CENTRAL NERVOUS SYSTEM LIGNOCAINE TOXICITY IN AN INFANT FOLLOWING VENTRICULO-PERITONEAL SHUNT AND SPINA BIFIDA REPAIR: A CASE REPORT

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SUMMARY

A 3.7kg one-month-old male infant with Spina Bifida Cystica with Hydrocephalus had Ventriculo-Peritoneal (VP) Shunting and repair of the Spina Bifida Cystica done. Lignocaine with Adrenaline 1:100,000 was infiltrated intra-operatively to reduce blood loss. Overenthusiastic infiltration, an oversight, led to Lignocaine toxicity. In Pediatrics, prior calculation of maximum dosage, based on child’s weight, and use of dilute solutions for multiple sites could have prevented this.

INTRODUCTION

A commonly used method of reducing bleeding during surgery involves local infiltration of the skin and sub-cutaneous tissues with a solution containing a vaso-constrictor drug, usually adrenaline (1). Diathermy is not readily available in our region, and so this is a frequently used method to reduce blood loss during surgery. A solution of lignocaine, saline and Adrenaline, locally termed as “jungle juice” is commonly used. The solution when infiltrated also assists in separating tissue planes for ease of dissection and augments anaesthesia. Overdose to the patient can occur if this solution is not meticulously constituted and maximum dosages not exceeded based on child’s weight and general health condition.

CASE REPORT

We report a case of lignocaine toxicity from over enthusiastic infiltration of the solution with adrenaline 1:100,000 in an infant. A one-month-old male infant weighing 3.7 Kg with a diagnosis of Spina Bifida Cystica with Hydrocephalus was scheduled for Ventriculo-Peritoneal Shunting plus repair of the Spina Bifida Cystica. Surgical diathermy was not available. The surgeon opted to use lignocaine with adrenaline 1:100,000 to reduce bleeding. The nurse prepared solution of 10 c.c 2% lignocaine plus 0.5 c.c adrenaline (1:1000) plus 39.5 c.c normal Saline for infiltration. The final solution of “jungle juice” was 0.4% lignocaine with adrenaline 1:100,000. General anaesthesia with oxygen/nitrous oxide/halothane was given. The surgeon infiltrated 5 c.c of solution at scalp site and 5 c.c at the abdominal wall site. Following insertion of Ventriculo-Peritoneal Shunt, the infant was repositioned to prone position. 15 c.c of solution was infiltrated around Spina Bifida Cystica site. At this juncture, anaesthesiologist noticed that total of 25 c.c of the solution had been injected. He cautioned the surgeon that calculated maximum dose of this solution was 6.475 c.c (25.9 mg of lignocaine with adrenaline, 7 mg/kg) and had been exceeded. Sinus tachycardia, up to 200 beats/minute that settled with time, was noted. Duration of operation was 2.5 hours. Surgery and anaesthesia reversal were uneventful. After 30 minutes at Post-anesthesia care unit (PACU), the infant was noted to develop myoclonus jerks of the upper limbs. Oxygen by mask and intravenous diazepam in appropriate incremental doses was administered. The infant admitted to I.C.U for close monitoring. Myoclonus was on and off and resolved after four hours. Infant recovery was uneventful and discharged in good condition.

DISCUSSION

Local infiltration of a vaso-constrictor is a frequently used blood conservation strategy in surgery (2,3). Lignocaine with Adrenaline 1:100,000 or more commonly 1:200,000 is used (4). The maximum dose limit for lignocaine with adrenaline for infiltration is 7mg/kg (5). That of adrenaline, when halothane
anaesthesia is used, is 10mcg/kg (6). If multiple sites of operation or large surfaces are involved, more dilute solutions may be used so as not to exceed the maximum dosage limits of both lignocaine and adrenaline. Exceeding the maximum dosage of lignocaine may lead to systemic toxicity. Intoxication with lignocaine is known to occur because of its popularity and wide safety margin attributed to it. As a result, guided use is either overlooked, ignored or the health workers have no knowledge of the guidelines and so overdose becomes the norm (7). Lignocaine CNS toxicity arises from inhibition of excitatory pathways in the CNS, producing a stereotypical sequence of signs and symptoms as the drug concentration in blood gradually increases.

In unmedicated patients initial symptoms of neurotoxicity include vertigo, tinnitus, ominous feelings, circumoral numbness, carrollousness, tremors, myoclonic jerks, convulsions, coma and cardiovascular collapse. These symptoms may not be detected in infants and children under general anesthesia. Diagnosis of local anesthetic toxicity under general anesthesia can be made with indirect signs such as muscular rigidity, hypoxemia without other causes, unexplained tachycardia, dysrhythmias or cardiovascular collapse (8). Exceeding maximum dose of adrenaline poses a risk of cardiac arrhythmias, especially with concurrent use of halothane anesthesia.

In our case, multiple sites in an infant 3.7 kg were infiltrated. This led to 4 times the toxic dose of lignocaine and 6.75 times that of adrenaline being given. Infant developed typical CNS lignocaine toxicity signs, myoclonus jerks of the upper limbs and a sinus tachycardia from adrenaline overdose. This was attributed to oversight that this was an infant, multiple infiltration sites and a solution that was not dilute being used.

Lignocaine toxicity was adequately treated with oxygen, incremental doses of diazepam and close monitoring. Halothane was switched off during Sinus tachycardia due to adrenaline. Areas where local anaesthetics are used must be prepared to diagnose and manage severe local anaesthetic toxicity (8). A multicenter case survey should be conducted locally to determine the extend, patterns and safe use of “jungle juice” among surgical teams operating on children.

**REFERENCES**