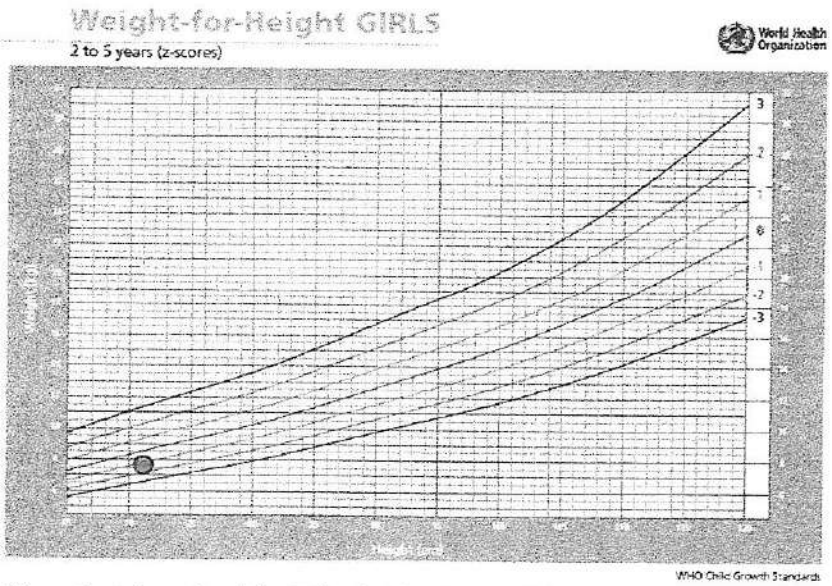

Figure 3. Grow chart showed weight 7.2 kg, height 71 cm and the percentile is -2.

Head and neck examination revealed no dyspnea, anemia, jaundice as well as cyanosis. There was white spot with pus on the right eye, from the mouth was founded moniliasis. The chest was symetric, no retraction on the observation, vesicular breath sound was equal on both lung. Neither rhales nor wheezing was heard. The heart revealed no murmur and no gallop rhythm. The examination of the abdomen showed that the abdomen was flat, no sign of ascites, the bowel

sound was normal, the liver and spleen were not palpable. The extremities were well perfused, no leg edema. From the skin presentation revealed hyperpigmentation in the patient's neck and face. From the chest x-ray revealed normal limit.

From the ophthalmology examination revealed hyperemia and secretion in the both conjunctiva, spasm both palpebra, infiltration in the both cornea with positive fluorecein test 6 mm x 4 mm in right cornea and 1 mm in left cornea, hypopyon in the right anterior chamber. Iris, pupil and lens of the right eye difficult to evaluate and normal in the left eye.

Initial laboratory finding revealed hemoglobin level of 9.8 g/dl, leukocytes 9.18 x 10^3/µL, platelets count 706 x 10^3/µL, sodium 143 mmol/l, potassium 4.1 mmol/l, chloride 108 mmol/l, CRP 29.47 mg/L. AST 54 u/l, ALT 51 u/l, BUN 5 mg/dL, creatinine serum 0.41 mg/dL, blood glucose 87 mg/dL, HbsAg rapid test non reactive.

Based on the history, clinical manifestation, laboratory finding, chest x-ray, the working diagnosis on admission was post measles infection with bilateral corneal ulcer and right eye hypopyon with moniliasis with wasted and severely stunted.

Patient was planned for work up hiv rapid test, tuberculin skin testing, acid-fast bacillus sputum testing and consulted to nutrition division. Ampicillin injection was administered 200 mg 6 times/day, nystatin drop 1 ml 6 times/day, Vitamin A 200.000 iu per oral. In the Emergency Department, the patient was consulted to ophthalmology department. The patient was planned to undergo eye swab culture and ocular ultrasonography. The patient got Moxifloxacin HCL eye drop 1 drops every hour for the right eye and 1 drops every 2 hour for the left eye, Natamycin eye drop 1 drops every 2 hour for the right eye and 1 drops 4 times a day for the left eye, Homatropine eye drop 1 drops every 12 hours for the both eyes. Ophthalmology department followed up in the ward.

Figure 4. Picture of the patient on the first day

On October 24th 2016 (2nd day of hospitalization), patient was still cough, the right eye was swelling. Vital sign showed the respiratory rate was 26 times/minute, the pulse rate was 106 times/minute regularly, the body temperature was 36.8 °C, peripheral oxygen saturation (SpO2) 99%. The nutrition status was wasted and severely stunted. The patient was given porridge 3 times/day equal to 150 kkal, pediasure milk 100 ml 5 times/day equal to 500 kkal, vitamin A 200.000 iu/day and vitamin B/C/E.

On October 27th 2016 (5th day of hospitalization), patient was still cough, no swelling on the right eye and hypopyon disappeared and the patient still got moniliasis in her oris. Vital sign showed the respiratory rate was 23 times/minute, the pulse rate was 110 times/minute regularly, the body temperature was 36.7 °C, peripheral oxygen saturation (SpO2) 99 %. From the examination revealed tuberculin skin test was negative and acid-fast bacillus sputum testing was negative. The patient got fluconazole oral and vitamin A was stopped.

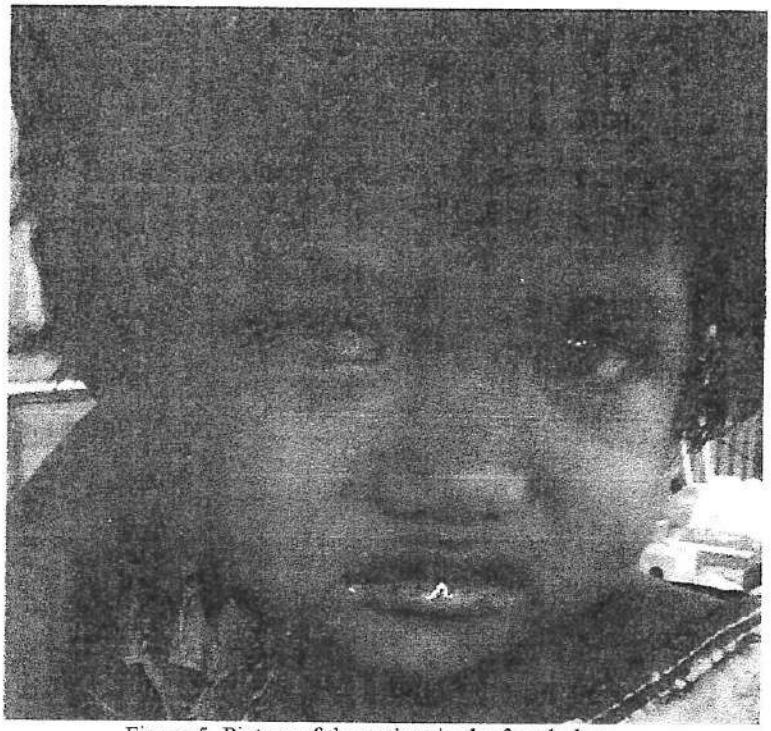

Figure 5. Picture of the patient in the fourth day

On October 29th 2016 (9th day of hospitalization), patient had no cough, no hypopyon and the moniliasis in her oris was already improved. Vital sign showed the respiratory rate was 24 times/minute, the pulse rate was 108 times/minute regularly, the body temperature was 36.7 °C, peripheral oxygen saturation (SpO2) 99 %. From the laboratory finding reveal HIV rapid test was negative. Patient discharged from the hospital.

On March 3rd 2017 patient came to ophthalmology outpatient clinic. From the ophthalmology examination revealed no hyperemia and secretion in the both conjunctiva, no spasm in the both palpebra, both cornea was intact, no hypopyon in the anterior chamber eye. Iris, pupil and lens was normal in the both eye. There was corneal leukoma in both eyes with normal vision. There was no specific therapy from ophthalmology division. The patient weight was 8 kg and the body height was 72 cm, from the nutrition status still wasted and severely stunted.

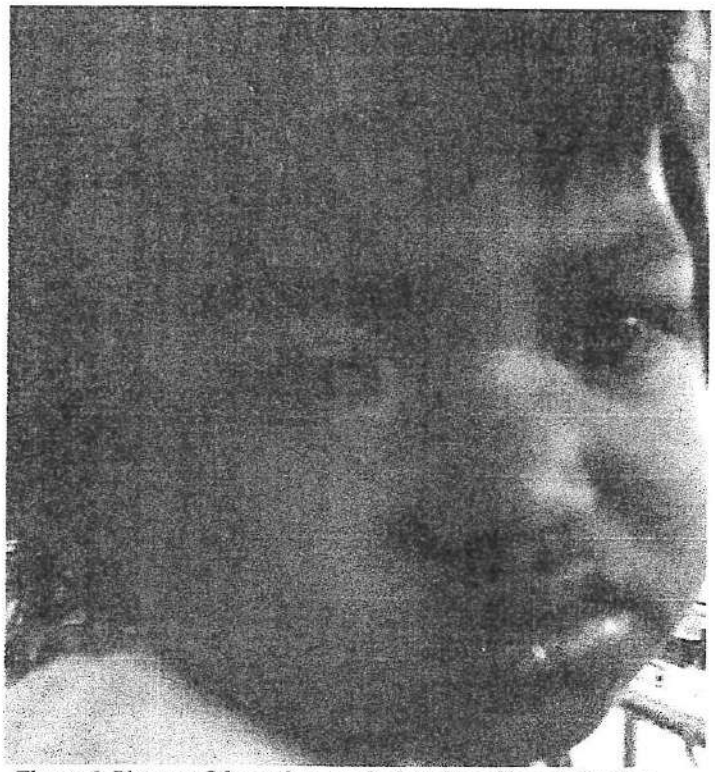

Figure 6. Picture of the patient on the last day of hospitalisation

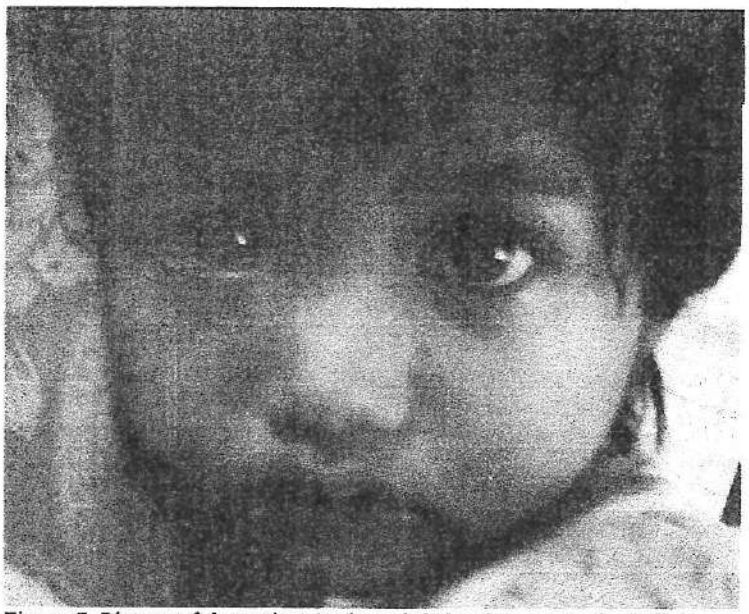

Figure 7. Picture of the patient in the ophthalmology outpatient clinic

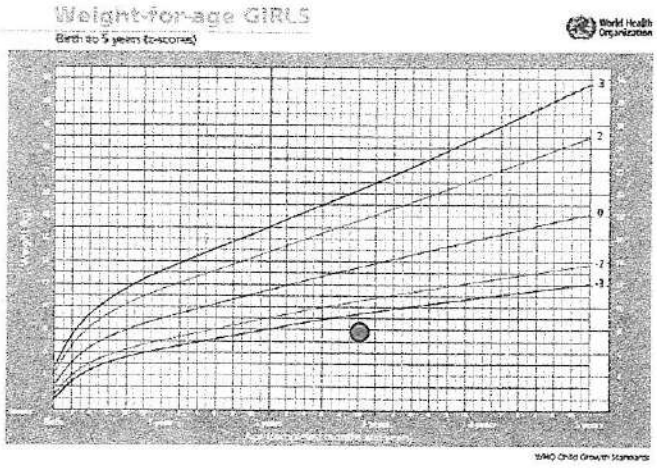

Figure 8. Growth chart showed body weight 8 kg, age 2 year 10 month. The percentile below -3.

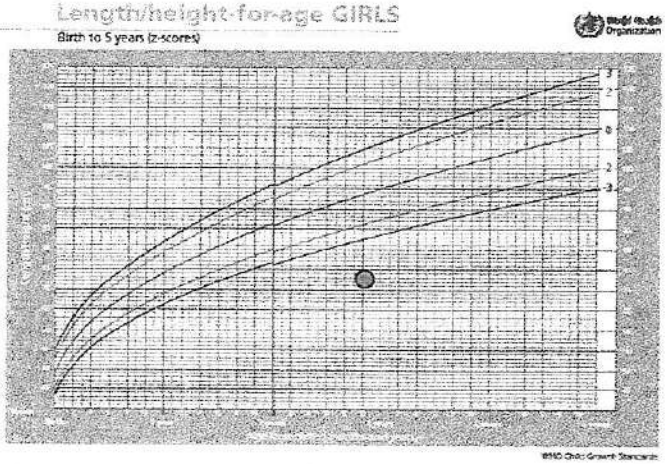

Figure 9. Growth chart showed body height 73 cm, age 2 year 10 month. The percentile below -3.

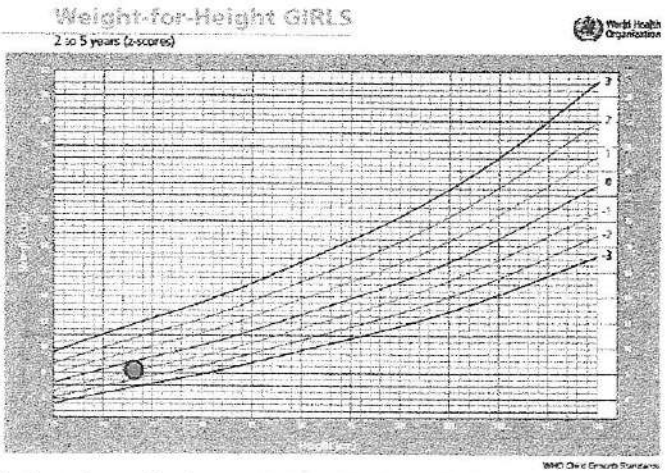

Figure 10. Growth chart showed body weight 8 kg, body height 73 cm. The percentile -1.

DISCUSSION

This case present a 2 year 8 month old female patient who came to emergency room because she got rash 15 days before admission. Rash came from her face and then spread at her whole body. The first rash appeared on the third day of fever. She also got cough, coryza and conjunctivitis. Physical examination revealed hyperpigmentation in the patient's neck and face.

Measles is a highly contagious acute viral illness characterized by a distinct exanthem, pathognomonic enanthem, accompanied by the classical triad of cough, coryza and conjunctivitis.[1,9] Measles virus infected 95%-98% of children by the age 18 years, and measles was considered an inevitable rite of passage. Exposure was often actively sought for children in early school years because of the greater severity of measles in adult.[3] Before the introduction of measles vaccines, one-third of children in many developing countries were infected in the first and second years of life, and most children were infected before age 5 years.[10] No difference has been noted between the sexes in either the incidence or the severity of measles.[11]

The rash and accompanying illness reach a climax on about the sixth day and in most case complete recovery.[9] The rash is usually first noted on the face and neck, appearing as discrete erythematous patches 3–8 mm in diameter. The lesions increase in number for 2 or 3 days, especially on the trunk and the face, where they frequently become confluent. Discrete lesions are usually seen on the distal extremities, and with careful observation, small numbers of lesions can be found on the palms of 25%–50% of those infected. The rash lasts for 3–7 days and then fades in the same manner as it appeared, sometimes ending with a fine desquamation that may go unnoticed in children who are bathed daily. An exaggerated desquamation is commonly seen in malnourished children. Fever usually persists for 2 or 3 days after the onset of the rash, and the cough may persist for as many as 10 days.[3]

In this case, conjunctivitis occurred in the fourth day of illness and never treated properly. She got white spot in her right eye since 4 days before asmission. Firstly the white spot felt in the both eyes, but the white spot in the left

eye was getting better. From the physical examination revealed pus in the right eye.

Conjunctivitis occurs in almost every measles cases, characterized by hyperemia on the eyes, edema of the lids and the caruncles. There is evidence of increased lacrimation and occasionally patient complains of photophobia. Measles virus and the antigen can be detected in the conjunctival lesion on the first days of illness. Conjunctivitis can be worsening so that occurred hypopyon and pan-ophthalmitis until blindness. It also can be corneal ulcer.[5]

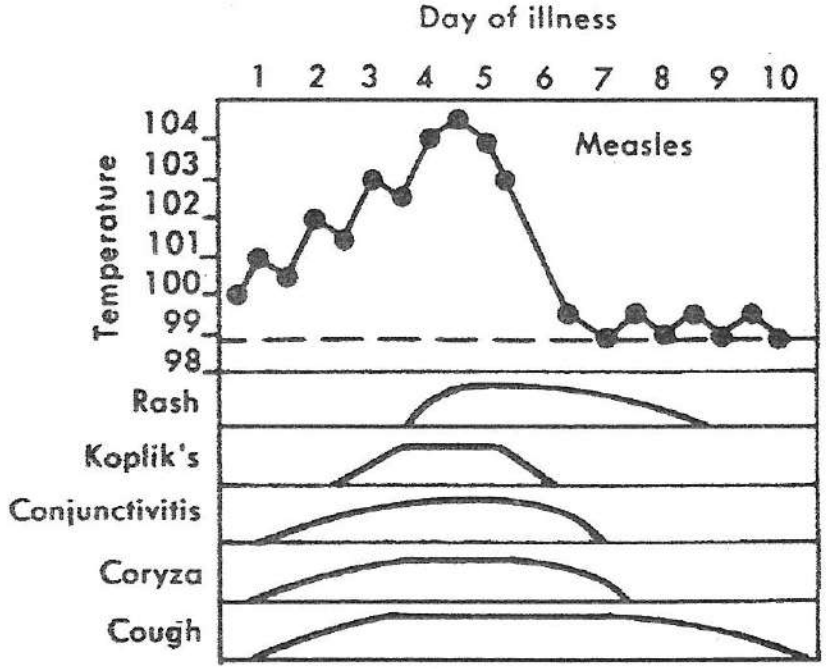

Figure 11. Schematic diagram of clinical course of typical case of measles.
Source : Krugman S, Ward R. Katz SL. Infectious diseases of children. Saint Louis: The C. V. Mosby Company; 1977. p. 132-47.

Infections of the cornea can lead to corneal opacity and blindness if not identified quickly and managed oppropriately. The terms infective keratitis, suppurative keratitis and microbial keratitis are all used to describe suppurative infections of the cornea. These are characterized by the presence of white or yellowish infiltrates in the corneal stroma, with or without an overlaying corneal epithelial defect and associated with signs of inflammation.[12]

Corneal scar is a significant cause of visual impairment and blindness in the developing world. Corneal infections are responsible for a large proportion of the scarring. A review of the data on indications for corneal transplantation in the developing world revealed that corneal scar was the most common indication (28.1%), of which keratitis accounted for 50.5%.[13] Hypopyon has been reported after corneal infections with diverse pathogens. Hypopyon is the accumulation of pus resulting from suppurative infection inferiorly in the anterior chamber. Hypopyon may be neither caused by infection nor lie at 6 o'clock in the anterior chamber.[14]

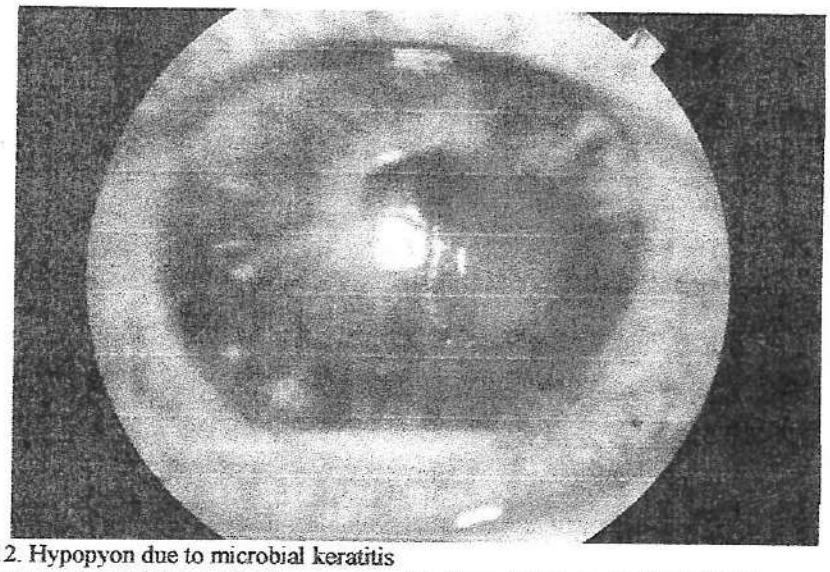

Figure 12. Hypopyon due to microbial keratitis
Source : Ramsay A, Lightman S. Hypopyon uveitis. Surv Ophthalmol. 2001;46:118.

In this case, the patient's past medical history, she was diagnosed severe malnutrition, but her parent seldom sent her to control to the public health center. The skin was normal before, and there wasn't bitot spot in the patient eye. The parent didn't observe whether the patient had the difficulty to see in the night or not. The patient was never breastfed, the parent often give her sugar water since she was 1 year old. Physical examination revealed body weight 7.2 kg and body height 71 cm, the nutrition status was wasted and severely stunted.

Acute malnutrition is the katabolic deterioration of the metabolism. As part of the total "disadaption" especially the protein metabolism is severely disturbed, the emphasis is on a decreases synthesis of serumproteins and a

suppression of the immune system. Malnutrition enhances infection, in malnourished children a prolonged excretion of measles virus from the nasal mucosa was found, also (bacterial) complications are more frequent.[6]

It also found that in cases of kwashiorkor the biochemical equilibrium between the different forms of collagen in the skin shifted towards the more soluble tropocollagen. Under normal circumstances no tropocollagen is to be found in the cornea. Measles and its sequelae provide collagenase, whereas malnutrition brings the collagen in a more soluble state which facilitates its disintegration.[6]

Deficiency of a vitamin may be primary (or exogenous) when the diet provides less than the requirement. Secondary (or endogenous) deficiency arises when the diet is adequate but one or more aspects of vitamin utilization by the body are defective (e.g. digestion, absorption, transport or cellular metabolism).[15]

Vitamin A, or retinol, is a fat-soluble substance found in liver (particularly fish liver) and in egg yolk and dairy products. Caretenoids –potential provitamin A precursor that can be converted to retinol in the wall of the gut- are present in green leafy vegetables, red palm oil, yellow fruits and the like. The availability of stored vitamin A also depends on a child's general status. Severely malnourished, protein-deficient children synthesis RBP at a much reduced rate. Serum retinol levels may therefore be subnormal, even if liver stores are high.[16]

Vitamin A deficiency is a systemic disease that affects cells and organ thoughtout the body, the resultant changes in epithelial architecture are termed "keratinizing metaplasis". Keratinizing metaplasia of te respiratory and urinary tracts and related changes in intestinal epithelia probably occur relatively early in the disease, even before the appearance of clinically detectable changes in the eyes. Among vitamin A deficient populations, therefore, children with measles, respiratory disease, diarrhea or significant protein-energy malnutrition should be suspected of being deficient ant treated accordingly.[16]

Uncomplicated, gradual depletion of vitamin A stores results in xerophthalmia of increasing severity, manifest as night blindness, conjunctival xerosis and Bitot's spot, corneal xerosis and corneal ulceration/keratomalacia. All these conditions usually respond rapidly to vitamin A therapy and the milder

manifestations generally clear up without significant sequelae. The loss of deep corneal tissue from ulceration/keratomalacia, however result in scarring and residual opacification. Sudden decompensation of marginal vitamin A status, as occurs in measles, can result in corneal ulceration that precedes the appearance of milder signs of xerophthalmia.[16]

In this case, the patient's immunization history was not complete. She was immunized except for BCG and polio.

Recommendations for the use of measles viru vaccine should be administeres when the child is about 12 months of age or shorthly thereafter in certain developing countries and in certain epidemic situations it may be wise to immunize between 6 and 12 monts of age.[9] Measles vaccination was associated with a 36% decline in overall death rate and a 57% reduction in the rate of death directly attributable or diarrhea, respiratory illness, or malnutrition in Bangladesh.[17] Virtually all unimmunized children will have been infected with measles by the age of 5 years. About half the cases occur in children below one year, the age group in which most death occur. [3]

In this case the patient was gave ampicillin injection 200 mg 6 times/day, nystatin drop 1 ml 6 times/day, Vitamin A 200.000 iu per oral for 2 days, Moxifloxacin HCL eye drop 1 drops every hour for the right eye and 1 drops every 2 hour for the left eye, Natamycin eye drop 1 drops every 2 hour for the right eye and 1 drops 4 times a day for the left eye, Homatropine eye drop 1 drops every 12 hours for the both eyes.

The patient should be admitted to the hospital to ensure adequate treatment and frequent follow up. For the topical treatment, natamycin 5% drops hourly or freshly reconstituted amphotericin-B 0.15% as drops hourly should be administered for anti fungal aiements. Antibiotics may have a limited role to play in such cases, Atropine 1% or homatropine 2 % could be used twice a day to dilate the pupil, this to prevent synechiae an relieve pain, anti glaucoma medication may be helpful and the vitamin A supplementation where the vitamin A deficiency is markedly prevalent.[12]

Severe infectious episodes, particularly measles but also malaria and chickenpox, can cause acute decompensation in vitamin A status. If vitain A

status is marginal to begin with, the resultant deficiency greatly increases the risk of blindness, systemic complication and death. All cases of measles in populations in which vitamin A deficiency is known occur, or where measles case fatality rates exceed 1 %, should receive the same initial treatment as is they had xerophtalmia a large dose of vitamin A (appropriate to age) on two successive days. These children are presumed to be deficient in vitamin A, regardless of their appearance. Children with severe, complicated, life threatening measles and all children with measles who are under 2 years of age should be considered for vitamin A therapy even if they do not come from a "high-risk" population.[16]

Prompt administration to massive amounts of vitamin A is essential (Table 1). Oral administration is preferred, because it is safe, cheap and highly effective 110 mg retinyl palmitate or 66 mg retinyl acetate (200.000 iu vitamin A) is administered bu mouth immediately upon diagnosis and the dose is repeated the following day. An additional dose is commonly given 1-4 weeks later in the hope of further boosing liver deficiency handle a massive dose poorly, it is essential that they are carefully monitored and given additional doses as needed, commonly every 4 weeks, until their protein status inpreves.[16]

Table 1. Treatment schedule for xerophthalmia

Timing	Dosage[a]
Immediately upon diagnosis[b]	110 mg retinyl palmitate or 66 mg retinyl acetate (200 000 IU) by mouth
Next day	110 mg retinyl palmitate or 66 mg retinyl acetate (200 000 IU) by mouth
Within 1–4 weeks, whenever clinical deterioration occurs, every 2–4 weeks in the presence of persistent kwashiorkor	110 mg retinyl palmitate or 66 mg retinyl acetate (200 000 IU) by mouth

[a] Children 6–11 months of age should receive only half the dose shown in this table and children less than 6 months one-quarter of the dose

[b] Intramuscular injection of 55 mg *water miscible* retinyl palmitate (100 000 IU) is substituted in rare instances when children with severe stomatitis cannot swallow, in cases of persistent vomiting, or if severe malabsorption (as in cystic fibrosis) prevents an adequate response

Source : Sommer A. Vitamin A deficiency and its consequences: a field guide to detection and control. 1995.

In the rare instances in which children are unable to swallow as occurs sometimes in stomatitis accompanying severe measles) in cases of persistent

vomiting, or in malabsoption of vitamin A, an intramuscular injection of 55 mg (100.000 iu) water-miscible retynyl palmitate should be substituted for the firs oral dose. Needles and syringes must be sterile. Oil-miscible preparations should never be given by injection because they are poorly absorbed from the injection site.[16]

For children aged 6-11 months, intramuscular and oral doses should be reduced by half, infant under 6 months of age should receive one-quarter of the normal dose. Where the vitamin A are not available, treatment should be instituted with foods rich in vitamin A.[16]

Children with xerophtalmia, particularly its blinding forms, are often severely ill, malnourished and dehydrated. Proper treatment will help save their vision as well as their lives and in cludes general supportive care, rehydration and frequent feeding (by nasogastric tube if necessary) with easily digestible energy and protein-rich foods. Concurrent illness, such as respiratory and gastrointestinal infections, tuberculosis, worm infestations and amoebiasis should be treated with appropriate agents (antibiotic, antihelmminthics, etc).[16]

In this case, the patient came to ophthalmology outpatient clinic with ophthalmology examination revealed no hyperemia and secretion in the both conjunctiva, no spasm in the both palpebra, both cornea was intact, no hypopyon in the anterior chamber eye. Iris, pupil and lens was normal in the both eye. There was corneal leukoma in both eyes with normal vision.

Superficial punctate lesions at the bulba conjunctiva and corneal side of the limbus can be seen to be synchronous with the body rash of measles. It commonly resolves without symptoms or sequalae in well-fed and vaccinated children. However, these lesions can progress into the central cornea, and exposure ulcerations at the 6 o'clock position may result with perforation or leukomas in children with protein energy malnutrition and vitamin A deficiency.[18]

SUMMARY

A rare case of hypopyon in a child with measles has been reported. The patient came with chief complain of white spot in the eyes. The pus was found in the right eye and the child was suffered from measles 2 weeks before admission. Her nutritional status was wasted and severely stunted and her past nutritional history was severely wasted. The diagnosis of post measles infection with bilateral corneal ulcer and right eye hypopyon with wasted and severely stunted was established based on anamnesis and physical examination.

The patient immunization was not complete, she just got BCG and polio immunization. She was never breastfed and she just got vitamin A 2 times until now. From the history taking we assumed that the patient already suffered from vitamin A deficiency before the patient got measles. Initial management of measles with eye complication should receive as they had xerophthalmia, a large dose of vitamin A on two successive days and the proper eye treatment. Children acutely ill with measles must be treated with adequate fluids, food and extra vitamins to compensate for their deteriorated nutritional status. Proper treatment will help their vision as well as their lives.

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Certificate of Attendance

This is to certify that

Dominicus Husada

Attended the sessions listed in the appendix below during the:

37th Annual Meeting of the
European Society for Paediatric Infectious Diseases

Held in:

Ljubljana, Slovenia | May 6 – 11, 2019.

Marko Pokorn **Goran Tešović**

Chairs, ESPID 2019 Meeting

Session Attendance

Title	Date	Time
INDUSTRY SYMPOSIUM 3	06/05/2019	14:45 - 16:15
INDUSTRY SYMPOSIUM 4	06/05/2019	16:45 - 18:15
INDUSTRY SYMPOSIUM 5	06/05/2019	18:30 - 20:00
INDUSTRY SYMPOSIUM 6	07/05/2019	08:00 - 09:15
INDUSTRY SYMPOSIUM 7	07/05/2019	09:30 - 11:00
INDUSTRY SYMPOSIUM 8	07/05/2019	11:30 - 13:00
INDUSTRY SYMPOSIUM 9	07/05/2019	13:45 - 15:15
PIDS/ESPID JOINT PLENARY SYMPOSIUM - THE FUTURE OF VACCINES (IS NOW)	07/05/2019	15:30 - 17:00
ESPID PLENARY 2 - OPENING SYMPOSIUM - ANTIBIOTIC USE ACROSS EUROPE – DIFFERENCES AND CHALLENGES	07/05/2019	17:30 - 19:30
ADVAC SESSION	07/05/2019	20:00 - 21:30
MEET THE EXPERT 5 - PREVENTION OF VERTICAL TRANSMISSION OF HIV	08/05/2019	07:00 - 07:50
PLENARY SYMPOSIUM 3 - ONE HEALTH – THE HUMAN – ANIMAL INTERFACE	08/05/2019	08:00 - 09:30
ORAL PRESENTATION SESSION 3 - NEONATAL INFECTIONS	08/05/2019	10:00 - 11:00
ESPID SYMPOSIUM 1 - PAEDIATRIC SEPSIS	08/05/2019	13:40 - 15:10
ESPID SYMPOSIUM 2 - VACCINE CHALLENGES	08/05/2019	13:40 - 15:10
ESPID SYMPOSIUM 3- PERINATAL INFECTIONS - THE MOTHER - INFANT PAIR	08/05/2019	13:40 - 15:10
ESPID SYMPOSIUM 8 - CONGENITAL CMV INFECTION	08/05/2019	15:40 - 17:10

ESPID 2019 Travel Award Notification

Yahoo Inbox

- **DiyanaYosifova** <dyosifova@kenes.com>

To:dominicushusada@yahoo.com

Mar 20, 03:39 PM

37th Annual Meeting of theEuropean Society for Paediatric Infectious Diseases

Ljubljana, Slovenia | May 6 – 11, 2019

Dear Dr. DominicusHusada,

We are pleased to inform you that your application was accepted to receive the **ESPID Annual Meeting Travel Award**. Accepted applicants receive benefits including support for economy class air and/or train travel to Ljubljana, Slovenia, accommodation for up to 5 nights at the Park Hotel, and free registration for the Meeting.

Please note: Applicants are required to register, book their accommodation, and contact the travel agency by March 12, 2019. Applicants who fail to do so will be removed from the award scheme entirely. It is essential that you follow the procedures set out below. Bookings done independently WILL NOT BE REIMBURSED.

REGISTRATION & HOTEL ACCOMMODATION

Please click here to register and book your accommodation.

TRAVEL SUPPORT

In order to receive support for your travel to the Meeting, you will need to make all travel arrangements to the Meeting via our officially appointed travel agent, Ophir Tours. **Travel bookings made on your own will not be reimbursed.** Please note that the conditions of the funding given to ESPID for the award scheme prevent us from reimbursing any expenses and payments you make yourself.

Please contact the official travel agent at: espid-grant@cwt.co.il with your required arrival and departure dates and the airport and/or railway station from which you will be travelling to the Meeting. Please send as well: names as in passport, gender, date of birth and mobile number. Please note that the offered travel options which meet your allocated travel amount may be direct or indirect flights and, for train travel, may be at off peak times.

- If you are travelling by air, please note that travel between your home and your local airport, and between the Ljubljana airport and the venue cannot be funded as part of the travel support. You will need to cover these costs yourself.
- Please note that once a flight/train ticket is booked, changes cannot be made to the booking.

ATTENDANCE DURING THE MEETING

ESPID requires recipients of the travel award to attend sessions throughout the entire Meeting. Attendance is logged by scanning the personal name badge on entry to each session hall. Any award recipients whose logged attendance falls below 80% of timetabled periods during the main Meeting (Tuesday pm to Friday am inclusive) will be permanently excluded from applying for ESPID travel awards in the future. Accordingly, you should not accept this award unless you intend to be present throughout the Meeting. **If you accept, it is critical that you log your presence at every session you attend.**

We look forward to seeing you in Ljubljana!

Best wishesESPID 2019 Meeting Organiser