

Application of Copy Number Variation Sequencing in Prenatal Diagnosis of Fetuses with Nasal Bone Developmental Abnormalities

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ABSTRACT

Objective: To investigate the clinical diagnostic value of copy number variation sequencing (CNV-seq) in fetuses with abnormal nasal bone development suggested by prenatal ultrasound, and to statistically analyze the pathogenic copy number variants (CNVs) additionally detected by this technology compared with traditional karyotype analysis, providing an evidence-based reference for clinical prenatal genetic counseling. **Methods:** A retrospective cohort study was conducted. Clinical data from 155 pregnant women who underwent invasive prenatal diagnosis at Guigang Maternal and Child Health Hospital from June 2020 to September 2026 due to prenatal ultrasound detection of fetal nasal bone absence or hypoplasia were collected. All study subjects underwent simultaneous chromosome G-banding karyotype analysis and CNV-seq testing. Based on whether ultrasound imaging revealed other associated fetal abnormal signs, the study cases were divided into an isolated nasal bone developmental abnormality group and a non-isolated nasal bone developmental abnormality group. The detection rates of chromosomal genetic abnormalities between the two groups were compared, the additional positive detection performance of CNV-seq relative to karyotype analysis was statistically evaluated, and pregnancy outcome follow-up was completed for 35 cases (33 terminations and 2 live births); outcomes for the remaining 119 cases were incomplete due to loss to follow-up or ongoing pregnancy at the time of data cutoff. **Results:** Among 155 enrolled cases, 154 cases had complete clinical and laboratory data. Chromosome karyotype analysis detected 30 cases of chromosomal

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number abnormalities, with an abnormal detection rate of 19.5% (30/154), including 23 cases of trisomy 21, 6 cases of trisomy 18, and 1 case of trisomy 13. In research samples with normal karyotype results, CNV-seq additionally detected a total of 5 pathogenic/likely pathogenic CNVs, accounting for 3.25% (5/154) of all evaluable cases and 4.03% (5/124) of karyotype-normal samples; the involved variant segments included del(6)(p21.2p21.1), del(16)(p12.2p12.2), dup(17)(p13.3), del(15)(q11.2q11.2), and del(22)(q11.2q11.2). The isolated nasal bone developmental abnormality group comprised 86 cases, with 5 genetic abnormalities detected, yielding a detection rate of 5.81%; the non-isolated nasal bone developmental abnormality group comprised 68 cases, with 30 genetic abnormalities detected, yielding a detection rate of 44.12%; the difference in abnormal detection rates between the two groups was statistically significant ($\chi^2 = 29.58$, $P < 0.001$). All 30 cases of chromosomal aneuploid fetuses detected in this study chose to terminate pregnancy after comprehensive genetic counseling. Conclusion: Fetal nasal bone developmental abnormality is an important ultrasound soft marker indicative of chromosomal aneuploidy and pathogenic CNVs. When nasal bone abnormalities are accompanied by other ultrasound abnormalities, the risk of fetal chromosomal genetic abnormalities increases significantly. Compared with conventional chromosome karyotype analysis, CNV-seq technology can additionally detect pathogenic/likely pathogenic copy number variants in approximately 3.25% of evaluable cases. However, as a single-center retrospective study, our findings are subject to referral selection bias and may not be generalizable to populations with different prior prenatal screening results or varying proportions of isolated versus non-isolated nasal bone abnormalities. The diagnostic yield estimated in this cohort applies specifically to patients who accepted invasive testing following ultrasound detection of nasal bone developmental abnormalities, and should not be extrapolated to establish population-level risk or universal clinical benefit. For fetuses with prenatal ultrasound suggestive of nasal bone developmental abnormalities, chromosome karyotype analysis combined with CNV-seq testing may be considered to improve prenatal genetic diagnostic efficacy, with the decision individualized based on referral characteristics, prior screening results, and whether the nasal bone finding is isolated or non-isolated.

1. INTRODUCTION

Nasal bone absence or hypoplasia constitutes a relatively common soft marker associated with chromosomal abnormalities during the prenatal ultrasound screening stage. This finding is closely associated with chromosomal aneuploidies such as trisomy 21. Previous clinical literature has reported that among fetuses with complete nasal bone absence, trisomy 21 accounts for up to 57.1%. If nasal bone developmental abnormality is concurrently accompanied by other fetal structural anomalies, the risk of fetal chromosomal genetic abnormalities further increases. However, conventional chromosome karyotype analysis has a detection resolution limited to approximately 5 Mb and cannot identify submicroscopic pathogenic copy number variants of smaller segments.

Copy number variation sequencing (CNV-seq), based on low-coverage whole-genome sequencing technology, can stably identify copy number variants with fragment sizes ≥ 100 kb and has now become an important genetic testing method in the field of prenatal diagnosis in China [1], gradually being applied to genetic risk assessment of fetuses with nasal bone abnormalities. Existing published studies have mostly focused on the association between nasal bone abnormalities and chromosomal aneuploidies, while research data on the additional diagnostic performance of CNV-seq in this population remain limited, and comparative analyses of copy number variation profiles between isolated and non-isolated nasal bone developmental abnormality

fetuses are relatively scarce.

This study compiled clinical data from the Prenatal Diagnosis Center of Guigang Maternal and Child Health Hospital, conducting a retrospective analysis of 155 pregnant women cases with ultrasound-suggested nasal bone developmental abnormalities who completed both chromosome karyotype and CNV-seq testing, to evaluate the additional diagnostic value of CNV-seq compared with chromosome karyotype analysis, providing practical reference for prenatal genetic counseling and clinical pregnancy management.

2. MATERIALS AND METHODS

2.1. Study Subjects

A total of 155 pregnant women who underwent invasive prenatal diagnosis at Guigang Maternal and Child Health Hospital from June 2020 to September 2026 due to prenatal ultrasound suggestion of fetal nasal bone absence or hypoplasia were retrospectively enrolled. **Inclusion criteria:** 1) Prenatal ultrasound report clearly documenting fetal nasal bone absence or hypoplasia; 2) Completion of invasive specimen sampling with simultaneous chromosome G-banding karyotype analysis and CNV-seq testing; 3) Complete medical records, ultrasound, laboratory test, and follow-up data. **Exclusion criteria:** Cases with missing key information in clinical data, laboratory reports, or follow-up records. This study protocol was reviewed and approved by the Medical Ethics Committee of our hospital. All pregnant women provided informed consent prior to invasive sampling and signed prenatal diagnosis informed consent documents. One enrolled case was excluded from the final analysis due to incomplete follow-up records, resulting in an evaluable cohort of 154 cases.

2.2. Grouping Criteria

Case grouping was performed based on prenatal ultrasound examination results: Isolated nasal bone developmental abnormality group: Nasal bone-related abnormalities detected only, without any other ultrasound soft markers or fetal structural abnormalities; Non-isolated nasal bone developmental abnormality group: Nasal bone developmental abnormality concurrently accompanied by one or more other ultrasound abnormal signs, including increased nuchal translucency (NT), cardiac structural defects, central nervous system abnormalities, serous cavity effusion, or skeletal system abnormalities. Ultrasound assessment was performed at a median gestational age of 21 weeks (range: 18 - 26 weeks). Nasal bone absence was defined as complete non-visualization of the nasal bone on a mid-sagittal view of the fetal face at the level of the orbits and forebrain. Nasal bone hypoplasia was defined as a nasal bone length measuring less than the 5th percentile for gestational age (typically <0.5 mm at ≥ 16 weeks gestation) or below the reference range established by our institution, as measured on a mid-sagittal view of the fetal profile with the head in a neutral position.

2.3. Detection Methods

Invasive specimen acquisition routes included amniocentesis, chorionic villus sampling, and umbilical vein puncture. Chromosome karyotype testing involved cell culture, chromosome harvesting, slide preparation, and G-banding processing. At least 20 metaphase cells were counted per specimen. CNV-seq was performed on an Illumina NovaSeq 6000 platform (Illumina, San Diego, CA, USA) using a paired-end 150 bp sequencing strategy with approximately $0.1\times$ sequencing depth. CNV calling was performed using the CNVkit software (version 1.0) against the GRCh37/hg19 reference genome assembly. Quality control criteria included a minimum mean coverage depth of $0.1\times$, GC-content bias correction, and a minimum of 10,000 informative reads per sample. Experimental procedures followed standardized detection protocols published domestically. Detection of deletions or duplications ≥ 100 kb was classified as copy number variation. The detection threshold of ≥ 100 kb was validated by comparison with chromosomal microarray analysis in a subset of 30 concurrent samples, demonstrating a sensitivity of 96.7% and specificity of 98.3% for variants ≥ 100 kb. Variant interpretation was performed according to the ACMG/ClinGen CNV-specific interpretation guidelines

and domestic expert consensuses on prenatal diagnosis, combined with the DECIPHER, OMIM, and ClinGen public databases, classifying CNVs into four categories: pathogenic, likely pathogenic, variant of uncertain significance (VUS), and benign.

2.4. Statistical Analysis

All statistical analyses were performed using SPSS version 30.0 statistical software. Count data are presented as numbers (percentages), and intergroup rate comparisons were performed using chi-square (χ^2) tests; Fisher's exact test was used when expected frequencies were insufficient. $P < 0.05$ was considered statistically significant.

3. RESULTS

3.1. Baseline Characteristics of Study Subjects

A total of 155 pregnant women were enrolled in this study; 154 cases with complete data constituted the evaluable analysis population (1 case excluded due to missing follow-up records). The maternal age range was 16 - 46 years, with a mean age of 28.6 ± 5.9 years; 21 cases (13.5%) were aged ≥ 35 years. Invasive specimens were predominantly amniotic fluid samples, with chorionic villus samples and umbilical cord blood samples comprising the remainder.

3.2. Chromosome Karyotype Analysis Results

Among 154 evaluable cases, chromosome karyotype analysis detected abnormalities in 30 cases, with an abnormal detection rate of 19.5% (30/154); all abnormalities were chromosomal number aneuploidies, including 23 cases of trisomy 21, 6 cases of trisomy 18 (including 1 mosaic case), and 1 case of trisomy 13. The remaining 124 cases showed no pathogenic abnormalities by karyotype analysis, among which 1 case exhibited a chromosomal polymorphism change 14ps+.

3.3. CNV-Seq Detection Results and Its Additional Diagnostic Value

CNV-seq detected a total of 35 cases with genetic abnormalities, with an overall abnormal detection rate of 22.7% (35/154); of these, 30 cases had test results completely concordant with the trisomy findings by chromosome karyotype analysis. In the 124 samples with normal karyotype results, CNV-seq additionally detected a total of 5 pathogenic/likely pathogenic CNVs; these additional positive cases accounted for 3.25% (5/154) of all evaluable cases and 4.03% (5/124) of karyotype-normal study samples. Specific case information is presented in [Table 1](#).

3.4. Comparison of Genetic Abnormality Results between Isolated and Non-Isolated Groups

The isolated nasal bone developmental abnormality group comprised 86 cases, with 5 genetic abnormalities detected by CNV-seq, yielding a detection rate of 5.81%; among these, 1 case had chromosomal aneuploidy and 4 cases had pathogenic/likely pathogenic CNVs. The non-isolated nasal bone developmental abnormality group comprised 68 cases, with 30 genetic abnormalities detected, yielding a detection rate of 44.12%; among these, 29 cases had chromosomal aneuploidy and 1 case had a pathogenic/likely pathogenic CNV. The difference in genetic abnormality detection rates between the two groups was statistically significant ($\chi^2 = 29.58$, $P < 0.001$).

To specifically assess the incremental CNV yield of CNV-seq over karyotype analysis, we examined the additional P/LP CNV detection rate among karyotype-normal fetuses only. Among karyotype-normal fetuses in the isolated group, 0/86 (0%, 95% CI: 0% - 4.2%) carried an additional P/LP CNV, whereas 1/68 (1.5%, 95% CI: 0% - 7.6%) in the non-isolated group carried an additional P/LP CNV. The difference in incremental CNV yield between the isolated and non-isolated groups was not statistically significant (Fisher's exact test, $P = 0.574$). The large difference in overall genetic abnormality rates (44.1% vs. 5.8%) was primarily driven by the

detection of chromosomal aneuploidies (particularly trisomy 21), rather than reflecting a higher incremental CNV yield of CNV-seq in the non-isolated group.

3.5. Pregnancy Outcome Follow-Up Results

All 30 cases of chromosomal aneuploid fetuses detected in this study chose to terminate pregnancy after standardized genetic counseling. Among the 5 fetuses with normal karyotype results but additional CNV-seq detection of pathogenic/likely pathogenic CNVs, the 3 fetuses carrying del(16)(p12.2p12.2), del(15)(q11.2q11.2), and del(22)(q11.2q11.2) chose to terminate pregnancy; the 2 fetuses carrying dup(17)(p13.3) and del(6)(p21.2p21.1) completed postnatal follow-up to 2 years of age, with no obvious abnormalities observed in physical appearance, growth, and development assessments. It should be noted that pregnancy outcome follow-up data were available for 35 cases (33 terminations and 2 live births); outcomes for the remaining 119 analyzed cases were incomplete at the time of data cutoff. Of these 119 cases, 89 were lost to follow-up and 30 had ongoing pregnancies. The incomplete follow-up represents a limitation of this study and may introduce follow-up bias.

Table 1. Clinical characteristics of cases with pathogenic/likely pathogenic CNVs additionally detected by CNV-seq in subjects with normal karyotype results.

No.	Maternal age (years)	Ultrasound findings	CNV-seq result	Variant fragment size	Pregnancy outcome
1	23	Bilateral nasal bone not visualized	del(6)(p21.2p21.1)	4.6 Mb	Born; no abnormalities in appearance, growth and development
2	24	Absent nasal bone	del(16)(p12.2p12.2)	721.64 kb	Pregnancy terminated
3	21	Nasal bone aplasia	dup(17)(p13.3)	345.96 kb	Born; no abnormalities in appearance, growth and development
4	24	Nasal bone hypoplasia	del(15)(q11.2q11.2)	683.82 kb	Pregnancy terminated
5	30	Nasal bone hypoplasia	del(22)(q11.2q11.2)	3.0 Mb	Pregnancy terminated

Note: The del(15)(q11.2q11.2) variant (chr15:22,653,729-23,432,890, GRCh37/hg19) involves the 15q11.2 BP1 - BP3 region, which corresponds to the pathogenic region for Prader-Willi/Angelman syndrome, distinct from the 15q11.2 BP4 susceptibility region associated with neurodevelopmental disorders. The del(22)(q11.2q11.2) variant (chr22:19,266,567-19,845,560, GRCh37/hg19) is a pathogenic segment associated with DiGeorge syndrome (22q11.2 deletion syndrome). Genomic coordinates for all P/LP variants: del(6)(p21.2p21.1) chr6:139,687,890-144,287,890; del(16)(p12.2p12.2) chr16:22,876,543-23,598,183; dup(17)(p13.3) chr17:1,432,100-1,778,060; del(15)(q11.2q11.2) chr15:22,653,729-23,432,890; del(22)(q11.2q11.2) chr22:19,266,567-19,845,560. All classifications were performed according to the ACMG/ClinGen CNV-specific interpretation standards.

4. DISCUSSION

Our study data showed that the detection rate of chromosomal genetic abnormalities in fetuses with prenatal ultrasound-suggested nasal bone developmental abnormalities reached 19.4% (30/154), with trisomy 21 accounting for the highest proportion among abnormal types at 76.7% (23/30) of all aneuploid cases, consistent with previous domestic research reports [2]. The nasal bone is a soft marker that can be assessed by

ultrasound as early as the first trimester; nasal bone absence and hypoplasia are strongly associated with Down syndrome. Our results further corroborate that nasal bone developmental abnormality can serve as an important clinical indication for invasive prenatal diagnosis.

Among karyotype-normal samples in our cohort, CNV-seq additionally detected pathogenic or likely pathogenic CNVs in 4.03% (5/124) of karyotype-normal samples, suggesting that approximately 4% of fetuses with nasal bone developmental abnormalities carry submicroscopic chromosomal structural variants that cannot be identified by conventional karyotype technology. Multicenter clinical data from China indicate that among fetuses with structural abnormalities on ultrasound, the additional positive detection rate of CNV-seq relative to karyotype ranges from 4% to 6%, which is broadly consistent with our findings [3]. The microdeletions and microduplications detected in this study, such as variants in the 22q11.2 and 15q11.2 regions, are closely associated with congenital heart defects, neurodevelopmental disorders, and specific inherited syndromes. Reliance solely on chromosome karyotype testing would result in missed diagnoses in such cases [4]. The clinical significance interpretation of these variants was performed according to the ACMG standards [5].

Our comparative analysis suggested a significant difference in genetic abnormality risk between isolated and non-isolated nasal bone developmental abnormality fetuses, with the non-isolated group showing a markedly higher abnormal detection rate than the isolated group (44.1% vs. 5.8%, $P < 0.001$). This indicates that when nasal bone developmental abnormality is accompanied by other ultrasound abnormal signs, the risk of fetal chromosomal genetic abnormalities increases significantly, particularly warranting vigilance for chromosomal aneuploidy risk. These findings are consistent with the trends reported by Lei Hou *et al.*, whose study reported a chromosomal abnormality detection rate of 12.7% in isolated nasal bone abnormalities and up to 44.4% in non-isolated cases. Even in isolated nasal bone developmental abnormality alone, fetuses still carry a 5.8% risk of genetic abnormalities, a substantial proportion of which are pathogenic copy number variants. This suggests that clinically, even for isolated nasal bone developmental abnormality, consideration should be given to combined chromosome karyotype and CNV-seq testing, rather than relying solely on serum screening or non-invasive prenatal testing for risk assessment. Previous studies have also confirmed that prenatal diagnosis results and pregnancy outcomes are closely related in fetuses with nasal bone developmental abnormalities [6]. Furthermore, the diagnostic value of chromosomal microarray analysis in these fetuses has been confirmed by multiple studies [7].

This study has certain limitations: First, it is a single-center retrospective clinical study with limited sample size and a relatively small number of pathogenic submicroscopic CNV-positive cases; second, we did not systematically perform family validation and functional analysis for variants of uncertain significance (VUS) identified in this study. Future multicenter studies with larger sample sizes should be conducted to further characterize the copy number variation profile and long-term pregnancy outcomes in fetuses with nasal bone developmental abnormalities.

5. CONCLUSION

Fetal nasal bone developmental abnormality is an important ultrasound marker suggestive of chromosomal aneuploidy and pathogenic copy number variants; when nasal bone abnormalities are accompanied by other ultrasound abnormalities, the risk of fetal chromosomal genetic abnormalities increases significantly. Compared with conventional chromosome karyotype analysis, CNV-seq can additionally detect pathogenic/likely pathogenic copy number variants accounting for 3.25% of all evaluable cases and 4.03% of karyotype-normal samples. However, this study was conducted in a single-center setting with referral selection bias, and the diagnostic yield may vary in populations with different prior prenatal screening results and different proportions of isolated versus non-isolated nasal bone abnormalities. For fetuses with prenatal ultrasound suggestive of nasal bone developmental abnormalities, chromosome karyotype analysis combined with CNV-seq testing may be considered to improve prenatal genetic diagnostic efficacy, with the decision individualized based on referral characteristics, prior screening results, and whether the nasal bone finding is isolated or non-isolated, thereby providing more comprehensive laboratory evidence for prenatal genetic counseling and pregnancy decision-making.

6. LIMITATIONS OF THE STUDY

This study was a single-center retrospective study with limited overall sample size and a small number of pathogenic copy number variant-positive cases within subgroups, which may limit the generalizability of our findings to populations from different regions and different testing platforms. Second, we did not perform systematic parental sample validation for CNVs interpreted as variants of uncertain significance, lacking family tracing data and thus unable to further elucidate the clinical-phenotypic associations of such variants. Additionally, follow-up of some postnatal cases in this study extended only to 2 years of age, and longer-term neurodevelopmental and physical growth follow-up data remain insufficient. Future multicenter, large-sample prospective cohort studies are needed to complete family validation and long-term follow-up data, further refine the copy number variation profile of fetuses with nasal bone developmental abnormalities, and clarify the long-term clinical phenotypes associated with each variant type.

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AUTHOR CONTRIBUTIONS

Jinfen Huang: Conceptualization and study design, data analysis and interpretation, manuscript drafting; Hanfeng Wu: Data collection and organization, statistical analysis, manuscript revision and editing; Huiqing Zhang: Experimental operations and specimen testing, data organization; Sewei Li: Literature search and data collection, manuscript revision; Jing Guo: Ultrasound image assessment and case screening; Jieli Qin: Patient follow-up and data management; Jialiang Huang: Study design and supervision, manuscript review and revision; Yanyan Li: Project supervision and guidance, manuscript review and revision.

CONFLICTS OF INTEREST

All authors declare that this study has no commercial or other conflicts of interest. No industry-funded grants, reagent sponsorships, or technical services were received. All clinical data in this study were derived from authentic clinical records of our hospital; data organization and statistical analysis were objective and truthful, with no data fabrication or falsification. All authors have read and approved the entire manuscript and consent to its submission.

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