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Case Report

Management of the acute noma: A case report

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ABSTRACT

Consultation in our countries of acute noma cases is rare due to delayed consultation due to the inaccessibility of health facilities and the poverty of patients living in remote areas. The early management of this case until the complete cure of its loss of jugal substance prompted us to report ca case.
The deaf and dumb 11-year-old patient exhibited pain and discomfort during feeding with losses through the jugal orostome. Dental mobility was associated with malnutrition and weight loss.
Faced with this table, the diagnosis of acute noma was made. The drug treatment consisted of antibiotic therapy based on amoxicillin associated with metronidazole. Treatment consisted of debridement of necrotized tissues using Dakin-soaked compresses and then iodine povidone followed by daily poses and adhesive tape. A gradual decrease in the diameter of substance loss had been observed between five and 30 days with healing at five months.
Early treatment of the acute phase prevents its evolution into the sequelae phase, which has repercussions that are difficult to take on with expensive means.

1. Introduction

Noma or Cancrum oris takes its name from the greek "nomein" which means "devour". It is a gangrenous stomatitis resulting in severe tissue destruction in the oro-facial sphere.[1] It most often occurs in early childhood (0-6 years) in areas where poverty and malnutrition are rife. It is sometimes associated with a general infection such as measles, malaria, or HIV.[2]

Mortality from this condition is estimated to be between 80 and 90%. Survivors with severely disabling after-effects include facial mutilation, speech and feeding difficulties, permanent constriction of the jaws. Disfigured, the patient is stigmatized and marginalized by society.[3] We report a case of acute noma treated by a team of oral surgery in the odontostomatology department of the Idrissa Pouye General Hospital in Dakar, Senegal.

2. Case History

This was an 11-year-old male child living with a disability (deaf-mute) in a very difficult socio-economic situation of parents who are poor peasants.

He had been consulted in the odontology department for a loss of substance that had punctured his left cheek, associated with pain and discomfort with the diet.

During questioning, a history of gingivorragia and dental mobility was reported. The patient was also a victim of poliomyelitis, which he had contracted in infancy; resulting in paralysis of the left leg.

The clinical examination revealed an alteration of the general condition, fever, dehydration and malnutrition. On exobuccal examination, a jugal perforation (orostome) about 3.5 cm in diameter on the main axis and 2.5 cm in the small

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axis had been observed, revealing the neighbouring dental crowns. [Figure 1]

The cookie cutter lesion had transfixed the cheek from side to side, leaving a yawning wound with clean contours, dotted with some necrotic tissues that released abundant saliva with a foul smell. The oral opening was limited and slightly deflected to the right with an amplitude of 2 cm. [Figure 2] The end-of-mouth examination revealed poor oral hygiene associated with generalized tartar gingivitis, dyschromia of 36, degree 3 mobility of the 26 in relation to perforation and persistence of the 63. Cavities were present on the proximal faces of teeth 84 and 85. The lining of the inner cheek of the surrounding cheek of the wound was normal in appearance and firm in consistency. Orthopantogram had reported the absence of tooth germs 34 and 35 with underlying osteolysis associated with root rhizalysis of teeth 73 and 75. [Figure 3] At the end of the clinical examination, the diagnosis of noma in the acute phase had been made.

The management was multidisciplinary with the collaboration of odontologists and paediatricians. The patient was hospitalized in pediatrics for nutritional rebalancing and necessary medical care. The drug treatment consisted of oral antibiotic therapy, based on amoxicillin 1g per 24 hours associated with metronidazole 500 mg per 24 hours. Local care began from the first consultation in the odontology department, by debridement of necrotized tissues using Dakin-soaked compresses and then yellow iodine polyvydone (betadine), followed by the installation of a vaseline compress against the yawning wound and finally the installation of sterile compresses before the application of an adhesive band. [Figure 4] They were renewed every 48 hours.

A gradual decrease in the diameter of substance loss was observed at 10 days, 16 days and then a closure of the wound 30 days later, but resulting in a retractable scar. [Figure 5]

In addition to local care, in order to prevent permanent constriction of the jaws, mechanotherapy had been introduced by asking the patient to perform daily mandible mobilization exercises using "clothespins". It has been associated with an acrylic resin "calage" gutter placed intermittently between the dental arches of the controllable molars to avoid a possible oral closure. A recovery in the amplitude of the oral opening was noted and a complete healing of the jugal wound five months later.[Figure 6] A condition of the oral cavity was subsequently carried out by scaling-surfacing, dental avulsions and restoration of the decayed teeth.

3. Discussion

Since 2003, WHO has implemented national noma control plans involving dental surgeons in six West African countries, including Senegal.[1,4] It is in this momentum that this management of a case of noma in acute in Dakar

Fig. 1: Loss of transfixant substance to the left cheek

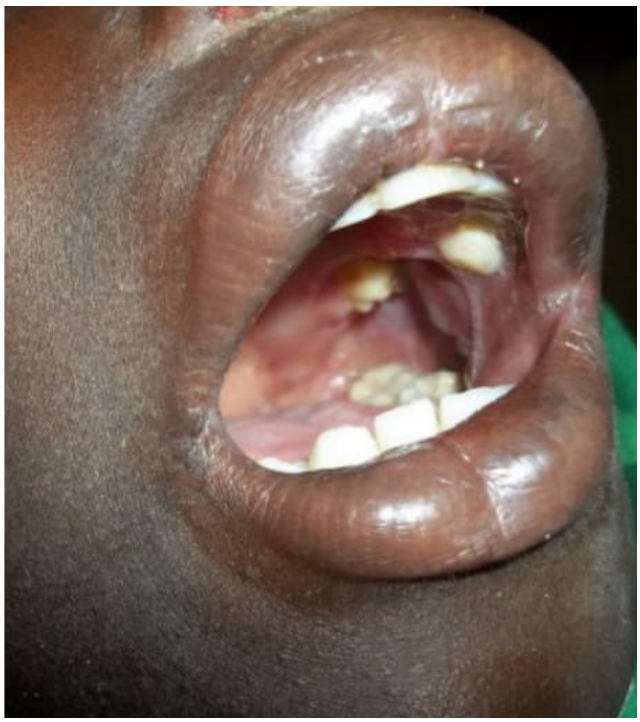

Fig. 2: Moderate trismus associated with jugal wound

Fig. 3: Orthopaontogram showing bone scarcity with tooth agenesis 34 and 35 and rhizalysis of teeth 73 and 75

Fig. 4: Local care, A-parage B-pose vaseline compress C-adhesive dressing

Fig. 5: Closure of the wound at 7 days (**A**), 15 days (**B**) and 30 days (**C**)

(Senegal). Noma is an endemic disease in many countries in Africa, South America, and Asia. [1]

In the literature, it has been reported that noma most often affects children in more than 95% of cases. [1] In Africa, Burkina Faso, an incidence of 5.5 cases per year was reported with an average age of 7.64±2 years. [5] According to Ibikunle in Nigeria, 87% of patients with noma were under the age of six. [6] The patient's age of clinical observation reported in this study was 11 years.

However, adults may be affected by this pathology in immunosuppressed field cases. [5,7]

Poverty and malnutrition that cause decreased immune response are factors that promote noma often reported by the authors. [2,3] This was the case in this clinical observation in which the patient came from a very low-income peasant family. The use of traditional medicine and the lack of

Fig. 6: Jugal scar and normal mouth opening, 5 months later

resources of parents are at the root of the consultation delays often observed in our developing countries.[3]

On the other hand, in developed countries where the socio-economic conditions and educational attainment of the population are optimal, noma has been eradicated.[7]

Certain infectious diseases, such as viral (measles, chickenpox), parasitic (malaria, bilharziosis) and bacterial (typhoid, tuberculosis, bronchopneumopathies) infections in poverty areas are predisposition factors to noma because they can weaken the individual's immune defences.[1,7] Ibikunle found a high prevalence of measles in young patients with acute noma (47.2%) protein-energy malnutrition (42.1%).[6]

Also, an alteration in the general condition is often observed in patients with noma as in the case reported in this study.[8] Indeed, our young patient, deaf mute, had paralysis of the lower limbs following poliomyelitis contracted in his infancy. Also his generally impaired condition associated with fever, dehydration and malnutrition.

The progressive lesions of noma can be localized at the labial, jugal, labio-commissural and/or nasal level, often resulting in feeding difficulties, which aggravate malnutrition.[1] Authors such as Enwonwu and Baratti-Mayer have reported that poor oral hygiene associated with malnutrition promotes the development of acute necrotizing gingivitis, which would be the starting point of Noma.[7,8] Acute necrotizing gingivitis develops into gangrenous stomatitis and tissue necrosis which will result in a loss of soft tissue substances exposing the jaw bone and teeth in relation to the affected area, as observed in this study.[4,8] Xu noted a weight loss secondary to a difficulty in eating.[9] The pathognomonic signs of this acute necrotizing gingivitis are spontaneous gingivorragia, decapitation of gingival papillae, pain and foul breath.[4]In our clinical observation, these symptoms were reported by parents who had seen them at the beginning of the child's illness.

The management of the acute-phase noma consists of combining: hydro-electrolytic and nutritional rebalancing, antibiotic therapy based on amoxicillin and metronidazole, and regular local care.[9,10] It has been established that this early-adopted therapeutic attitude halts the progression of the disease to tissue necrosis.[10] Nutritional rebalancing will be ensured by a balanced hyper-vitamin diet and adequate hydration.[10]

Local food products can be used to treat protein-energy malnutrition during outpatient noma.[2]

Antibiotic prescribing in this observation, based on amoxicillin and metronidazole, allowed for a regression of symptoms. According to Costini, this combination of amoxicillin and metronidazole has resulted in a cure rate of more than 75% of noma cases.[10] In addition, authors advocate the use of other molecules such as aminosides, spiramycin or vancomycin, used alone or in association with metronidazole.[10]

These antibiotics are active on anaerobic bacteria, including prevotella, spirochetes and peptostreptococcus.[2] Feller advocates a combination of penicillin/clavulanic acid, aminoside/metronidazole to stop disease progression and reduce mortality.[11] On the other hand, Ibikunle introduced venous injectable antibiotic therapy based on metronidazole and ceftriaxone (3rd generation cephalosporin).[6]

Local care in this observation consisted of slodroining the wound with oxygenated water to remove necrotized tissue, followed by cleaning and disinfection with sodium hypochlorite permanganate, followed by the installation of sterile compresses and adhesive tape. Local treatment of noma should always include the removal of bedsores and necrotized tissues.[5,11]

Other authors recommend multi-daily irrigations and sprays with local antiseptics, after debridement of necrotized tissue, bone or dental receivers using precelle or curette.[5,10] The condition of the oral cavity carried out in this observation through scaling-surfacing, avulsion of the

decayed teeth and conservative care of the decayed teeth is also advocated by authors such as Costini.[10] Debridement was performed in 159 Nigerian children using compresses with 3% hydrogen peroxide followed by abundant irrigation with saline solution to cause bleeding.[6] After hemostasis a gauze compress soaked in iodine povidone was applied to the loss of skin substance.[6] A 0.9% sodium chloride solution was continuously applied by Xu for 24 hours in a 6-year-old child.[9]

In the reported clinical case, during the healing phase of the jugal wound, a retractable scar was observed resulting in a gradual decrease in the amplitude of the oral opening. Thus, preventive mechanotherapy of permanent constriction of the jaws has been introduced. It was done using a clothespin, used by the patient who practices to make movements of closing and opening the maxillas by manipulating the clamp whose end is inserted between the teeth.

Mechanotherapy, combined with the installation of a resin calibration gutter, restored the normal amplitude of the patients' oral opening.

Authors recommend intra-lesion injections of delayed corticosteroid to prevent retractable scarring to limit the formation of retractable fibrosis that prevents oral opening.[10]

4. Conclusion

Noma affects young children living in poor general conditions with poor oral hygiene. Its management in the acute phase consists of hydro-electrolytic and nutritional rebalancing, antibiotic therapy based on amoxicillin and metronidazole and regular local care. Early treatment of the acute phase prevents its evolution into the sequelae phase, which has repercussions that are difficult to take on with expensive means. The patient and his entourage were satisfied with the treatment because from an aesthetic point of view, the patient was more and more accepted by his friends and his diet was done without loss of jugular fluid linked to the closure of the orostome.

5. Source of Funding

None.

6. Conflict of Interest

The authors declare that there is no conflict of interest.

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