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E-mail :
editor.ijpast@gmail.com
editor@ijpast.in

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PROLONGED PROSTAGLANDIN INFUSION AND CORTICAL HYPEROSTOSIS IN AN INFANT WITH CYANOTIC CONGENITAL HEART DISEASE

Mrs. Azmath Fatima[1], Dr S. Satyanandam[2], Mr. Wasifuddin Ahmed[3]

ABSTRACT: Neonates with cyanotic congenital cardiac disorders sometimes need the administration of prostaglandins (PG) to maintain patency of the ductus arteriosus. For many problems, surgery is the only certain solution. It finds sporadic application inlasting for a longer period of time in premature infants. One adverse effect of extended prostaglandin medication that has just lately been noticed is reversible cortical hyperostosis. We provide a case of a newborn exhibiting this consequence as a result of long-term PGE1 treatment, with usual and comprehensive radiological findings. The spleen, kidney, adipose tissue, colon, liver, testicles, and lungs are just a few of the numerous arterial beds that are home to the prostaglandin degradation enzymes (prostaglandin -13 reductase and prostaglandin 15-OH dehydrogenase). In most cases, these tissues quickly degrade PGE, especially when it's in the lungs.18 There may be an increased systemic concentration of PGE in newborns with cyanotic congenital heart disorders due to impaired clearance of PGE and decreased pulmonary blood flow. The patency of the ductus arteriosus is also maintained in these individuals by administering pharmacologic dosages of PGE. Osteoarthritis might develop as a consequence of the elevated PGE levels in the blood. Nevertheless, the specific cause of cortical hyperostosis remains unknown. It might be a direct activation of osteoblastic cells that is dose-dependent.

Keywords: Prostaglandins(PG), Intermediate care nursery (ICN) Neonatal intensive care unit (NICU)

INTRODUCTION:

Dosage and length of time of continuous Prostaglandin E1 infusion seem to be associated with prostaglandin-induced thickening of the cortical layers. Months may pass after PGE therapy causes cortical hyperostosis. This side effect is most affected by how long it lasts, although it may also be dose-dependent. In most cases, the process resolves on its own when PGE is stopped, and it doesn't seem to affect the growth and development of bones thereafter. A full-term male Saudi infant weighing 3.7 kg was born properly with an Apgar score of 7 at 1 minute and 8 at 5 minutes, according to a case report. Cyanosis set in shortly after delivery in the intermediate care nursery (ICN), and he desaturated to 60% on a nasal cannula with 25% oxygen in just a few hours. After he was intubated and hooked up to a ventilator, he was sent to the neonatal intensive care unit (NICU). Situs solitus, levocardia, dilated right atrium, minor atrioseptal defect (ASD), severe tricuspid valve regurgitation (pressure gradient 70 mm Hg), tiny patent ductus arteriosus (PDA) of 2 mm, and prostaglandin E1 (PGE1) were all found during the echocardiography. I was infusing 0.05 micrograms per kilogram each minute. began on day three. On the fifth day, the baby made great progress and was able to be extubated. After delivery, chest x-rays revealed mild cardiomegaly; however, on days 3 and 9, further chest x-rays revealed cardiomegaly together with pulmonary plethora. The PG dosage was decreased to 0.04 microgram/kg/min on day 15. Further reduction to 0.02 microgram/kg/min of PG occurred on day 49.

Assistant professor[1,2,3]

Department of Pharmaceutics

Global College of Pharmacy, Hyderabad. Chilkur (V), Moinabad (M), Telangana- 501504.

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It was presumed that an isolated increase in alkaline phosphatase level (688 U/L; Normal<=500) on day 15 of PG treatment was associated with the therapy. On the 46th day of PG treatment, the alkaline phosphatase level peaked at 1860 U/L. On day 123, the alkaline phosphatase level was back to normal, but on days 140 and 149, there was a little increase in its value again.

The periosteal reaction/cortical thickening that was seen on the chest radiograph of the patient on day 53 had advanced by the time the babygram was taken on day 113, affecting all of the long bones. The patient had partly visible humerii. No discernible change in periostitis interval was seen in the visible bone skeleton in the last chest radiograph taken at day 157.

Due to the complicated etiology and dismal prognosis, the local cardiology and cardiothoracic doctors opted against performing any corrective surgery. The patient was flown overseas for potential surgical intervention after 5 months and released on prostaglandin E1 0.02 microgram/kg/min. At 8 months of age, the patient passed away after palliative surgery from which she could not recover.

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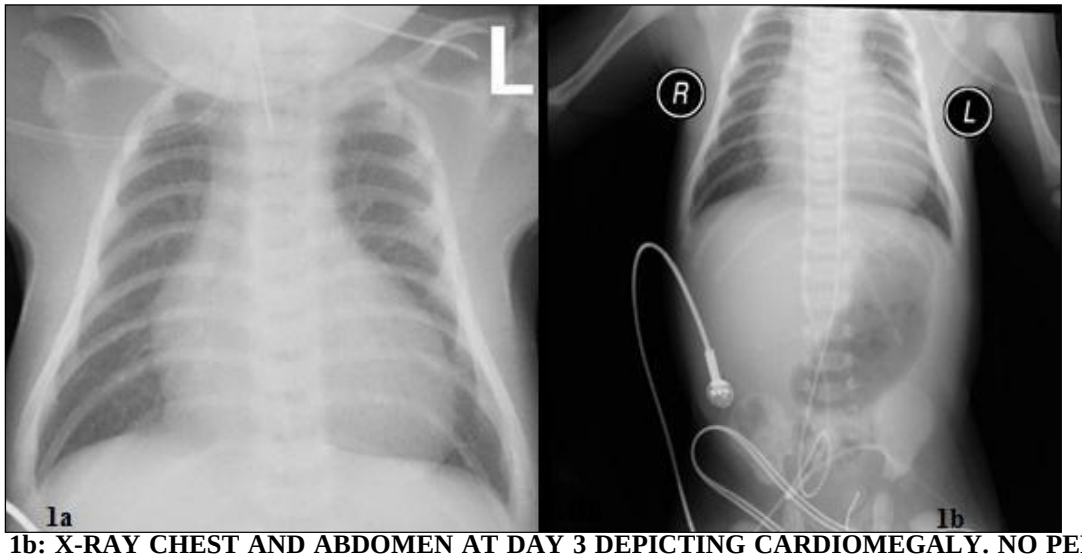

FIG. 1a AND 1b: X-RAY CHEST AND ABDOMEN AT DAY 3 DEPICTING CARDIOMEGALY. NO PERIOSTEAL REACTION ALONG THE RIBS, CLAVICLES OR HUMERII

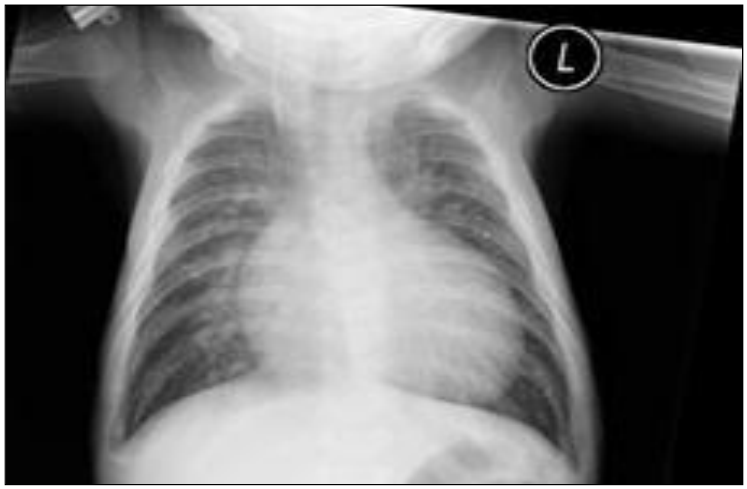

FIG. 2: CHEST X-RAY AT DAY 53, FIRST INCIDENCE OF SMOOTH PERIOSTEAL REACTION ALONG THE RIBS, CLAVICLES AND PARTIALLY VISUALIZED HUMERII

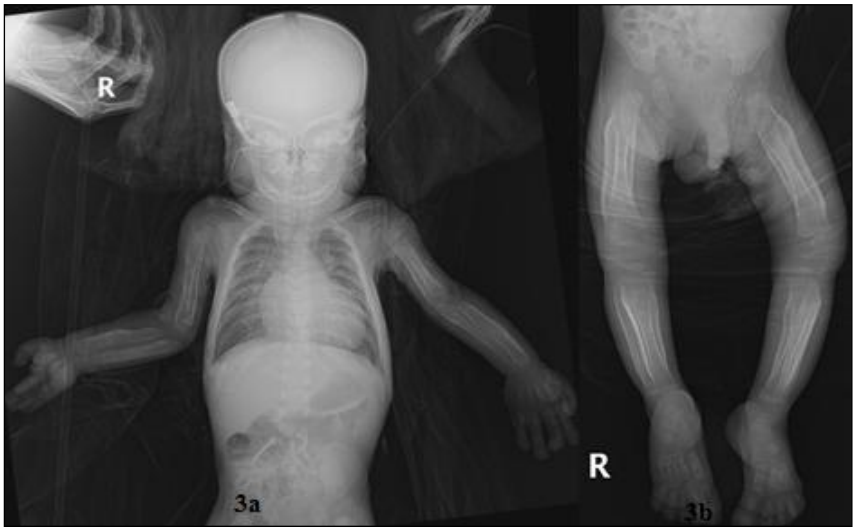

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FIG. 3a AND 3b: EXTENSIVE PERIOSTEAL REACTION ALONG THE LONG BONES

DISCUSSION: In 1975, Elliot maintained the ductus arteriosus patent 1 by using prostaglandins in congenital cyanotic heart disease. Neonatals with congenital ductal-dependent cardiac disorders have been shown to benefit from E-type prostaglandins (PGE), namely prostaglandin-E1 (PGE1, injectable form) and prostaglandin-E2 (PGE2, oral form), in terms of keeping the ductus arteriosus patency. 2.

Intravenous infusions of PGE1 at doses of 0.05-0.1 mcg/kg/minute are the norm for a brief period of time (anything from 6 hours to 20 days) prior to surgical intervention. More recent trials have successfully tested even smaller dosages, ranging from 0.003 to mcg/kg/minute. 3. Babies with low birth weight, infections, premature delivery, lack of access to a specialist hospital, or those waiting for a heart transplant may need its usage for an extended period of time, lasting weeks or months, in order to facilitate the development of their pulmonary arteries to an appropriate size for surgery. 4.

Apnea, fever, seizures, rash, flushing of the skin, vasodilatation with hypotension, necrotizing enterocolitis, diarrhea, and blockage of the gastric outlet are common adverse effects of short-term prostaglandin treatment. Soft tissue swelling, reversible cortical hyperostosis in the long bones, friability of the ductus arteriosus, and injury to the pulmonary arterial smooth muscle 5 are the most serious adverse effects of the uncommon case of chronic usage of alprostadil (PGE1).

It is thought that cortical thickening is a less prevalent adverse effect of prostaglandins. throughout the first few days after starting prostaglandins. It seems that the length or dose of continuous PGE1 infusion is associated to prostaglandin-induced cortical thickening. As the period of PGE infusion increases, the proportion of newborns developing hyperostosis rises from 42% at less than 30 days to 100% at more than 60 days. As early as 9–11 days of PGE infusion, these alterations become apparent.

The scapula and clavicles are less often damaged, but the long bones of the upper and lower limbs are the most prevalent, followed by the ribs. Multiple regions may be affected by cortical hyperostosis, which always seems symmetrical but does not necessarily manifest evenly. It has also been observed that there is an un-ossified zone at the borders of the cranial sutures, which causes the sutures to seem wider than they really are. However, there is no abnormal increase in head circumference. Skin and subcutaneous tissue thickening, roughness, coarse facial characteristics, hypertrichosis 6, large supraorbital ridges, and prominent facial features were all documented by Zarur et al.

The length of PGE1 medication is inversely proportional to the severity of periostitis and increases the serum alkaline phosphatase (ALP) activity. A similar finding regarding the use of ALP activity as a marker for PGE1-induced hyperostosis 7 was made by Nadroo et al.

The mandible is usually implicated in Caffey's illness, however this time it's not. The impact on long bones is usually symmetrical. Diaphysis is elevated over the periosteum, although no detrimental alterations have been seen.

Saved is metaphysis. Although there is no pathogenic increase in head size, several writers have documented radiological broadening of cranial sutures. Even while cortical hyperostosis caused by PGE therapy might last for months, it usually goes away once the medication is stopped.

CONCLUSION: Awareness of late complications of prolonged PGE therapy is essential. Hyperostosis is a common side effect of prolonged prostaglandintherapy if it is continued for more than three weeks. Although this is commonly seen with a prolonged use of PGE, it can present as early as 9 days after initiation of therapy. Isolated increase in serum alkaline phosphatase level might be helpful in making a diagnosis of cortical hyperostosis and canbe used as a marker for monitoring the activity of the

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periostitis. Early suspicion and detection may avoid costly and unnecessary laboratory evaluations or inaccurate diagnoses such as child abuse or osteomyelitis, during the resolution phase.

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