Amniotic band syndrome (ABS): can something be done during pregnancy in African poor countries? Three cases and review of the literature

D.B. Mian[1], K.L.P. Nguessan[1], G. Aissi[2], S. Boni[1]

[1]Department of Gynecology and Obstetrics of the University Hospital of Cocody, Abidjan (Ivory Coast)
[2]Department of Gynecology and Obstetrics, HUS-CMCO, Schiltigheim, Strasbourg (France)

Summary

Amniotic band syndrome (ABS) is a fetal congenital malformation, affecting mainly the limbs, but also the craniofacial area and internal organs. Two mains pathogenic mechanisms are proposed in its genesis. Firstly the early amnion rupture (exogenous theory) leading to fibrous bands, which wrap up the fetal body; secondly, the endogenous theory privileges vascular origin, mesoblastic strings not being a causal agent. The authors believe that the second theory explain the occurrence of ABS. The outcome of the disease during pregnancy depends on the gravity of the malformations. Interruption of the pregnancy is usually proposed when diagnosis of severe craniofacial and visceral abnormalities is confirmed. Whereas minor limb defects can be repaired with postnatal surgery. In case of an isolated amniotic band with a constricted limb, in utero lysis of the band can be considered to avoid a natural amputation. In an African country, such treatment is not possible as far as the antenatal diagnosis.

Key words: Amniotic band syndrome; Pathogenesis; Prenatal diagnosis and neonatal management; Fetal surgery; Doppler.

Introduction

Amniotic band syndrome (ABS) is a set of complex congenital malformations. They concern mainly limbs but also the craniofacial region and thoraco-abdominal axis. They are asymmetric, polymorphic, and have no embryological systematization [1]. If clinical signs are well known for many years [1, 2], the etiology and pathophysiology are always discussed [3-8]. B mode ultrasound (US) and color Doppler are essential in its antenatal diagnosis. In utero surgery in the treatment of amniotic band (AB) is an opportunity which then makes essential the understanding of its physiopathogenic process [9, 10]. It is however limited to developed countries where it is used everyday. In African poor regions, echography is not available in all the public maternities, as it is too expensive and therefore is inaccessible for the majority of pregnant woman.

The main objective of this work is to highlight the difficulties in an underdeveloped country, in the diagnosis and management of ABS, and their effects on the fetus. Thus, in three differing cases of ABS, the authors recall the epidemiology, clinical features, and prognosis of this disease. In a second step, they discuss the mechanisms of its supposed etiopathogenesis, its etiology, natural course, and therapeutic possibilities in their context.

Case Report

Case 1

The patient, 25-years-old, with no particular antecedent is followed in the present department for her fourth pregnancy (G4P3). US scan performed at 13 weeks of gestation (WG) was normal with a neck measuring 1.9 mm. Screening for trisomy 21 with serum markers of first trimester had not been performed. The routine US scan for fetal morphology was made at 22 WG. It showed anomalies of all the right hand fingers (syndactyly) except the thumb, associated to a voluminous edema of the right foot (Figures 1a, 1b). A second US scan was then requested for suspicion of ABS. It was taken three weeks later by another operator and confirmed abnormalities of the fingers of both hands and the right foot with the presence of amniotic band (Figures 2a, 2b). Fetoscopy in a reference center was then requested but refused by the family who asked for pregnancy medical interruption due to their small income, anguish, and anxiousness. The medical termination of pregnancy was obtained with prescription of oral prostaglandin (misoprostol). The macroscopic analysis confirmed the diagnosis by viewing a large amniotic string on the right foot with downstream huge edema of the instep and the sole. There were also upper limb disorders with syndactyly of the right hand (digits 2-3-4-5), a distal amputation, and syndactyly of the fingers of the left hand (digits 2-3-4) which preserved integrity of the thumb (Figures 3, 4). Pathological examination of the placenta and amnion confirmed the diagnosis of ABS.

Case 2

The patient, 35-years-old, without medical history, was in the fifth pregnancy of four living children. (G5P4) She was a housewife with low level of education as well as her husband. She was received for a systematic morphological US scan at 30 weeks. It showed an amputation of the right leg associated with edema of the left foot (Figure 5) and a clubfoot on the left. The fetal heart activity was normal and regular. Pregnancy was then carried to completion and childbirth was completed without major incident. Examination of the newborn found him in good health. The authors also noted the presence of an amputation of the right leg associated with club-foot (Figure 6) of left leg.

The newborn was then transferred to a neonatal orthopedic surgery where a splint was made for the foot. The output of the mother

Revised manuscript accepted for publication February 24, 2013

Figure 1. ·— Ultrasonography at 22 weeks gestation.
a) Right hand (syndactyly of digits 2-3-4-5.
b) Right foot edema.

Figure 2. — Ultrasonography at 25 weeks gestation.
(a) Right foot large downstream edema
(b) Amniotic band.

Figure 3. — Right foot large furrow constriction and downstream edema.

Figure 4. — Left hand syndactyly of digits 2-3-4.

Figure 5. — Right leg amputation with edema of left foot at US.

Figure 7. — Newborn at birth. Deep circumferential constriction band of right lower limb extremity associated with marked lymphedema and right clubfoot. No signs of soft-tissue necrosis or ulcerations are present.

was allowed 12 hours after birth in the absence of postpartum complications.

Case 3

The patients, 28-years-old, primiparous was received in emergency maternity for uterine contractions and loss of water on a term pregnancy. This pregnancy was characterized by poor monitoring because no prenatal diagnosis or prophylaxis had been performed. The examination on admission found woman at the end of the pregnancy term with imminent delivery. The prognosis of natural vaginal delivery was good, so the authors permitted it to continue. One hour later, she gave birth, without difficulty after perineum section, a newborn in apparent good health. Score Apgar 9 at third minute then 10 at 5 minutes. After delivery perineum

Figure 6. — Right leg amputation and left clubfoot.

suture the general examination of the newborn in the delivery room found at the left lower limb many cutaneous constriction furrows of the foot, with large downstream edema (Figure 7). The newborn was transferred to a neonatal orthopedic surgery for better decision support. The output of the mother was allowed 12 hours after delivery. Pathological examination of the placenta and amnion were not done to confirm diagnosis of ABS.

Discussion

Epidemiological factors

ABS is not frequent and its incidence is between one in 1,200 and one in 15,000 live births in developed countries [5]. Higher numbers of one in 55 to one in 250 [11], were recognized histological at examination of products miscarriages and reflect the lethal character of the syndrome, due to their incompatibility with life [2, 6, 8, 12, 13], or by strangling with the umbilical cord (UC) [5, 11, 14-17]. No racial predisposition or link to sex were found [18, 19]. Although a few familial cases have been described, the usually sporadic nature of this syndrome does not suspect a hereditary factor [19-23]. This frequency is underestimated in the underdeveloped countries as in Africa. Indeed, fetal pathological examination and statistics are not available. Moreover, antenatal US is not usually performed and most births take place in peripheric maternity hospitals, so the statistics are not available. These three reported cases were observed in the present maternity hospital and this is the reason why they could be reported.

Etiopathogenesis

Amniotic disease is a congenital disease whose etiology is unknown [24]. Despite numerous embryological, pathological, and experimental studies, there is no conviction about the origin or the ABS etiological factors [1]. Two opposing theories were found in literature to explain its genesis. The exogenous theory of Torpin [4] and the endogenous theory of Streeter [5].

According to Torpin's *exogenous theory* [5], the most commonly shared, the first factor was (oligoamnios) consecutive to breaking amnio-chorionic. It leads to an amnio-chorionic separation with leakage of amniotic fluid. Some anatomical parts of the fetus could then be partially exteriorized through the amniotic cavity, and thus become in close contact with chorion. The external surface of the amnion (mesodermic) then produces fibrous bands that surrounds and strangles them. This explains the phenomenon of constrictions and amputations [2, 5, 7]. AB is a very fibrous band of chorio-amniotic tissue which is often in contact with constriction skin furrows, digital amputations, pseudo-syndactyly, and facial clefts [25]. This is the common element that brought together this heterogeneity of the ABS. [25]. Fetus compression is secondary cause by oligohydramnios who plays a role in the genesis of certain malformations such as clubfoot [5, 26]. The severity of the malformation depends on the term of amniotic rupture [8, 27]. Craniofacial and visceral anomalies result from early rupture of membranes (28-45 days of gestation), whereas a constriction or amputation results from a late break (45 days to 18 weeks of gestation) [2]. Causal factors of early rupture of the amnion are not determined with certainty. The traumatic nature of amniocentesis is often reported as a possible cause of ABS [16, 20, 26, 28]. This theory, however, is deeply contested by many authors [4-9] because it does not explain the number and variety of malformations described [13, 29, 30]. In addition, for Bronshtein *et al.*, despite the multitude of US scans performed antenatally, no one has reported the following sequence: normal fetus –> membranes rupture –> amnion adhesion to fetus –> constriction or amputation [31]. In fact, he rarely found an AB in contact with an amputated limb segment when combining intrauterine AB and amputation segment. Other authors found no amniotic rupture in many fetuses with their anatomical elements in contact with an AB [8, 13]. Lastly, after amniotic membranes rupture, many extra-amniotic pregnancies were described without any AB or neonatal abnormality [32].

For the endogenous theory defended by Streeter, "there is no evidence demonstrating that intrauterine amputation was secondary to AB or an adhesive process or a constrictive process" [4]. An embryonic disc development anomaly, before the third week of gestation, is the cause of malformations as well as straps. The straps have no causal role [7, 16]. For Van Allen *et al.*, This anomaly would be responsible for infarction of embryonic vessels, causing tissue necrosis, cessation of embryonic development, hemorrhagic necrosis and the formation of AB by adhesion of the amnion to the necrosis parts [6]. Thus, the formation of straps could be compared to that of intraperitoneal adhesions after surgery [6]. To Hartwig *et al.*, this theory explains the variety of clinical manifestations of this disease, and in particular the internal organ attack [33]. Experimental works helped to support this theory, or at least the endogenous basic element. Glucose injection into uterine subsidiaries of pregnant rabbits causes constrictions and limb amputations associated with ABs [34]. Hemorrhagic extravasation superficial vessels of fetus can explain the pathogenesis. Finally, constitutional collagen anomaly [34], imperfect osteogenesis [35], and bullosa epidermolysis [36] could also be the cause of some ABS. Despite all these numerous works, none of them have demonstrated the two main theories. It is therefore highly likely that several non-exclusive mechanisms involved in the formation of different lesions of the disease [8]. In the present cases the authors believe that ABS seems more to be explained by the endogenous theory of Streeter, than that of Torpin [4, 5]. Indeed, lesions occurred when there were no abnormalities of amniotic fluid and the membranes were intact.

Clinical aspects

The ABS is a syndrome associated with multiple malformations, polymorphics, and asymmetrics without any embryonic systematizing [12]. Its severity is highly variable. The authors have found isolated skin furrow, craniofacial and visceral malformations, often incompatible with life [2-5]. The frequency of these malformations in the ABS is very variable according to some authors [3, 8, 13], but 77% of cases would at least present two anomalies [37]. Anomalies of limb are the most common, especially in patients with a single anomaly [2] (Figures 6, 7).

in furrows occur in decreasing order of frequency in the fingers, with relative sparing of the thumb (Figures 1a, and 5), toes, legs, forearms, hands, feet, arms, and legs [25]. Occurrence in the trunk is exceptional. Regarding amputations, they are asymmetric, often reaching the distal ends (Figure 6) and most often reaching the toes [39] (Figure 4). Amputation of an entire limb segment is uncommon [3]. The clubfoots, less specific abnormalities, would be a consequence of oligohydramnios (Figure 7) [1]. Craniofacial abnormalities affect especially the skull (anencephaly, or encephalocele). They have a lateralized character or anterior evocative without being pathognomonic [12]. Deformities such as facial clefts are possible [40]. Contrary to genetic malformations, these craniofacial anomalies are often asymmetric and outside embryonic development lines [25]. They are attributed to reach this region by AB which create an obstacle to the fusion of embryonic bud. Visceral malformations of thoraco-abdominal axis are more uncommon and never isolated. [2] They often correspond to closing parietal anomalies (gastroschisis and omphalocele). As regards the annexes, funiculars anomalies (short cord) are considered as classic AB, although highly controversial [11].

Prenatal diagnosis

US scan remains the main complementary investigation that can be coupled to fetoscopy. They are unavailable in our poor countries. For some authors, it allows the diagnosis of

some ABS in the first trimester, according to the nature and severity of malformations [27, 41]. Craniofacial defects and thoraco-abdominal can be detected from the first US scan at 10-12 WG. Isolated anomalies of limbs are generally diagnosed at a new ultrasound. In the present first observation, the diagnosis was made early. It was late or unknown in the other observations. In poor countries, rarity of the disease makes the training of sonographers difficult. Besides, the US machines are not high-performance. In fact, the prenatal diagnosis of AB is not essential to the diagnosis of ABS. It should however be considered in the presence of some characteristic signs such as asymmetric constriction or amputation of a limb with lymphedema end downstream of the constriction (Figure 7), a craniofacial malformation asymmetric (encephalocele, cleft lip, and palate); coelosomies the presence of pseudo-syndactyly or BA in contact with the injured fetal pole. All these lesions are accessible for trained sonographers with a high-performance machines.

Color and pulsed Doppler is also essential to confirm antenatal diagnosis of AB. It shows no vascularization in color and pulsed Doppler in the straps [42]. Most of the US scan machines used in the present country do not include color and pulse Doppler. Other associated signs may be find such as oligohydramnios, decreased fetal movement, and clubfoot that are a consequence of oligohydramnios [7, 8]. The study of umbilical Doppler could theoretically detect constrictions cord in utero. Color Doppler and pulsed of downstream limb of a constriction could be a key parameter to determine the natural history of the constriction. Tadmor *et al.* [41] have recently report the occurrence of in utero amputation secondary to constriction of a limb by an AB. There was a decrease and an absence of vascular flow, downstream of the constriction, the day before amputation [41]. In the present observations, this review has not been completed, due to its unavailability (observations 1 and 2), or late diagnosis at birth (observation 3). The study of Doppler strangled limb would assess the extent of the consequences of the constrictor ring to determine the natural history of the disease and inform parents of postnatal care. However, it is only an attractive hypothesis which must be demonstrated [1].

Radiography of the uterine contents can be use. Its diagnosis skeletal abnormalities (vertebral and limb deformities) and objectify the preservation of bone mineralization in the strangled limb. The karyotype is still regular. However, its realization seems justified in cases of diagnostic doubt to eliminate differential diagnoses. It is rare that it is done in the present context because it is too expensive. Histology examination remains indispensable in all cases. It confirms the diagnosis by showing the presence chorion separated partially or all of the amnion in front of an ABS [13]. It can also highlight areas of necrosis and remodeling of the amnion associated with proliferation of fibroblasts in the lamina propria, which may correspond to the stigma of a earlier rupture of the amnion [11].

Differential diagnosis

When craniofacial or limbs anomalies are accompanied of AB, there is little doubt about the diagnosis. However, some anomalies and syndromes, sometimes hereditary, have similar characteristics to ABS. [43] It may be an anencephaly due to primary defect closure of neural tube, facial clefts (lip and palate), Cantrell pentalogy [9], limb-body wall complex [8, 43], and congenital tegumentary aplasia [25]. We must also consider sub-chorionic hemorrhage [41], lack of fusion of the amnion [8], second twin evanescence [44], biamniotic twin pregnancy [43], bilobed placenta [44] or circumvallata [41].

Disease natural evolution

The natural evolution of ABS is unpredictable [11]. However, two parameters have not yet been sufficiently evaluated, could have a useful predictive value. It is either the occurrence of distal lymphedema reflecting constriction of neurovascular structures and lymphatics, or the study of vascular flow (color and pulsed Doppler) within the strangled limb which could be the most predictive parameter of eventual amputation.

Despite a regular ultrasound monitoring, it would be impossible to differentiate constrictor rings leading to a future in utero amputation (fetal indication for surgery) and those responsible for a isolated constriction skin furrow (indicating an attitude not interventionist). In the presence of craniofacial and visceral polymalformations, the natural history is of little importance, the prognosis is known and dark. The situation is quite different in the case of isolated and superficial constriction of a limb, as was the case in the present observations 2 and 3. Schematically, we can retain 4 scenarios. Firstly, exceptional spontaneous regression without after-effects of constriction [45]. Secondly, the limb constriction becomes deep, with neurovascular structures strangulation and cutaneous necrosis and subcutaneous risk downstream. Thirdly ring constrictor causes limb in utero amputation (observations 2 and 3). Finally, the occurrence of lymphedema downstream of a constriction is a result of vascular compression of arterio-venous structures and lymphatic fearing a decrease in vascular perfusion in the distal segment and amputation in utero [46].

Prognosis

Obstetrical prognosis of patients with abnormal fetuses associated with the ABS do not change compared to the general population [2, 3, 18]. The prognosis of surviving children is variable and depends on the severity of fetal injury. Survivors with severe craniofacial malformations number are few and then have motor, behavioral, and cognitive disorders [18]. A medical interruption of pregnancy may be proposed when the fetal abnormalities are recognized.

In the case of isolated anomaly of the extremities, the prognosis is excellent [2]. However, the prognosis of isolated constriction is closely correlated with the state of the

cutaneous tissue and subcutaneous downstream, as well as a possible infringement of neural structures [38]. No statistically significant cohort study has evaluated the functional outcome of a constriction according to its severity. However, it is estimated that postnatal surgery preserves functional capacity close to normal in 50% [44]. The psychological trauma felt at birth of such children and the more frequent proceedings, must lead us to a better diagnosis in front of such malformations. As long as the diagnosis is made, which is not always the case with our poor countries treatment must be proposed to the parents.

Treatment

Only isolated or associated anomalies must be concerned by treatment (furrows constriction, pseudo-syndactyly, and club-feet). The severe craniofacial and visceral polymalformations generally incompatible with life, are inaccessible to any therapeutic option. Without any in utero treatment (observations 2 and 3), skin creases and pinch pseudo-syndactyly are irreversible but do not worsen after birth [47]. In case of vascular impact (venous stasis and lymphedema), excision with release of neurovascular bundle is urgent and performed within the first 48 hours of life. In front of constrictive circumferential furrows, microsurgical excision in one time gives a better cosmetic result [48]. Instituted edema does not regress. If the constricting furrow is very tight, the skeletal lesions with thinning of bones or multiple fracture can be observed [49]. Pseudo-syndactyly will be released early in the neonatal period, to limit the movement towards camptodactyly. When the end of the two fingers is connected by a single membrane, it may simply be excised and sutured. The most important mergers, will be associated with dissection of a skin graft to prevent adhesions during healing [25, 47].

In the case of digital amputation, a secondary release can be performed to give the maximum length to amputated fingers [49]. The clubfeet with a knee furrow, respond less well to orthopedic treatment than idiopathic idiopathic. To avoid the occurrence of in utero amputation, some authors have suggested in utero release of constricting ring. This was the case of Quintero *et al.* [10] in 1997 that released in utero constrictor amniotic ring under US and endoscopy. Since no other similar experience has been reported in the literature. In fact, in utero treatment of isolated constriction of limb is controversial. Indeed, the natural evolution of a limb constriction remains unpredictable. There is currently no indication for in utero surgery in the ABS. Many authors dispute the fact that in utero amputation is linked to the hypothetical process of constrictive AB [6, 9, 11]. In addition, this surgical procedure is not simple nor free of complications. Finally, infants with a superficial skin furrows have an excellent functional prognosis, in contrast to those with neurovascular structures' constriction [1]. Beyond 32 weeks, fetal extraction could be an interesting compromise between the functional prognosis and prematurity, and an alternative to in utero surgery. Nonetheless, before considering in utero treatment, it is essential to ensure the absence of other malformations. In the present second observation, the expectation was observed due to our inability to perform in utero surgery.

Conclusion

ABS is an embryo-fetopathy acquired with a set of asymmetric malformations, primarily in the limbs. Although its pathogenesis remains controversial, it probably results due to many different pathological processes. Therefore in poor Africans countries were illiteracy is higher and incomes of population lower, the support of ABS always remains difficult and fetal prognosis is dismal. More monitoring of pregnancies and in utero surgery must be practiced more frequently in African countries to improve it.

References

[1] Sentilhes L., Verspyck E., Patrier S., Eurin D., Lechevallier J., Marpeau L.: "Maladie des brides amniotiques: étiopathogénie, diagnostic anténatal et prise en charge néonatale". *J. Gynecol. Obstet. Biol. Reprod.*, 2003, *32*, 693.

[2] Seeds JW, Cefalo RC, Herbert WNP. Amniotic Band Syndrome. *Am. J. Obstet. Gynecol.,* 1982, *144*, 243.

[3] Chemke J, Graff G, Hurwitz N, Liban E.: The amniotic band syndrome. *Obstet. Gynecol.,* 1973, *41*, 332.

[4] Streeter GL. Focal deficiencies in fetal tissues and their relation to intra uterine amputation. *Contrib. Embryol.,* 1930, *22*, 1.

[5] Torpin R**.** Amniochorionic mesoblastic fibrous strings and amniotic bands. *Am. J. Obstet. Gynecol.,* 1965; *91*, 65.

[6] Van Allen MI, Curry C, Gallagher L. Limb Body Wall Complex: I. Pathogenesis. *Am. J. Med. Genet.,* 1987, *28*, 529.

[7] Lockwood C, Ghidini A, Romero R, Hobbins JC. Amniotic band syndrome: reevaluation of its pathogenesis. *Am. J. Obstet. Gynecol.,* 1989, *160*, 1030.

[8] Moerman P, Fryns JP, Vandenberghe K, Lauweryns M. Constrictive amniotic bands, amniotic adhesions, and limb-body wall complex. Discrete disruption sequences with pathogenetic overlap. *Am. J. Med. Genet.,* 1992, *42*, 470.

[9] Zimmer EZ, Bronshtein M. Early sonographic diagnosis of fetal midline disruption syndrome. *Prenat. Diagn.,* 1996, *16*, 65.

[10] Quintero RA, Morales WJ, Philips J, Kalter CS, Angel JL. *In utero* lysis of amniotic bands. *Ultrasound Obstet. Gynecol.,* 1997, *10*, 316.

[11] Byrne J, Blanc WA, Baker D. Amniotic band syndrome in early fetal life. *Birth Defects,* 1982; *OAS XVIII*, 43.

[12] Mahony BS, Filly RA, Callen PW, Golbus MS. The amniotic band syndrome: antenatal sonographic diagnosis and potential pitfalls. *Am. J. Obstet. Gynecol.,* 1985, *152*, 63.

[13] Herva R, Karkinen-Jaaskelainen M. Amniotic adhesion malformation syndrome: fetal and placental pathology. *Teratology,* 1984, *29*, 11.

[14] Hong CY, Simon MA. Amniotic band knotted about umbilical cord: a rare cause of fetal death. *Obstet. Gynecol.,* 1963, *22*, 667.

[15] Burrows, Philips N. Strangulation of umbilical cord by amniotic band. *Am. J. Obstet. Gynecol.,* 1976, *124*, 697.

[16] Heifetz SA. Strangulation of umbilical cord by amniotic bands: report of 6 cases and literature review. *Pediatr. Pathol.,* 1984, *2*, 285.

[17] Kanayama MD, Gaffey TA, Ogburn PL. Constriction of the umbilical cord by an amniotic band, with fetal compromise. *J. Reprod. Med.,* 1995, *40*, 71.

[18] Ossipoff V, Hall BD. Etiologic factors in the amniotic band syndrome. A study of 24 patients. *Birth Defects.,* 1977, *OAS XIII*: 117.

[19] Keller H, Neuhauser G, Durbin-Stamm MV, Kaveggia EG, Schaaff A. "ADAM Complex". A pattern of craniofacial and limb defects. *Am. J. Med. Genet.,* 1978, *2*, 81.
[20] Etches PC, Stewart AR, Ives EJ. Familial congenital amputations. *J. Pediatr.,* 1982, *101*, 448.
[21] Lubinsky M, Sujansky E, Sanger W, Salyards P, Severn C. Familial amniotic bands. *Am. J. Med. Genet.,* 1983, *14*, 81.
[22] Zionts LE, Osterkamp JA, Crawford TO, Harvey JP. Congenital annular bands in identical twins. A case report. *J. Bone Joint Surg. Am.,* 1984, *66A*, 450.
[23] Pauli RM, Lebowitz RM, Meyer RD. Familial occurence of terminal transverse defects of the arm. *Clin. Genet.,* 1985, *27*, 555.
[24] Ameziane L., EL Bardouni A., EL Manouar M. La maladie amniotique à propos d'un cas. *Méd. du Maghreb.,* 1999, *77*, 33.
[25] Bahadoran P, Lacour JP, Ortonne JP. Le syndrome des brides amniotiques. *Ann. Dermatol. Venereol.,* 1997, *124*, 416.
[26] Moesinger AC, Blanc WA, Byrne J, Andrew D, Warburton D, Bloom A. Amniotic band syndrome associated with amniocentesis. *Am. J. Obstet. Gynecol.,* 1981, *141*, 588.
[27] Higginbottom MC, Jones KL, Hall BD, Smith DW. The amniotic band disruption complex. Timing of amnion rupture and variable spectra of consequent defects. *J. Pediatr.,* 1979, *95*, 544.
[28] Rehder H, Weitzel H. Intrauterine amputations after amniocentesis. *Lancet,* 1978, *1*, 382.
[29] Clavert JM, Clavert A, Wagner NI, Buck P. Experimental approach to the pathogenesis of the anomalies of amniotic disease. *J. Pediatr. Surg.,* 1980, *15*, 63.
[30] Young ID, Lindenbaum RH, Thompson EM, Pemburg ME. Amniotic bands in connective tissue disorders. *Arch. Dis. Child.,* 1985, *60*, 1061.
[31] Bronshtein M, Zimmer EZ. Do amniotic bands ampute fetal organs? *Ultrasound Obstet. Gynecol.,* 1997, *10*, 309.
[32] Yang SS, Sanborn JR, Levine AJ. Amniotic rupture, extra-amniotic pregnancy, and vernix granulomata. *Am. J. Surg. Pathol.,* 1984, *8*, 117.
[33] Hartwig NG, Vermeijs-Keers CHR, De Vries HE, Kagie M, Kragt H. Limb body wall malformation complex: an embryological etiology? *Hum. Pathol.,* 1989, *20*, 1071.
[34] Van [34] De [34]-Elejalde BR, De Elejalde MM. Prenatal diagnosis of perinatally lethal osteogenesis imperfecta. *Am. J. Med. Genet.,* 1983, *14*, 353.
[35] Marras A, Dessi C, Macciotta A. Epidermolysis bullosa and amniotic bands. *Am. J. Med. Genet.,* 1984, *19*, 815.
[36] Rushton DI. Amniotic band syndrome. *Brit. Med. J.,* 1983, *286*, 920.
[37] Light TR, Ogden JA. Congenital constriction band syndrome: pathophysiology and treatment. *Yale J. Biol. Med.,* 1993, *66*, 143.
[38] Miyajima K, Natsume N, Kaiwai T, Izuka T. Oblique facial cleft, cleft palate secondary to amniotic bands. *Craniofacial cleft – Palate J.,* 1997, *31*, 483.
[39] Takenori N, Ryosuke N. Amniotic band syndrome: serial ultrasound observations in the first trimester. *J. Clin. Ultrasound,* 1994, *22*, 275.
[40] Abuhamad AZ, Romero R, Shaffer WK, Hobbins, JC. The value of Doppler flow analysis in the prenatal diagnosis of amniotic sheets. *J. Ultrasound Med.,* 1992, *11*, 623.
[41] Tadmor OP, Kreisberg GA, Achiron R, Porat S, Yagel S. Limb amputation in amniotic band syndrome: serial ultrasonographic and Doppler observations. *Ultrasound Obstet. Gynecol.,* 1997, *10*, 312.
[42] Roth PH, Clerc-Bertin FL, Chabroud JP, Arbez-Gindre F, Maillet R. La maladie des brides amniotiques. *Médecine foetale et échographie en gynécologie* 1997, 4.
[43] Deruelle P, Hay R, Subtil D, Chauvet MP, Duroy A, Decocq J, Puech F. Diagnostic anténatal du "limb body wall complex". *J. Gynecol. Obstet. Biol. Reprod.,* 2000, *29*, 385.
[44] Pedersen TK, Thomsen SG. Spontaneous resolution of amniotic bands. *Ultrasound Obstet. Gynecol.,* 2001, *18*, 673.
[45] Evans MI. Amniotic bands. *Ultrasound Obstet. Gynecol.,* 1997, *10*, 307.
[46] Patterson TJS. Congenital ring constrictions. *Br. J. Plast. Surg.,* 1961, *14*, 1.
[47] Lamesch A. Le traitement des sillons de striction : plastie en Z multiple circulaire ou excision simple? *Chir. Pediatr.,* 1980, *21*, 411.
[48] Bouche-Pillon AM, Lefort G, Daoud S. Maladie amniotique. À propos d'une série de 20 cas. *Chir. Pediatr.,* 1987, *28*, 235.
[49] Tada T, Yonenobu K, Swanson AB. Congenital constriction band syndrome. *J. Pediatr. Orthop.,* 1984, *4*, 726.

Address reprint requests to:
D.B. MIAN, M.D.
BPV 13 Abidjan (Ivory Coast)
e-mail: bostondehimian@yahoo.fr