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SCIENTIFIC LETTERS

Poisonous snakebites: A five-year experience☆

Mordedura de serpientes venenosas: experiencia durante 5 años

Dear Editor:

Snakebite is an unusual reason for visits to Emergency Departments. This may lead to uncertainty about its treatment, and especially on the use of a very specific, expensive antidote with limited distribution and potential adverse effects.[1,2] Given the potential gravity of the situation, we need to know the most appropriate therapeutic approach.[3,4]

We present a study of a series of clinical cases carried out in the Emergency Department of a third-level Maternity and Children's Hospital, describing the cases of snakebite reported between January 2011 and December 2013, with the aim of detecting the points that could be improved when dealing with them, in the light of their outcomes and the new recommendations.[2]

A total of five patients came for consultation, one in 2011 and the rest in 2013. All of them had previously visited another health centre and only one came to our hospital in the first 12 h after being bitten. At the time of their arrival all the patients presented grade 2 envenomation. Table 1 shows the classification of symptoms after snakebite.[4] Table 2 lists the clinical features of the patients included in the study. The five patients were hospitalised. Blood tests were performed on all of them to rule out secondary complications, with normal results. Initially, conservative treatment was pursued. However, the four cases recorded in 2013 progressed poorly, deteriorating to grade 3, at which point the antiophidic serum (Viperfav®) was administered. Two patients required fasciotomy to be performed for compartment syndrome. The administration of the antiophidic serum did not result in adverse reactions, and the treatment brought favourable outcome. Only the case in 2011 had a favourable outcome with the initial medical treatment, without requiring antivenom.

The snakes that most commonly produce envenomation in the Iberian Peninsula belong to the Viperinae family (vipers).[2,4,5] However, management of bites will depend on the degree of envenomation the patient shows, regardless of its aetiology.[5,6]

Firstly, an initial assessment will be made, using the ABCDE algorithm.[6] As for specific treatment of the bite, the wound will always be cleaned and treated with antiseptic, the analgesia will be administered and the tetanus vaccination record will be checked.[2,5,6] Systematic use of antibiotics is controversial and it is currently recommended to be administered only if superinfection is suspected.[1,2]

Table 1 Degree of envenomation by snakebite.

Degree of envenomation	Clinical manifestations
Grade 0 (dry bite)	Fang marks Mild or non-existent pain No local or systemic symptoms
Grade 1 (mild envenomation)	Moderate/intense pain Local inflammatory oedema Absence of general symptoms
Grade 2 (moderate envenomation)	Widespread or rapidly progressive oedema (to the ends of the extremity) Ecchymosis, painful local adenopathies, lymphangitis, blisters, necrosis Moderate general symptoms Neurological symptoms: palpebral ptosis, accommodation deficit, ophthalmoplegia, diplopia, dysarthria, dysphagia, paralysis of the orbicularis oris, lethargy, vertigo or pareses
Grade 3 (severe envenomation)	Intense local reaction extending beyond the extremity Severe general symptoms Severe neurological symptoms

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Table 2 Clinical presentation and management of venomous snakebites treated in the Paediatric Emergency Department.

	Sex, age and date	Anatomical site	Time since bite	Clinical presentation	Initial management	Initial progress	Fasciotomy	Antiophidic serum	Final outcome
Case 1	♂, 10 years, April 2011	1st finger LH	24 h	Grade 2 (purplish oedema ULE)	Blood analysis, antibiotic, corticosteroid, antihistamine, analgesia	Good	No	No	Good
Case 2	♂, 5 years, April 2013	3rd finger RH	14 h	Grade 2 (oedema and ecchymosis URE)	Blood analysis, antibiotic, analgesia, tetanus prophylaxis	Compartment syndrome (grade 3)	At 24 h	At 36 h	Good
Case 3	♂, 8 years, April 2013	4th finger LH	13 h	Grade 2 (purplish oedema ULE, lymphangitis, self-limited vomiting)	Blood analysis, antibiotic, analgesia, tetanus prophylaxis	Oedema spread to thorax (grade 3)	No	At 24 h	Good
Case 4	♂, 2 years, April 2013	Interdigital space 1st–2nd fingers RH	48 h	Grade 2 (purplish oedema URE, lymphangitis)	Blood analysis, antibiotic, corticosteroid, antihistamine, analgesia	Oedema spread to thorax (grade 3)	No	At 48 h	Good
Case 5	♀, 10 years, July 2013	2nd finger RH	4 h	Grade 2 (oedema and ecchymosis finger, haematoma proximal 1/3 arm)	Blood analysis, antibiotic, corticosteroid	Compartment syndrome (grade 3)	At 24 h	At 48 h	Good

LH, left hand; RH, right hand; ULE, upper left extremity; URE, upper right extremity; ♂, male; ♀, female.

Similarly, the use of antihistamines and corticosteroids is not indicated, as their efficacy has not been demonstrated.[1,2] The use of antiophidic serum has a particularly important place in therapeutic management. The most commonly used serum in Spain is Viperfav®. This contains heterologous proteins obtained by immunising horses.[1,2,4] The classic recommendations were to reserve the use of antivenom for cases of grade 3 envenomation, due to their potential risk of anaphylaxis. However, the thorough purification process by which the serum currently available (Viperfav®) is obtained makes it a safe antidote with low allergenicity.[1,2,4] For this reason, the latest treatment recommendations for venomous snakebites set forth by the Spanish Panel of Experts in December 2012 indicate early use of serum in cases of grade 2 envenomation.[2,4,5] Moreover, it is considered the treatment of choice for compartment syndrome, leaving fasciotomy for cases that do not respond after administration of the antidote.[2,4,5] Since the new recommendations appeared at the same time as the cases presented here, the hospital's current protocol, which did not include them, was followed in these cases. The review of the cases led to the updating of the protocol.

As regards to additional tests, a blood analysis needs to be performed to rule out associated complications such as thrombocytopaenia, fluid and electrolytic imbalances or renal insufficiency.[4–6]

Finally, all patients require hospital observation to monitor their progress (grade 0: 6 h; grade 1: minimum 24 h; grades 2 and 3: admission to hospital or Intensive Care Unit depending on severity).[2,4,5]

In conclusion, we want to emphasise that in 2013 we witnessed a peak of incidence of venomous snakebites in our department, as well as greater severity and poorer progress. Moreover, we have observed a tendency for patients treated conservatively to deteriorate, and therefore, in line with the new recommendations, we consider that earlier use of antiophidic serum could avoid subsequent poor progress. In addition, we have found that side effects of the antiophidic serum in the form of allergic reactions are infrequent, and that other drugs used in our patients (corticosteroids and antihistamines) did not prevent the progression of local inflammation.

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Early infantile dysphonia as an alert symptom of juvenile laryngeal papillomatosis☆

Disfonía infantil precoz como síntoma de alerta de la papilomatosis laríngea juvenil

Dear Editor:

Juvenile laryngeal papillomatosis (JLP) is the second most common cause of dysphonia in children, after true vocal cord nodules. The most frequent causative agent is human papillomavirus (HPV) serotypes 6 and 11. The most widely accepted mode of transmission is vertical, through the birth canal of a mother with an active or latent genital infection.[1] The prevalence is 1.11–2.59 per 100,000 children.[2] We present 6 cases of JLP, a recurrent disease with non-specific clinical symptoms, which should be taken into account in cases of persistent dysphonia.

We conducted a retrospective study of diagnosed cases of JLP during the period from 1 January 2000 to 31 December 2013. We analysed epidemiological data (age at diagnosis, sex), treatment received, sequelae and number of surgical interventions (Table 1). The male–female ratio found was 1:1. The median age at diagnosis was 18 months, with an interquartile range (IQR) of 26.5 months. The common symptoms were dysphonia, nocturnal breathing difficulty and inspiratory stridor. Patient 1 was an uncontrolled pregnancy with maternal serology compatible with a resolved syphilis and hepatitis B infection, premature at 25 weeks gestation, with a birth weight of 1000 g. She had associated malnutrition and laryngomalacia. She needed tracheostomy from 14 to 20 months of age (because of respiratory failure secondary to papillomatosis). Patient 2 was the product of an adolescent pregnancy of a mother vaccinated against HPV (Cervarix® 3 doses) at the age of 14. He was diagnosed with recurrent laryngitis. Patient 3 had been an uncontrolled pregnancy. Patient 4 had a maternal history of papillomas on the hands, and was diagnosed with adenoidal hypertrophy and bronchitis. The mother of patient 5 had had previous genital papillomatosis and patient 6 had language acquisition difficulties.

The laryngeal papillomas were detected by fibre-optic laryngoscopy, and an excision was performed (Fig. 1) by endolaryngeal microsurgery. In 4 of the 6 cases they received intraoperative adjuvant treatment with intralesional cidofovir on at least one occasion. Case 2 also received oral propranolol daily for a year. The histological findings were consistent with papillomatosis, and serotype HPV-11 was identified in cases 2 and 3 and HPV-6 in case 6.

The vocal cords are the most frequent site of JLP. Distal spread has been associated with poorer prognosis and with HPV-11 infection.[3] The classic presentation is persistent dysphonia with weak crying between 2 and 4 years of age. The second most common symptom is stridor, first inspiratory and then biphasic. Less common presenting symptoms include chronic cough, recurrent pneumonia, failure to thrive, dyspnoea, dysphagia and acute respiratory distress, especially in infants with an upper respiratory tract infection.[4]

The differential diagnosis with this disease must be made with other causes of airway obstruction, primarily at the pharyngo-laryngeal level (laryngomalacia, vocal cord paralysis, glottic or subglottic granulomas or cysts, laryngitis, congenital or acquired subglottic stenosis, haemangiomas and traumas) and at the tracheal level (tracheal stenosis, tracheomalacia, extrinsic compression by abnormal vascular structures). Less commonly it will be confused with abnormalities at the nasal level (choanal atresia/stenosis, pyriform aperture stenosis, adenoid hypertrophy, tumours, rhinitis and foreign bodies), as well as at the pharyngeal level (nasopharyngeal stenosis, tonsillar hypertrophy, macroglossia and craniofacial abnormalities).

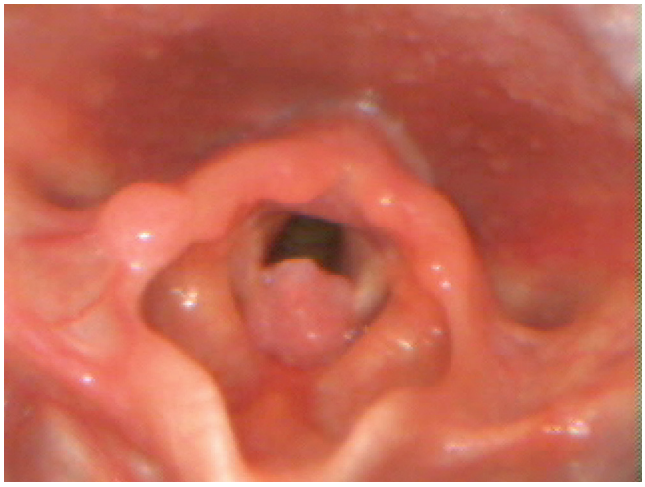

Figure 1 Fibre-optic laryngoscopy: lesion of papillomatous appearance, occupying the anterior third of the vocal cords and partially obstructing the glottic lumen.

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