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Ductus arteriosus aneurysm in a neonate with inspiratory stridor

SHORT CASE REPORT

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Background

Inspiratory stridor in neonates is typically attributable to laryngomalacia, vocal cord paralysis, or subglottic stenosis. Less commonly, vascular anomalies can cause inspiratory stridor.

Case presentation

A neonate developed progressive, position-dependent inspiratory stridor and episodic oxygen desaturation on the third day of life. Laryngomalacia was excluded on initial investigation. Imaging revealed a ductus arteriosus aneurysm that was likely compressing the left recurrent laryngeal nerve, resulting in left-sided vocal cord paralysis. Despite marked respiratory symptoms, the neonate improved spontaneously and was managed conservatively.

Interpretation

This case underscores the importance of considering vascular anomalies – such as a ductus arteriosus aneurysm – in the differential diagnosis of neonatal stridor, particularly when symptoms are progressive or position-dependent. Timely recognition and targeted imaging facilitate accurate diagnosis, prevent unnecessary interventions and enable individualised management.

A neonate developed progressive inspiratory stridor and respiratory distress. Diagnostic evaluation confirmed an aneurysm of the ductus arteriosus. The condition improved spontaneously. This case report underscores the importance of considering vascular anomalies in neonates presenting with unexplained upper airway pathology.

A neonate was admitted to the neonatal intensive care unit (NICU) for observation as a result of respiratory distress following induction of labour due to meconium-stained amniotic fluid and pathological cardiotocography. Birth measurements were within the normal range and, following clinical assessment, the infant was considered stable and transferred to the maternity ward.

At 2.5 hours of age, transient postnatal hypoglycaemia occurred, and the infant was therefore admitted to the NICU for treatment and stabilisation, without subsequent complications. Stridor developed later in the clinical course.

On day 3, the infant developed inspiratory stridor with oxygen desaturation to 82 %, which resolved with supplemental oxygen. Laryngomalacia was suspected, but flexible laryngoscopy revealed only mildly oedematous, yellowish vocal folds and aryepiglottic folds, without definite signs of laryngomalacia.

Despite continued supplemental oxygen as required, the stridor persisted, and by day 4 minimal jugular retractions had developed. On day 5, fluids were restricted, as mild fluid overload was considered contributory to laryngeal oedema. The infant was evaluated for jaundice, but bilirubin concentrations remained below the treatment threshold for phototherapy. A single oral dose of dexamethasone 0.15 mg/kg was administered on day 6, without clinical effect.

On day 7, the inspiratory stridor worsened, with frequent oxygen desaturation to around 80 % and brief apnoeic episodes. Significant jugular and subcostal retractions were observed. The symptoms resolved completely in the supine position with the head rotated to the left, but were present in other head positions, including the neutral position and rotation to the right. Continuous positive airway pressure (CPAP) therapy was initiated, and the infant was transferred to a university hospital for further evaluation.

On arrival at the university hospital on the same day, pronounced inspiratory stridor with subcostal and jugular retractions was observed; however, the infant maintained adequate oxygen saturation, and the CPAP therapy administered during transport was discontinued. Echocardiography performed on day 8 revealed a small patent ductus arteriosus and mild narrowing of the aortic arch. Repeat laryngoscopy (also on day 8) revealed left vocal cord paresis. In view of the position-dependent stridor and the absence of definitive upper airway findings, a vascular cause was suspected. The clinical picture remained unchanged throughout day 8.

CT angiography on day 9 revealed a ductus arteriosus aneurysm with probable compression of the left recurrent laryngeal nerve. This explained the position-dependent stridor secondary to left vocal cord paresis (Figure 1). From day 9 to day 12, stridor and retractions persisted. Oxygen saturation was predominantly > 95 % on room air, with occasional brief desaturation to around 90 %. The CT also showed mild narrowing of the aortic arch (Figure 1). Echocardiography was therefore repeated on day 12 and showed no clinically significant aortic arch narrowing. Surgical intervention was considered but not deemed necessary in view of spontaneous clinical improvement and the absence of significant compressive features or other high-risk radiological findings. The infant was transferred back to the local hospital on day 13.

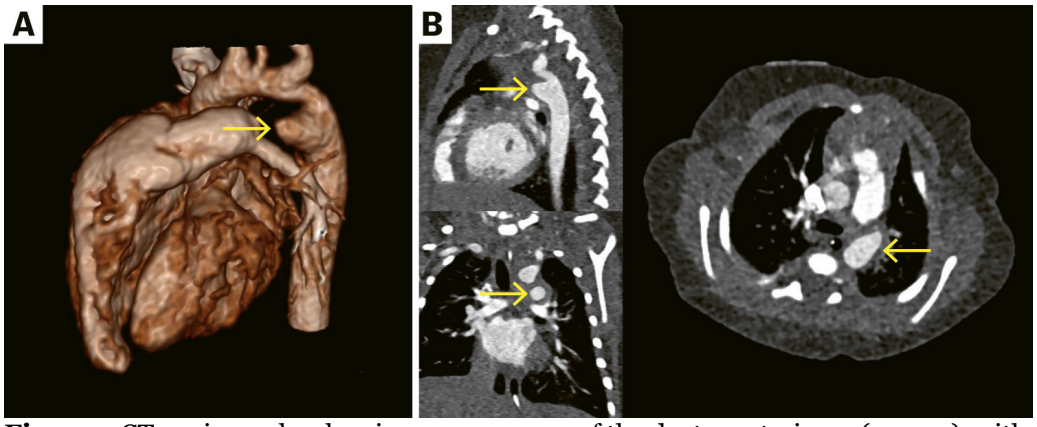

Figure 1 CT angiography showing an aneurysm of the ductus arteriosus (arrows), with 3D volume rendering (A) and multiplanar reconstructions (B).

The patient remained clinically stable, with spontaneous and complete regression of stridor and retractions following readmission to the local hospital. The ductus arteriosus aneurysm was considered a pathological variant of delayed ductal closure, with expected spontaneous regression. The patient was discharged on day 15 with ongoing multidisciplinary follow-up, including echocardiography and monthly assessment by an ENT specialist for three months.

Discussion

Stridor in neonates is typically caused by laryngomalacia, vocal cord paresis, or subglottic stenosis (1). In cases of atypical or progressive stridor, particularly when symptoms are position-dependent, vascular anomalies should be considered.

In this case, the stridor was caused by unilateral vocal cord paresis, which could be explained by mild compression of the left recurrent laryngeal nerve secondary to a ductus arteriosus aneurysm. The left recurrent laryngeal nerve loops around the ligamentum arteriosum, in close proximity to the aneurysm, and innervates all intrinsic laryngeal muscles except the cricothyroid muscle. Dysfunction of this nerve likely resulted in impaired neuromuscular control of the laryngeal musculature thereby reducing vocal cord mobility. This was considered the most plausible explanation for the observed clinical findings. Supine positioning with the head rotated to the left likely reduced the degree of compression and resulted in complete symptom relief.

Ductus arteriosus aneurysm is a rare condition, but increased use of advanced antenatal and postnatal imaging has led to higher detection rates. Reported prevalence is 0.5–1 % in historical neonatal autopsies and 1.5 % on modern fetal ultrasound (2–4). Most cases of ductus arteriosus aneurysm are asymptomatic, regress spontaneously and can be managed with echocardiographic follow-up (5). The condition is typically diagnosed by fetal echocardiography in the third trimester or in the early postnatal period, often as an incidental finding (5). CT or MRI is recommended in cases of persistent symptoms, diagnostic uncertainty, or suspected complications such as thromboembolism, infection, or airway compression (5).

Surgical treatment is considered in persistently symptomatic patients with significant compressive features affecting adjacent structures, thrombus formation, embolisation, extravasation or haematoma (which can indicate rupture), or imaging evidence of infection, regardless of aneurysm size (5, 6). Surgery can also be considered if the condition persists beyond the neonatal period, as a persistent patent ductus can increase the risk of complications (6, 7). In our case, conservative management was sufficient due to spontaneous improvement and the absence of high-risk radiological findings.

This case report illustrates the importance of including vascular anomalies in the differential diagnosis of persistent, unexplained, or position-dependent stridor in neonates. Prompt diagnosis using appropriate imaging modalities enables accurate management and helps avoid unnecessary interventions.

The patient's parents have consented to publication of the article.

The article has been peer-reviewed.

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