Brief Communication

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Gynaecology

UVULA OEDEMA AFTER APPLICATION OF NITROUS OXIDE DURING LABOUR

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KEYWORDS :

Letter to the Editor

We would like to share our experience of uvula oedema during labour after application of inhaled nitrous oxide.

A 31 year old gravida 2 para women with neat family and obstetric history. During second pregnancy, gestational diabetes mellitus was diagnosed in 24nd gestational week, regulated by diet.

Patient was hospitalised at 39th gestational week due to planned labour. Labour induction started at 1, 5 cm delated cervices by PgE2 vaginal gel application. Six hours later amnion rupture happens spontaneously and when cervices was dilated 5 cm analgesia started with inhaled nitrous oxide and infusion of oxytocin (5 i.u. /mL).

Two hours later symptoms occurred as difficulty in speaking and swallowing, choking feeling and urge to vomiting. A doubled enlarged, oedematous, reddish uvula was observed without tongue oedema and enlarged tonsils.

That time cervices was completely opened and contraction frequency was 2/10 min with normal CTG values.

Treatment of uvula oedema started with oxygen application and corticosteroid therapy. Blood pressure (RR 130/80 mmHg), pulse 100 bpm and oxygen saturation (98%) were monitored. Anaesthesiologist was also consulted. Examination of the respiratory system was unremarkable.

Fifteen minutes after the appearance of the first symptoms of uvula oedema started labour which finished successfully and male infant was born (4.170 g, 52 cm) with Apgar score 9/10 and umbilical arterial pH 7,31.

Response to the therapy started half hour later. After six hours symptoms of difficulties in speaking, swallowing and feeling of chocking were minimised but uvula oedema was still present but decreased by 1/3. Patient was monitored in ICU 24 h after delivery and uvula oedema has withdrown within 24 hours. Patient left the hospital three days after delivery.

Complete blood count, coagulation, routine biochemical, liver function tests and CRP were done half hour after delivery. Deviations from normal values shown CRP=53,3 mg/L and L=30,6x10^{9}/L and which were normalised within 24 hours.

Uvula oedema sympthoms were treated with methil-prednisolon 125 mg i.v., chloropiraminchlorid 20 mg, adrenalin 1:1000, calcium-gluconat 10 mL.

Acute uvula oedema is rare occurence and could be potentially life-threatening condition involving other upper airway structures such as apiglotis, causing rapid partial or complete airway obstruction and difficulties in speaking and breathing as it affects the vocal cord. Early and accurate diagnose and treatment is exceptionally important.

Etiology of isolated angio-oedema is usually due to type 1 hypersenitivity reaction and angio-oedema of uvula is not assotiated with other hypersensitivity reaction such as pruritis, skin rash, hypotension or tachicardia. It is localised, recurrent, non-pruritic subcutaneous swelling that usually appears rapidly and resolves spontaneously.

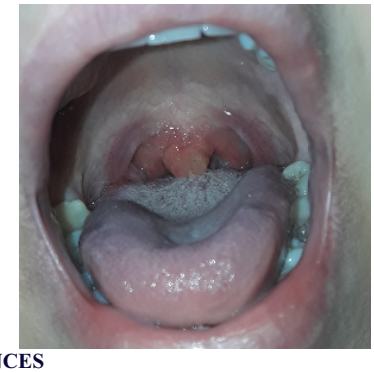
The possible causes of reccurent uvula oedema are: idiopatic, heraditary, trauma, infections, allergy, medications and obstructive sleep apnea.

In our case, there is possibility of isolated uvula oedema occurrence due to nitrous oxide use in labour analgesia.

There is a possibility of oxidation of nitrous oxide to nitric oxide by hydrochloric acid due to urge to vomiting and aspiration of nitous oxide. Nitric acid is a potent vasodilator and might cause uvula angio-oedema.

Oedema is thought to result of inflammatory induced increases in airway microvascular permebility. Inflammatory mediators released in the airway mucosa include histamin, leucotriens and PAF are potent inducers of increased oedema and airway mucosal swelling.

It is unclear wheather our patient's uvula oedema was resualt of an antigen response to nitrous oxide or is related to trauma caused by dehydration due to exposure to dry gas and improper breathing technique (too deep or open mouth breathing).



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