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CASE STUDY

Amniotic Band Syndrome: A Case Series

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Abstract

Amniotic band syndrome is an uncommon condition associated with multiple congenital anomalies. It affects about 1 in 1,200 to 1 in 15,000 live births and is often a consequence of fibrotic amniotic bands extending from the placenta to the fetus. It is associated with various fetal structural anomalies, notably craniofacial and limb anomalies. Six fetuses were collected from the Department of Obstetrics and Gynecology, RIMS, Imphal, after obtaining permission from the concerned authorities and parents. The fetuses were examined externally, dissected, and radiological examination was done wherever appropriate. On examination, Fetus 1 had encephalocele in the frontal region, cleft palate, malformed nose, abdominoschisis, bilateral clubfoot, and amputation of the right index, middle, and ring fingers. Fetus 2 had amputation of the left hand associated with left-sided gastroschisis. Fetus 3 had amputation of the right 1st, 2nd, 3rd, and 4th toes. Fetus 4 had an anterior abdominal wall defect associated with amputation of the right 2nd, 3rd, and 4th fingers. Fetuses 5 and 6 showed no brain tissue in the area of the defect. The amniotic membrane was attached to the scalp in both cases. Amniotic band syndrome is a sporadic condition that can result in various degrees of limb defects and major organ malformations. Amniotic band syndrome should be considered in any fetus presenting with congenital anomalies, especially those involving defects of the extremities and/ or body wall.

Keywords Amniotic band syndrome, Amniotic bands, Intrauterine amputation, Constriction ring

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1 Introduction

Amniotic band syndrome (ABS) is an uncommon condition affecting parts of the developing fetus when fibrous strands from the amniotic sac entangle it. The estimated incidence of ABS ranges from about 1 in 1,200 to 1 in 15,000 live births, but the incidence is much higher in stillbirths and miscarriages.[1-3] Out of the total 3% of major malformations in the general population, ABS accounts for 1-2%.[4, 5] There is no preference for gender or ethnicity; males and females are equally affected. These fibrous bands can lead to various birth defects, depending on when and where they attach, including constricted limbs, fused fingers or toes (syndactyly), cleft lip or palate, and more serious conditions, such as missing portions of the brain (anencephaly) or defects in the body wall. This condition usually occurs during a crucial period of fetal development, i.e., between 28 days after conception and 18 weeks of pregnancy.[6]

The study was conducted between June 2020 and August 2022 at the Department of Anatomy, Regional Institute of Medical Sciences, Imphal. Permission was obtained from the Research Ethics Board of the institution. The fetuses were collected from the Department of Obstetrics and Gynecology, RIMS, Imphal, after obtaining informed consent from the parents. The fetuses were the result of stillbirth, spontaneous abortion, or medical termination of pregnancy. They were subjected to external and internal examinations, as well as radiological examinations wherever appropriate.

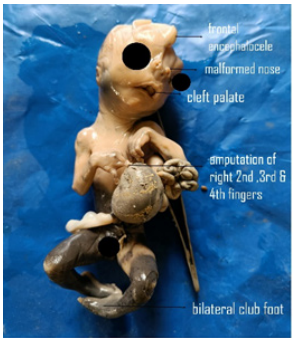
In this series of six fetuses, all were born to non-consanguineous marriages with no family history of ABS. The common anomaly in all six fetuses was the presence of fibrotic bands.

Case 1

The fetus was a 15-week-old male. External examination showed a left supraumbilical anterior abdominal wall defect (2.5 × 1.5 cm), suggesting gastroschisis. The herniated organs included the liver, stomach, spleen, and small and large intestines. A frontal encephalocele with cleft palate and a malformed nose was observed. There was amputation of the right 2nd, 3rd, and 4th fingers, and the left big toe, with fibrous bands attached to them. Bilateral clubfoot was present (Figure 1a). Encephalocele was confirmed by CT scan (Figure 1b).

Case 2

The fetus was a male with a gestational age of about 38 weeks, weighing 3.2 kg. External examination showed amputation of the left hand (Figure 2a). In addition, there were associated defects, e.g., left-sided gastroschisis with a hiatus measuring about 4.5cm by3cm through which the liver and large intestine were seen herniating. The appendix and caecum were present in the left lumbar area with a right-sided sigmoid colon (Figure 2b). There was also an imperforate anus (Figure 2c). Radiography of the affected limb showed the absence of all metacarpals and phalanges (Figure 2d).



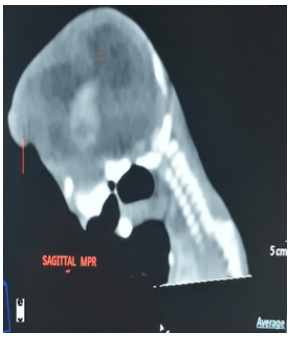


Figure 1a (left) Anterior abdominal wall defect, frontal encephalocele with cleft palate and malformed nose was seen, amputation of the right 2nd, 3rd, and 4th fingers and left big toe, bilateral club foot. **Figure 1b (right)** CT scan showing encephalocele

Figure 2a Amputation of the left hand

Figure 2b Anterior abdominal wall defect

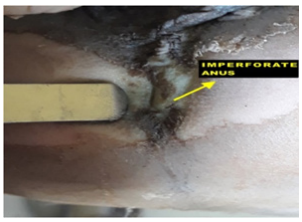


Figure 2c Imperforate anus

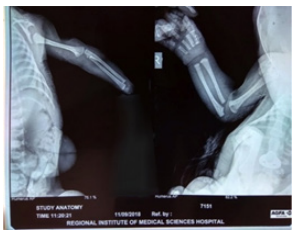


Figure 2d Absence of all metacarpals and phalanges

Case 3

A female fetus with a gestational age of 17 weeks showed amputation of the right 1st, 2nd, 3rd, and 4th toes (Figure 3).

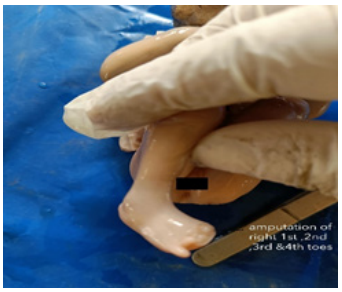


Figure 3 Amputation of the right 1st, 2nd, 3rd, and 4th toes

Case 4

The fetus was a 19-week-old female. External examination showed an anterior abdominal wall defect with herniation of the abdominal viscera. The herniated organs included the liver, stomach, spleen, and small and large intestines. There were also a calvaria and a cleft lip. There was amputation of the right 2nd, 3rd, and 4th fingers (Figures 4a and Figures 4b).

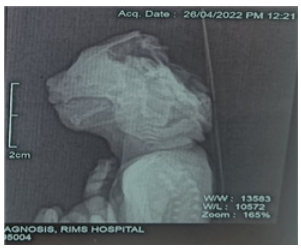


Figure 4a (left) Anterior abdominal wall defect with herniation of the abdominal viscera, acalvaria, cleft lip, and amputation of right 2nd, 3rd, and 4th fingers. **Figure 4b (right)** X-ray showed acalvaria

Cases 5 and 6

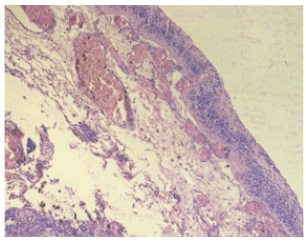
Both were male foetuses, approximately 24 and 38 weeks of gestational age. Grossly, there was no brain tissue in the area of the defect. Only a part of the brainstem and spinal cord was seen. There were bulging eyes, broad noses, and folded ears. The amniotic membrane was attached to the scalp in both cases. Histological examination of the tissue in the defect area revealed the presence of granulosa cells and degenerated vascular tissue with absent neuronal tissue (Figure 5a, Figure 6a, Figure 5b, Figure 6b and Figure 7).

Figure 5a Absence of brain tissue in the area of the defec

Figure 6a Absence of brain tissue in the area of the defect

Figure 5b Amniotic band attached to the scalp

Figure 6b Amniotic band attached to the scalp



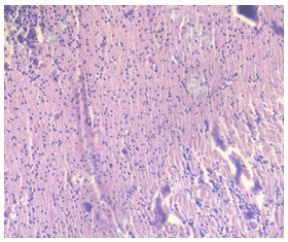


Figure 7 Granulosa cells and degenerated vascular tissue with absent neuronal tissue

2 Discussion

ABS deformities are likely caused by early rupture of the amniotic membrane, leading to the formation of mesodermal strands. These strands form both tight, constrictive tissue bands and wispy amniotic adhesions. Initially, the bands adhere to and loosely entangle the fetus; however, over time, they become constrictive rings and lead to an acute and later chronic hypoxic state. The presence of hypoxia and constrictive bands results in complex anomaly patterns, leading to a variety of fetal malformations.[7] However, not all anomalies associated with ABS are explained by the early amniotic rupture theory.[8]

These bands cause tissue indentation and encircle the digits, primarily affecting the extremities or limbs, and in rare cases, the neck, thorax, or abdomen .[9] Anencephaly, anophthalmia, cardiac anomalies, tracheoesophageal fistula, and renal agenesis are some of the anomalies that are not explained by the amniotic rupture theory. [10] Several risk factors can increase the likelihood of ABS during pregnancy, including obesity, being a first-time mother (nulliparity), smoking, a history of uterine procedures, intrauterine infections, Müllerian anomalies, living at high altitudes, and pre-existing maternal diabetes. [11] However, none of the cases in this series showed any of these risk factors.

Although the exact cause of ABS remains unknown, two primary theories regarding its pathogenesis have been proposed: the “extrinsic model” and the “intrinsic model”. [12] The intrinsic model, proposed by Streeter in 1930, suggests that both anomalies and fibrous bands originate from a common cause, resulting from a disruption in the developing germinal disc of the early embryo.[13] In contrast, Tropin’s extrinsic theory, introduced in 1965 [14], states that birth defects occur as a result of fibrous amniotic bands forming after the amnion ruptures. This rupture leads to a loss of amniotic fluid, allowing parts of the fetus to extrude into the chorionic cavity. Once trapped there, the fetus’s limbs experience vascular compression, which eventually leads to tissue necrosis.

The constrictive bands can be superficial, resulting in skin indentations, or can be deep, leading to severe vascular compromise. The severity of vascular compromise

can result in simple digit amputation or complex fetal anomalies such as encephaloceles and large facial clefts.[15] In the six cases presented above, the complexity of fetal injury was directly related to the level of vascular injury, with cases 1, 2, 4, 5, and 6 having the most severe vascular insults.

In ABS, involvement of the calvaria can lead to encephalocele, and in severe cases, it may resemble anencephaly. However, unlike typical anencephaly, where the calvarium is completely absent, anencephaly associated with ABS often shows asymmetric preservation of the skull.[16] Craniorachischisis, characterized by defects in the vertebrae and cranial vault, presents with bulging eyes, a broad nose, and folded ears. In such cases, the amniotic membrane is fused to the scalp, and the skull bones are missing above the point of attachment, as observed in the current study, i.e., in cases 5 and 6.

ABS can be diagnosed prenatally through ultrasound, which may occasionally reveal amniotic bands but more commonly detects malformations associated with ABS, along with oligohydramnios and reduced fetal movement.[17] ABS can be identified as early as 12 weeks of gestation, and most defects are detectable during routine ultrasound screenings.[18]

The treatment of ABS primarily involves surgery, with each case requiring a tailored approach. If prenatal diagnosis reveals fetal anomalies that are incompatible with life, termination of the pregnancy is recommended.[1]

3 Conclusion

ABS is sporadic and can lead to varying limb defects and major organ malformations. It should be considered in every fetus with congenital anomalies, particularly those involving the extremities and/or body wall. Prenatal diagnosis is possible through careful pregnancy monitoring and early first-trimester ultrasound. Early detection can help reduce the severity of the anomalies.

Declarations

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Artificial Intelligence Disclosure

The authors declare that this manuscript was prepared without the use of AI tools.

Authors' Contributions
All authours have contributed in the perperartion of this study.

Availability of Data and Materials
The data analyzed in this study are available upon reasonable request.

Conflict of Interest
The authors declare that they have no conflict of interest.

Consent for Publication
Not applicable.

Ethical Considerations
Ethical approval code for the study is A/206/REB-Comm(FP)/RIMS/2015/201/07/2023.

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References

1. Burk CJ, Aber C, Connelly EA. Ehlers-Danlos syndrome type IV: keloidal plaques of the lower extremities, amniotic band limb deformity, and a new mutation. Journal of the American Academy of Dermatology. 2007;56(2 Suppl):S53-4.
2. Poeuf B, Samson P, Magalon G. [Amniotic band syndrome]. Chirurgie de la main. 2008;27 Suppl 1:S136-47.
3. Ross MG. Pathogenesis of amniotic band syndrome. American journal of obstetrics and gynecology. 2007;197(2):219-20; author reply 20.
4. Brent RL. Environmental causes of human congenital malformations: the pediatrician's role in dealing with these complex clinical problems caused by a multiplicity of environmental and genetic factors. Pediatrics. 2004;113(4 Suppl):957-68.
5. Martínez-Frías ML. Epidemiological characteristics of amniotic band sequence (ABS) and body wall complex (BWC): are they two different entities? American journal of medical genetics. 1997;73(2):176-9.
6. Radhakrishnan SA. Amniotic band syndrome (ABS): A review. Asian J Nur Edu Res. 2011;1:41-4.
7. Kahramaner Z, Cosar H, Turkoglu E, Erdemir A, Kanik A, Sutcuoglu S, et al. Amniotic band sequence: an extreme case. Congenital anomalies. 2012;52(1):59-61.
8. Hunter AG, Carpenter BF. Implications of malformations not due to amniotic bands in the amniotic band sequence. American

journal of medical genetics. 1986;24(4):691-700.

9. Kim JB, Berry MG, Watson JS. Abdominal constriction band: A rare location for amniotic band syndrome. Journal of plastic, reconstructive & aesthetic surgery : JPRAS. 2007;60(11):1241-3.
10. Gonçalves LF, Jeanty P. Amniotic band syndrome. The Fetus. 1992;2:6588-1.
11. Ossipoff V, Hall BD. Etiologic factors in the amniotic band syndrome: a study of 24 patients. Birth defects original article series. 1977;13(3d):117-32.
12. Cignini P, Giorlandino C, Padula F, Dugo N, Cafà EV, Spata A. Epidemiology and risk factors of amniotic band syndrome, or ADAM sequence. Journal of prenatal medicine. 2012;6(4):59-63.
13. Streeter GL. Focal deficiencies in fetal tissues and their relation to intrauterine amutations. Contrib Embryol Carnegie Inst. 1930;22:1-46.
14. Torpin R. Amniochorionic mesoblastic fibrous strings and amnionic bands: associated constricting fetal malformations or fetal death. American journal of obstetrics and gynecology. 1965;91(1):65-75.
15. Lekovich J, Stewart J, Anderson S, Niemasik E, Pereira N, Chasen S. Placental malperfusion as a possible mechanism of preterm birth in patients with Müllerian anomalies. Journal of perinatal medicine. 2017;45(1):45-9.
16. Burton DJ, Filly R. Sonographic diagnosis of the amniotic band syndrome. AJR American journal of roentgenology. 1991;156(3):555-8.
17. Allen LM, Silverman RK, Nosovitch JT, Lohnes TM, Williams KD. Constriction rings and congenital amputations of the fingers and toes in a mild case of amniotic band syndrome. Journal of Diagnostic Medical Sonography. 2007;23(5):280-5.
18. Merrimen JL, McNeely PD, Bendor-Samuel RL, Schmidt MH, Fraser RB. Congenital placental-cerebral adhesion: an unusual case of amniotic band sequence. Case report. Journal of neurosurgery. 2006;104(5 Suppl):352-5.