Lessons to be learned

Sir,

I read the article on fatal airway obstruction following arterial trauma during internal jugular venous cannulation by Williams et al. with great interest. While arterial puncture is a known risk in central venous cannulations, I would like to bring to attention several points that were clearly to be highlighted. First of all, as a standard guideline, any elective central venous cannulation should be done under ultrasonic guidance. This is not technically difficult but has tremendously reduced the incidence of inadvertent arterial injury. Use of ultrasound also would point one toward arterial injury in case of doubt, as in this index patient. Color of the blood is not the gold standard to assess the nature of the vessel. Quite often, patients who require central venous cannulations are already unstable and their arterial blood can equally be dark as well to naked eye. When in doubt, one should always check saturations in blood aspirated from the vessel before attempting to dilate to great nature. As is always, if in doubt, dilatation should be barred, as a dilated neck artery would only need a surgical repair and is rare to get control in an elderly patient to stop on its own. Moreover, assessment of line pressure from the introducer would possibly help in doubtful situations. A simple bedside manometry in intensive care unit setup is not very difficult and can be life saving. And most important of all is the need for surgical cricothyroidotomy. In an emergent situation like this, with no control over airway, I am surprised to see that this is not done as a way of securing airway control and ventilation, rather than attempting to do a tracheostomy with its difficulties in a displaced airway (secondary to an expanding hematoma). Cricothyroidotomy is done at the level where a thyroid cartilage of airway (even when displaced) is felt and a surgical incision opens the airway for a tube to be passed and subsequently to be ventilated. It is always life saving, and can be converted to a proper airway once the situation is brought under control. As reinforced repeatedly, expanding hematoma from an arterial injury in the neck deep to deep fascia needs immediate and quick attention to both airways and the artery itself, as this can further compromise cerebral circulation.

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Reference

Acute multifocal dystonic reaction: An unusual presentation of vitamin D-deficient rickets

Sir,

A 7-year-old boy born of non-consanguineous marriage presented with inability to open his mouth. He had episodic painful spasm of the jaw muscles since last 7 h with each attack lasting between 2 and 5 min. During each attack he was unable to open his mouth due to spasm of bilateral masticatory muscles. This was followed by complete recovery in between the attacks. His sensorium was normal during the attacks. The painful spasm of the jaw muscles subsided spontaneously within 5 min after admission. There was a history of intermittent spasm of the right hand since 1 day. There was no history of spasm in other muscles of the body. There was no history of fever, convulsion, dyspnea, paresthesia, altered sensorium, involuntary movements, drug intake, or trauma. He was completely immunized as per his age. His developmental and birth history was normal. He was breastfed till 19 months and weaning started at 10 months of age. He was a pure vegetarian and a detailed dietary history revealed deficient intakes of vitamins D. There was no history of any treatment received for rickets in the first 2 years of life. On admission, he was hemodynamically stable. Hyperventilation test and Trousseau sign were negative. Chvostek sign was positive bilaterally. His weight was 17 kg and height was 111 cm (both below 5th percentile for age and gender). Systemic examination was normal. Mild pallor was present. There was no pain...
or restriction of movements at the temporomandibular joint bilaterally. A repeat attack of painful spasm of jaw muscles occurred while examining the oral cavity. During this episode he was not able to close his mouth [Figure 1]. He also developed carpopedal spasm of the right hand. Intravenous calcium gluconate (100 mg/kg) was given, which was followed by complete resolution of the spasm within 2 min. His investigations revealed the following: Serum calcium (Ca) 7.1 mg/dL, serum magnesium 2.2 mg/dL, serum phosphorous (P) 2.9 mg/dL, alkaline phosphatase 981 IU/L, serum parathormone 272 pg/mL (N, 9–65 pg/mL), 25-OHD 12 ng/mL (N, 30.0–74.0 ng/mL). His hemogram was suggestive of iron deficiency anemia. The Ca × P product of our patient was 20.59. Radiography of the wrist showed evidence of rickets in the form of metaphyseal splaying/fraying and decreased bone density. His liver and renal function test, arterial blood gas analysis, and serum electrolytes were normal. A diagnosis of acute multifocal (multiple noncontiguous body parts affected) dystonic reaction secondary to vitamin D–deficient rickets (VDDR) was made. He was started on vitamin D, calcium, and iron supplements. On follow-up after 3 months he is asymptomatic and well.

VDDR is an important and common problem in the developing countries. Presentations of VDDR vary widely, from asymptomatic to life-threatening situations.[9] Various atypical presentations, such as myelofibrosis, stridor, and dilated cardiomyopathy have also been described.[2-4] Thompson et al described a case of a 32-year-old woman with episodic and unilateral spasm of masticatory and facial muscles due to tetany. To the best of our knowledge our case is the first to be described with bilateral jaw muscle spasm due to tetany. The various causes of tonic or clonic jaw spasms include tetanus, bruxism, trauma, tetany, hysteria, pontine lesions, cold exposure, and convulsion.[5] These should be differentiated from involuntarily movements of the jaw, which occur in conditions, such as generalized and cranial dystonia, acute dystonic reactions to neuroleptic drugs, and tardive dyskinesia. Various masticatory movements may also be seen in epileptic automatisms and in response to oral/facial stimulation in patients with severe damage of the cerebral hemispheres/brainstem.[6] The prompt resolution of spasms after intravenous calcium distinguishes our case from the other causes. In VDDR, bone mineralization has a definite relation to the Ca × P product in the serum. Rickets would be present if the Ca × P product is below 30 as seen in our patient; and it would not develop if this product is above 30.[6] Vitamin D deficiency leads to impaired intestinal absorption of calcium, resulting in decreased serum calcium. This results in secondary hyperparathyroidism that initially maintains serum calcium levels at the expense of the skeleton. However, with prolonged vitamin D deficiency resulting in osteomalacia, calcium stores in the skeleton become relatively inaccessible, since osteoclasts cannot resorb unmineralized osteoid, and frank hypocalcemia ensues.[7] This could probably explain the hypocalcemia seen in our patient. Various hypotheses have been put forward to explain the mechanism of dystonia in vitamin D deficiency. The increase in serum calcium due to secondary hyperparathyroidism can stimulate dopamine release, resulting in dystonia.[8] As per another hypothesis, calcitriol is known to decrease the production of inflammatory cytokines in the brain. Thus in the absence of calcitriol, excessive production of inflammatory cytokines can occur, which could be responsible for the dystonia.[9]

In conclusion, physicians should be aware of the different ways in which hypocalcemia can present to ensure its early diagnosis and treatment. Also, vitamin D deficiency rickets should be considered in the differential diagnosis of children presenting with bilateral jaw muscle spasm.

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References