COCAINE USE AS A RISK FACTOR FOR ABDOMINAL PREGNANCY

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Failure to diagnose abdominal pregnancies can have disastrous morbidity/mortality consequences for mother and fetus. To make the diagnosis of abdominal pregnancy requires that the physician have a high index of suspicion and that he or she have a good understanding of the risk factors of abdominal pregnancy. This article presents data suggesting that maternal cocaine use is a risk factor for abdominal pregnancy, reviews the literature on the maternal/fetal effects of maternal cocaine use and the risk factors of abdominal pregnancy, and analyzes 55 cases of abdominal pregnancy. Maternal cocaine use correlated with a 20% rate of increase in the incidence of abdominal pregnancy compared with the 70% rate of decrease in the "before cocaine" time period. Recommendations are offered for management. (*J Natl Med Assoc.* 1998;90:277-283.)

Key words: abdominal pregnancy ◆ pregnancy ◆ cocaine use

Abdominal pregnancies are pregnancies that are located within the peritoneal cavity. Tubal, ovarian, and intraligamentous pregnancies are excluded from this definition. Abdominal pregnancies arise when a fertilized ovum, upon rupturing or exiting from the fallopian tube, finds a nidus of implantation within the peritoneal cavity.[1] Abdominal pregnancies are rare, with estimates of prevalence ranging from 1 in 6000 to 1 in 9000.[2] Yet consequences of abdominal pregnancy for maternal-fetal morbidity and mortality are sufficiently severe to warrant the immediate and full attention of the examining physician.

The purpose of this article is to heighten physician awareness by analyzing 55 cases of abdominal pregnancy and demonstrating that cocaine use is a risk factor for abdominal pregnancy. This objective was pursued for four reasons. First, there has been little updating of the early literature on the risk factors of abdominal pregnancy. Second, although there is a sizable literature on the maternal/fetal effects of maternal cocaine use, that literature does not appear to support the possibility of abdominal pregnancy as one of those effects. Third, the use of cocaine, particularly within the inner city, is a problem of significant proportions.[3] Finally, in light of the daunting diagnostic challenge that abdominal pregnancy presents,[2] physician analysis of risk factors remains a useful diagnostic tool.

The topic of cocaine use in pregnancy has recently and rapidly coalesced into a sizable body of scholarly literature. This literature forms the foundation for a piece such as the present one that purports to analyze the use of cocaine as a risk factor in the occurrence of abdominal pregnancies. It is instructive therefore to consider the nature of this foundation.

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LITERATURE REVIEW

Maternal Cocaine Use

There are at least five broad conclusions that can

be gleaned from a survey of the literature on cocaine use during pregnancy. A first broad conclusion is that there is enormous variation within this literature.[4] This variation exists with respect to methodology (eg, animal versus human studies and various statistical approaches) as well as conclusions regarding the effects of cocaine on mother and fetus.

A second broad conclusion is that the list of the possible fetal/neonatal effects of maternal cocaine consumption is indeed a long list. Some of these effects are discussed briefly below.

Abruptio Placentae. Several authors have presented data that suggest that abruptio placentae is associated with maternal cocaine use.[5-9] Two sets of authors[5,7] agree that the strength of the association increases with the intensity and frequency of maternal cocaine use.

Cardiac/Cardiovascular Effects. This set of effects is varied in its content. Identified effects include cardiac malformations[4]; hypoxemia, blood pressure, and heart rate increases[10]; and thrombocytopenia.[11]

Fetal Congenital Malformations. A few studies[8,12,13] have suggested an association between fetal congenital malformations and maternal cocaine use.

Intrauterine Growth Retardation. Intrauterine growth retardation was discussed by only one study.[8]

More Rapid Fetal Delivery. One study[14] found that there was a shorter latency period between labor and delivery, or, more rapid fetal delivery, among women who had recently used cocaine.

Meconium-Stained Amniotic Fluid. Meconium-stained amniotic fluid was found by only one study.[6]

Fetal Nervous System Effects. Three studies[4,12,15] discussed an association between maternal use of cocaine and nervous system (particularly central nervous system) damage.

Placenta Previa. One study[16] established that women who used cocaine were 1.4 times more likely to experience placenta previa than women who did not use cocaine.

Delivery of a Premature Neonate. The delivery of a premature neonate is an effect of maternal cocaine use that has been found in a number of different studies.[7,8,12,14,15,17] One study[5] however, found no association between cocaine use and preterm delivery.

Premature Rupture of Membranes. Premature rupture of membranes was found in two studies.[12,14]

Sudden Infant Death Syndrome. Only one study suggested an association between sudden infant death syndrome (SIDS)[8] and maternal use of cocaine.

Stillbirth. Several studies[4,7,8,15,18] have suggested an association between stillbirth and maternal use of cocaine.

Maternal Vaginal Bleeding. Maternal vaginal bleeding is an effect of maternal use of cocaine identified in two studies.[7,8] This effect is relevant to the fetus/neonate because of the potential for maternal vaginal bleeding to complicate delivery.

Fetal Birthweight. Fetal birthweight as an effect of maternal cocaine use has garnered the most literature and the most controversy. Five studies[3,7,8,15,18] can be found that suggest that maternal cocaine use is associated with low fetal birthweight. However, four studies[5,6,19,20] can be found that refute such an association. It is significant to note that two[19,20] of the four studies refuting the association of maternal cocaine use and low birthweight were based on animal models. None of the studies finding such an association involved animal models.

Of course, a number of these effects can be explained by understanding that cocaine acts by blocking the uptake and thereby increasing the amount of catecholamines in the neuronal cleft.[8] For example, increased peripheral catecholamines lead to increased contractility of the uterus, which in turn can lead to premature labor.[8] Deleterious effects on the fetus can result indirectly through maternal effects (eg, maternal vasoconstriction resulting in decreased placental blood flow) or directly once the cocaine crosses the placenta (eg, intrauterine growth retardation because of fetal vasoconstriction).[8]

The third broad conclusion in reviewing the literature on maternal cocaine use is that with rare exceptions, there are few effects of cocaine use on the mother that are unique to the status of pregnancy. To be sure, maternal use of cocaine has consequences for the pregnancy. However, those maternal effects (eg, uterine contraction) have their greatest implication for the well-being of the fetus (eg, prematurity from contractions).

Many authors have found that there are no maternal effects of maternal cocaine use that are unique to pregnancy.[4,20,21] However, one author has suggested that maternal use of cocaine can give rise to a temporary "preeclampsia-like" syndrome (ie, hypertension, edema, and proteinuria) as well as a high mortality cocaine bronchiolitis in the mother.[8] Parenthetically, it should be clear that pregnancy

does not insulate the mother from the regular complications of cocaine use (eg, tachycardia,[10] seizures, and arrhythmia).[8]

The fourth broad conclusion is that authors who study maternal cocaine use do not appear to have reported an association between maternal cocaine use and the occurrence of abdominal pregnancy. However, one case can be found in which a cocaine-addicted mother experienced a uterine rupture and a live baby was delivered from the abdominal cavity.[22]

The fifth broad conclusion is that maternal cocaine use is a numerically significant problem,[5,8] particularly within the inner-city population (eg, 31% of patients sampled in one population[23]).

Abdominal Pregnancy Risk Factors

A review of the literature on the risk factors for abdominal pregnancy reveals that there are five risk factors that have been identified or can be identified from the writings of five different authors. These five risk factors are maternal age, gestational age, race, gravidity/infertility, and socioeconomic background. These five risk factors are presented in Table 1. For ease of presentation, study A refers to a study by Atrash et al[24] with a database of 11 maternal mortalities secondary to abdominal pregnancy as reported by the Centers for Disease Control from 1979 to 1982. Study B refers to a study by Delke et al[25] with a database of 10 cases of abdominal pregnancy at the Brookdale (New York) Hospital Medical Center from 1965 to 1981. Study C refers to a study by Clark and Jones[26] with a database of 35 cases of abdominal pregnancy at Freedmen's Hospital (Washington, DC) and its successor, Howard University Hospital, from 1946 to 1975. Study D refers to a study by Beacham et al[27] with a database of 65 cases of abdominal pregnancy at Charity Hospital (New Orleans) from 1937 to 1961. Study E refers to a study by Strafford and Ragan[28] with a database of 11 cases of abdominal pregnancy at Indiana University Hospital from 1935 to 1972.

Maternal Age Risk Factor. It is relatively clear from the data in Table 1 that older mothers are at greater risk of having abdominal pregnancies. The maternal age column in Table 1 is the arithmetic mean age of the subjects in each study. The unweighted arithmetic mean of the column is 31.2 years of age.

Gestational Age Risk Factor. It appears that either the occurrence or the detection of abdominal pregnancy is on average a third-trimester event. The gestational age column in Table 1 is the arithmetic mean gestational age for the subjects in each study. The unweighted arithmetic mean of that column is 27 weeks. Study D was excluded from that computation because mean gestational age could not be computed for its subjects.

Race Risk Factor. The race risk factor appears to suggest that abdominal pregnancies are the province of nonwhite and more particularly, black women. This conclusion is problematic for at least two reasons. The first reason involves the intellectual difficulty of defining what constitutes a race or determining who should or should not be included within the definition of a race. Indeed, Audain[29] has expressed allegiance to the theory of nonrace within cultural anthropology, which maintains that races are arbitrary social fictions (eg, Why skin color? Why not size of ear lobe?). This difficulty is highlighted in Study D. It is difficult if not impossible to know how the authors in that study classified the Creole subjects within their study. The second problem with suggesting that race is a risk factor is that it is not possible to know what dimensions of the individual (eg, socioeconomic status or lack of prenatal care)[30] are being masked by a label of race.

Gravidity/Infertility Risk Factor. On the basis of the data cited in Table 1, it is difficult to support a contention that low gravidity is a risk factor for abdominal pregnancy. The gravidity column reflects the arithmetic average gravidity for the subjects in each study. The unweighted arithmetic mean of that column is 2.5.

Perhaps a better case can be made for the secondary infertility of patients who experience abdominal pregnancy. Indeed, 80% of the subjects in Study B had secondary infertility ranging from 3 to 21 years in duration. The fertility status of the subjects in Studies C and D is not known.

Socioeconomic Risk Factor. The final risk factor, low socioeconomic status, was reported only by Study B. More research on this issue is needed.

Of course, more research also is needed on the issue of whether there are any maternal behavioral variables that are associated with the occurrence of abdominal pregnancy. Our experience suggests that maternal use of cocaine is one such variable. It is to a discussion of this variable that we now turn.

MATERIALS AND METHODS

Fifty-five cases of secondary abdominal pregnan-

Table 1. Summary of Abdominal Pregnancy Risk Factors

Risk Factor	Study*	Results (Mean or %)	Comments
Maternal age	A	30.6 years	Range: 19 to 41 years
	B	32.4 years	Range: 29 to 37 years
	C	29.6 years	Range: 19 to 42 years
	D	32 years	Range: 17 to 66 years
	E	31 years	Range: 24 to 37 years
Gestational age	A	20 weeks	Range: 8 to 38 weeks
	B	28.1 weeks	Range: 16 to 43 weeks
	C	26.4 weeks	Range: 12 to 40 weeks
	D	>8 weeks	Range: 8 to 12 weeks
	E	33.4 weeks	Range: 10 to 40 weeks
Race	A	7/11 (B=63.6%)	
		4/11 (W=36.4%)	
	B	8/10 (B=80%)	
		2/10 (P=20%)	
	C	35/35 (B=100%)	
	D	62/65 (B=95.4%)†	
		3/65 (W=4.6%)	
	E	5/11 (B=45.5%)	
		6/11 (W=54.5%)	
Gravidity	B	2.1	Range: 0 to 4
	C	2.5	Range: 1 to 8
	D	3.0	Range: 1 to 9
Low socioeconomic status	B	100% subjects	

Abbreviations: B=black, W=white, and P=Puerto Rican.
*Study A refers to a study by Atrash et al[24]; study B refers to a study by Delke et al[25]; study C refers to a study by Clark and Jones[26]; study D refers to a study by Beacham et al[27]; and study E refers to a study by Strafford and Ragan.[28] Refer to the text for more information regarding these studies.
†"Creole" in blacks unknown.

cy seen from 1947 to 1994 at Freedmen's Hospital and its successor, Howard University Hospital, were analyzed. Thirty-five of the 55 cases have been presented or discussed in prior publications by one of the authors (J.F.J.C.).[26,30-36]

Drug testing of patients for cocaine by urine drug screen was instituted at the Howard University Hospital in 1986. Other means for testing for the presence of cocaine were available prior to that time. In our study, a given patient was tested based on the physician's diagnosis and index of suspicion of maternal use of cocaine. Prior to 1986, the most frequently observed drug among maternity patients was heroin. These data spanning 47 years were analyzed by chi-squared testing, trend analysis, and qualitative analysis.

RESULTS

The overall results from the analysis of 55 cases of abdominal pregnancy at Freedmen's/Howard University Hospital from 1947 to 1994 are presented in Table 2. The incidence of cases of abdominal pregnancy at Freedmen's/Howard University Hospital peaked at 17 cases in the period from 1947 to 1956 while reaching a minimum number of 5 cases during the period from 1977 to 1986. Table 2 also indicates that the placenta was left in situ (in the peritoneal cavity) in a small number of cases. Similarly, only a small number of operations resulted in live births.

Significantly, cocaine use was not found among abdominal pregnancy patients until 1987 to 1994 when the incidence of such use was tabulated in five of the six cases of abdominal pregnancy. Also significant is the fact that no maternal deaths occurred until the single case in the 1987 to 1994 period. No fetuses survived the operation in the 1987 to 1994 period while a small number survived prior to that

Table 2. Abdominal Pregnancies at Freedmen's/Howard University Hospital From 1947 to 1994*

	1947 to 1956	1957 to 1966	1967 to 1976	1977 to 1986	1987 to 1994
Cases	17	13	14	5	6
Placenta situ	2	2	0	1	3
Live births	3	3	3	1	0
Cocaine use	0	0	0	0	5
Maternal deaths	0	0	0	0	1
Survival	1	2	2	1	0
Alive	1	2	2	1	0

*Total no. cases=55, total no. live births=10, and total no. remained alive=6.

time period. Of the fetuses who survived the operation, all of them remained alive beyond the neonatal period.

A quantitative analysis of the data in Table 2 yields the following conclusions. First, a chi-squared test on the significance of cocaine to the occurrence of abdominal pregnancies can be conducted by comparing the period before 1986 to the 1986 to 1994 period. If cocaine use was not a significant variable, one would expect the frequency of cocaine use after 1986 to equal zero. This expectation of zero cocaine use after 1986 as well as before 1986 therefore becomes the null hypothesis. The alternative hypothesis is that cocaine use became significant to the occurrence of abdominal pregnancies after 1986 and therefore the use of cocaine after 1986 is nonzero. An application of the chi-squared formula[37] to this situation yields a chi-square of infinite value (ie, because zero is in the formulas' denominator). The null hypothesis therefore can be rejected at an alpha level of .01. Accordingly, after 1986, maternal cocaine use was significant to the occurrence of abdominal pregnancy at Howard University Hospital.

A second but similar conclusion regarding the importance of maternal cocaine use is possible if one considers the trend of abdominal pregnancies since 1947. That is, looking at Table 2, the general trend from 1947 to 1987 is a trend of decline in the incidence of abdominal pregnancies, with a total rate of decline of 70% (ie, [17 cases−5 cases]/17 cases). In contrast to this total rate of decline, the period during which maternal cocaine use was observed, 1986 to 1994, experienced a 20% increase in the incidence of abdominal pregnancies over the prior period (ie, [6 cases−5 cases]/5 cases).

This conclusion of increasing abdominal pregnancies is further buttressed when one considers the trend in the number of deliveries at Howard University Hospital during the time period under analysis. Specifically, when adjusted for number of deliveries, the incidence of abdominal pregnancies decreased from a rate of approximately 1 per 1765 deliveries in the late 1940s and 1950s to a rate of 1 per 5000 deliveries in the late 1970s and early 1980s. However, during the period from 1987 to 1994, the rate of abdominal pregnancies at Howard University Hospital increased to approximately 1 per 1333 deliveries.

A qualitative analysis of the data presented in Table 2 also is possible. Note that the only maternal death of 55 cases of abdominal pregnancy treated over the course of 47 years was recorded during the period of maternal use of cocaine. That particular patient presented with an abdominal pregnancy that was <20 weeks in gestation. She was admitted to the emergency room in shock after being found outside of a crack house. She eventually died in desseminated intravascular coagulation (DIC). It also can be noted from Table 2 that in contrast to the periods prior to 1987 in which some abdominal pregnancies resulted in live births, there were no live births from 1987 to 1994, which is the period under investigation.

DISCUSSION

On the basis of the preceding data, the central claim or thesis of this article is that we have identified a new risk factor for abdominal pregnancy: maternal cocaine use. We propose that this risk factor be added to the preexisting list of risk factors presented in Table 1. A cocaine use risk factor is unique in the sense that it may be more preventable than the other risk factors detailed in Table 1. Because cocaine is a chemical substance, understanding its role in the pathogenesis of abdominal pregnancies

also may get us closer to postulating a biochemical mechanism for the etiology of abdominal pregnancies. Such a discussion is far beyond the scope of this article.

A cocaine use risk factor has enormous social relevance because it appears that the use of cocaine is a societal ill that will be with us for some time to come. Indeed, the use of crack cocaine, particularly by pregnant women in the inner city, raises the prospect of subjecting some of the neediest members of our community to abdominal pregnancy: "one of the most dreadful calamities to which women can be subjected."[38]

Our central claim has been discussed in the context of a fortuitous database–a 47-year series of abdominal pregnancies. That series was divided into "before cocaine" and "after cocaine" time periods. After adjusting for the declining number of deliveries at Howard University Hospital over the past 50 years, we have been able to show an absolute as well as per delivery increase in the number of abdominal pregnancies during the "after cocaine" time period. These data also have shown an increase in maternal/fetal mortality during the "after cocaine" time period.

This database is statistically fortuitous because it is relatively clear that the *ceteris paribus* or "all other things constant" assumption has been met. That is, all of the other risk factors (eg, nonwhite status and socioeconomic status) have remained relatively constant in this population, allowing us to single out maternal use of cocaine as a significant variable. Stated differently, if the potentially confounding variables (eg, other risk factors) have remained constant over time and the main variable that has changed is maternal use of cocaine, then the quantitative and qualitative changes in abdominal pregnancies that have been observed cannot be explained by the confounding variables.

But what can be said about the management of the cocaine-using woman with an abdominal pregnancy? One of the themes that we pursued in this article is that questions such as these involve an overlap of two relevant bodies of literature–an overlap about which little has been written.

On the one hand, the literature on the cocaine-using mother discussed above, features some excellent pieces on the management of the cocaine abusing mother. Specifically, detection (eg, urine drug screen) is the key to early intervention. It is especially important for a physician to be familiar with the signs and symptoms of cocaine use (eg, dilated pupils; increased heart rate, blood pressure, respirations, and reflexes; agitation, arrhythmias, and seizures) and withdrawal (eg, "muscle aches, abdominal pain, hunger, sleepiness, and depression.")[8] The authorities agree that comprehensive care including prenatal care, drug treatment, and psychiatric care early in the first trimester are the most effective forms of intervention.[3,39-43]

On the other hand, a number of articles can be found that discuss the management of the patient with an abdominal pregnancy. The authorities agree that although the diagnosis is difficult, what is required is a good history, physical, high index of suspicion, and early intervention.[2,35] Although some authors disagree, the intervention recommended by most authors remains immediate laparotomy upon confirmation of the abdominal pregnancy.[35]

In the absence of literature that overlaps the literature on abdominal pregnancy and maternal use of cocaine, we turn to our own experience in this regard. The critical lesson we have learned in managing the cocaine-using woman with an abdominal pregnancy is this: these patients often present as acute gynecologic emergencies that overshadow the usual signs and symptoms of abdominal pregnancy. This unhappy circumstance unfortunately tends to result in a delay of the development of an orderly approach on how to diagnose and manage their condition. In sum, the already difficult task of diagnosing an abdominal pregnancy is made all the more difficult when the patient presents as a cocaine user who has an abdominal pregnancy.

CONCLUSION

The cocaine-using patient who presents with an abdominal pregnancy may not be a medical accident at all. Maternal use of cocaine along with the other risk factors of abdominal pregnancy should raise within the physician an index of suspicion as to the possibility of an abdominal pregnancy. More research is needed to determine the relationship between cocaine use and abdominal pregnancy.

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