دائـــرة الـصـحـة
DEPARTMENT OF HEALTH

GUIDELINES FOR CLINICAL MICROARRAY TESTING

● PUBLIC / عـــــام

Document Title:	Guidelines For Clinical Microarray Testing		
Document Ref. Number:	DoH/GD/HLS/GCMT/V1/2025	Version:	V 1
New / Revised:	New		
Publication Date:	December 2025		
Effective Date:	Jan 2026		
Document Control:	DoH Strategy Sector		
Applies To:	- DoH licensed Healthcare Providers - DoH licensed Healthcare Facilities		
Owner:	Health Life Science Sector		
Revision Date:	December 2026		
Revision Period:	1 year		
Contact:	Health Life Science Sector – hlss@doh.gov.ae		

1.Guideline Purpose and Brief

This guideline outlines best practices for microarray testing in clinical settings. It incorporates internationally recognized standards from organizations such as the American College of Medical Genetics and Genomics (ACMG), the American College of Obstetricians and Gynecologists (ACOG), and Clinical Genome Resource (ClinGen).

It provides healthcare professionals and clinical laboratory facilities with a clear and practical framework for the clinical use of microarray (also known as chromosomal microarray analysis CMA) in the detection of copy-number variants (CNVs) across the genome.

This guideline outlines the key technical, operational, and clinical considerations to support the use of microarray testing. It is intended to guide healthcare facilities in applying microarray technology in a standardized, evidence-informed manner, consistent with UAE Federal Law and Department of Health (DoH) regulations, including considerations from Health Technology Assessment (HTA). The overall aim is to promote consistent, accurate, and high-quality CNV analysis and reporting across relevant clinical settings.

2. Definitions and Abbreviations

No.	Term / Abbreviation	Definition
2.1	aCGH	Array Comparative Genomic Hybridization – a microarray method that detects DNA copy-number changes across the genome.
2.2	Balanced Chromosomal Rearrangement	Structural change in chromosomes where no DNA is gained or lost (e.g., balanced translocations, inversions).
2.3	BAC array	Bacterial Artificial Chromosome array
2.4	CN-LOH	Copy-Neutral Loss of Heterozygosity – a region where both chromosome copies come from one parent without a gain or loss of DNA.
2.5	CNV	Copy-Number Variant – a segment of DNA that is duplicated or deleted compared to the reference genome.
2.6	Dosage-sensitive region	A part of the genome were having an extra copy (duplication) or missing copy (deletion) is likely to cause disease.
2.7	DoH	Department of Health.
2.8	FISH	Fluorescence In Situ Hybridization – uses fluorescent probes to detect specific DNA sequences on chromosomes.
2.9	G-banded karyotyping	A chromosome analysis method in which stained chromosomes are examined under a microscope to detect large structural changes or abnormal chromosome numbers.
2.10	Germline	Genetic variants present from birth in every cell of the body; may be inherited or occur early in development and can be passed to offspring.
2.11	Indels	Insertions or deletions of bases in the genome, typically involving a small number of nucleotides, which may cause frameshift mutations and affect gene function.
2.12	ISCN	International System for Human Cytogenomic Nomenclature
2.13	MCC	Maternal Cell Contamination (MCC)
2.14	Mosaicism	The presence of two or more genetically distinct cell populations in the same individual.

2.15	Multi-Omics	Combining different biological data types (genomics, transcriptomics, proteomics, etc.) for analysis.
2.16	Ploidy	The number of complete sets of chromosomes in a cell. Normal human cells are diploid (two sets).
2.17	Proteomic profiling	The large-scale study of proteins, including their expression levels, modifications, functions, and interactions within cells or tissues.
2.18	ROH	Region of Homozygosity – identical DNA sequence inherited from both parents in a continuous stretch.
2.19	SNP	Single-Nucleotide Polymorphism – a single DNA base change common in the population.
2.20	SNV	Single-Nucleotide Variant – a single DNA base change that may be benign or disease-causing.
2.21	Somatic	Genetic variants acquired after conception that are present only in certain cells or tissues and are not inherited or passed on to offspring.

3.Guideline Content

3.1 Overview of Microarrays

3.1.1. Microarrays are a high-throughput genomic technology that enables simultaneous analysis of thousands of DNA targets across the genome. Using probes on a bead chip, it detects differences in copy-number or known variants between patient DNA and reference.

3.1.2. Microarray testing provides greater sensitivity, resolution and throughput than karyotyping and is used both diagnostically and in research.

3.1.3. There are two main clinical microarray technologies:

3.1.3.1 Chromosomal Microarray Analysis (CMA): Includes array comparative genomic hybridization (aCGH) and single nucleotide polymorphism (SNP) arrays. CMA is validated for genome-wide detection of copy-number variants (CNVs), deletions, duplications, and regions of homozygosity (ROH) or copy-neutral loss of heterozygosity (CN-LOH).

3.1.3.2 Genotyping Arrays: Primarily designed to interrogate known single nucleotide polymorphisms (SNPs) or predefined variants. While some genotyping arrays can provide CNV information, they are not validated for comprehensive diagnostic CNV analysis and are best suited for targeted variant screening.

3.1.4. Hybrid platforms exist that combine SNP genotyping with CNV detection, allowing simultaneous detection of targeted variants and structural changes. Their clinical application depends on validation and regulatory approval.

3.2 Clinical Use of Microarrays

Microarray technologies do not receive broad U.S. Food and Drug Administration (FDA) approval as platforms. Instead, the FDA reviews and authorizes each device for a specific medical use through either the 510(k) clearance or Premarket Approval (PMA) pathway. The FDA also issues general and special controls and supports quality initiatives such as the MicroArray/Sequencing Quality Control (MAQC/SEQC) Project. In Abu Dhabi all microarray-based technology requires Health Technology Assessment (HTA) approval from the DoH prior to clinical use.

CMA

3.2.1 Developmental and Intellectual Disorders

3.2.1.1 CMA is a recommended diagnostic test for individuals presenting with:

- Developmental delay (DD)
- Intellectual disability (ID), with or without dysmorphic features
- Autism spectrum disorder (ASD)
- Multiple congenital anomalies (MCA)

3.2.2 Prenatal Testing and Preimplantation Testing

3.2.2.1 CMA is a recommended diagnostic test for:

- Invasive prenatal diagnosis when structural fetal anomalies are detected via ultrasound.
- Evaluation of intrauterine fetal demise or stillbirth (where clinically indicated and based on specimen availability).
- Follow-up diagnostic testing to confirm and further characterize copy-number changes reported by noninvasive prenatal screening (NIPT), where clinically indicated.
- Microarray (aCGH and/or SNP-array) may be used for preimplantation genetic testing for aneuploidy (PGT-A) in some settings.

3.2.3 Neoplastic Disorders

3.2.3.1 CMA may support diagnosis, prognostication, and therapeutic decision-making in patients with cancer.

3.2.3.2 Used for detecting copy-number alterations in hematologic malignancies and selected solid tumors, and may be used alongside conventional cytogenetics and targeted methods such as G-banded karyotyping and fluorescence in situ hybridization (FISH).

Genotyping

3.2.4 Carrier and Population Screening

3.2.4.1 Genotyping microarrays may be used in carrier and population screening to detect predefined variants relevant to:

- Oncology
- Cardiovascular
- Inherited conditions
- Pharmacogenomics

Specialized Arrays

3.2.5 DNA methylation arrays may support integrated molecular profiling in selected contexts.

3.2.5.1 In pediatric central nervous system (CNS) tumors and other complex disorders, integration of CNV findings (where available) with methylation and/or expression-based profiling may improve diagnostic accuracy and support molecular classification, consistent with integrated histo–molecular diagnosis approaches referenced in the WHO 2021 CNS Tumor Classification.

3.3 Comparison with Other Technologies

3.3.1 Multiple cytogenetic and molecular technologies are used in clinical practice to detect chromosomal and genomic abnormalities. Each method offers unique strengths and limitations, making them complementary rather than interchangeable. CMA is recognized as the standard for genome-wide detection of CNVs, while other methods may be chosen depending on the clinical question. A detailed comparison is provided in Table 1.

Table 1. Comparison with Other Technologies

Technology	Primary Application	Strengths	Limitations	Typical Clinical Use
Chromosomal Microarray Analysis (CMA)	Genome-wide detection of CNVs, regions of homozygosity (ROH), uniparental disomy	High resolution for submicroscopic CNVs; genome-wide coverage;	Cannot detect balanced rearrangements; limited detection of low-level mosaicism	Diagnostic test for unexplained developmental disorders and congenital anomalies
Genotyping Microarrays	Targeted SNP and variant detection (e.g., disease-associated SNPs, pharmacogenomics)	Cost-effective; high-throughput; useful for population-scale screening	Limited to predefined variants; not genome-wide, Tetraploidy	Pharmacogenomics, carrier and population Screening
Next-Generation Sequencing (NGS)	Single nucleotide variants, small indels, CNVs, structural rearrangements	Base-pair resolution; broad variant spectrum; expanding clinical utility	Higher cost; longer turnaround time; requires advanced bioinformatics	Exome/genome sequencing rare diseases, cancer genomics
Conventional Karyotyping	Chromosomal abnormalities (numerical and large structural changes)	Detects balanced rearrangements (translocations, inversions); low cost	Low resolution (>5–10 Mb); labor-intensive	Pre and postnatal diagnosis of birth defects (e.g., Down's syndrome), cancer
Fluorescence in situ Hybridization (FISH)	Rapid, targeted detection of specific chromosomal abnormalities	High specificity; quick turnaround; useful for mosaicism detection	Limited to targeted regions; not genome-wide	Prenatal testing for high risk pregnancy, rapid cancer prognosis testing
Multiplex Ligation-dependent Probe Amplification (MLPA)	Gene-level CNV detection in specific syndromes	Sensitive, cost-effective; complements CMA for confirming findings	Restricted to targeted loci; not genome-wide	Confirming deletions/duplications in known genetic syndromes (e.g., Duchenne muscular dystrophy)

Advantages of Microarrays

CMA and genotyping arrays provide several important benefits in clinical practice:

3.4.1. CMA can be applied to a wide range of clinical specimens that yield DNA of sufficient quality, including peripheral blood, amniotic fluid, chorionic villi, cultured fibroblasts, saliva, buccal swabs, and formalin-fixed paraffin-embedded (FFPE) tissue (where platform validation is available).

3.4.2. CMA provides higher resolution than standard G-banded karyotyping, allowing detection of submicroscopic deletions and duplications that would otherwise be missed.

3.4.3. CMA refines abnormalities initially identified by karyotyping, offering more precise genomic characterization and context.

3.4.4. Arrays can be customized to increase probe density in clinically relevant regions, enabling focused evaluation for microdeletion/duplication syndromes or carrier screening.

3.4.5. CMA generates objective, genome-wide data for copy-number variant detection. When SNP probes are included, CMA can also identify regions of homozygosity (ROH) and copy-neutral loss of heterozygosity (CN-LOH), findings not detectable by karyotype or FISH.

3.4.6. Genotyping arrays provide cost-effective testing for predefined variants, making them suitable for large-scale carrier and population screening.

3.4.7. Hybrid arrays integrate SNP detection with CNV analysis, allowing broader genomic information to be obtained within a single assay.

3.5 Limitations of Microarrays

3.5.1 CMA cannot detect balanced chromosomal changes (e.g., balanced translocations or inversions). These may require karyotyping or FISH.

3.5.2 CMA has reduced sensitivity for low-level mosaicism or tumor-specific changes when only a small number of cells are affected. The detection threshold depends on the array design and probe density, with mosaicism typically detectable at levels of ~10–20% or higher.

3.5.3 For neoplastic (tumor) samples, CMA interpretation is limited by tumor purity and DNA quality. Low tumor cellularity, necrosis, or degraded DNA can prevent reliable copy-number calling. A matched normal control (e.g., patient blood or uninvolved tissue) is recommended to distinguish somatic CNVs from germline variants; if no matched normal is available, some CNVs may represent germline variation rather than tumor-acquired change.

3.5.4 CMA is not suitable for minimal residual disease (MRD) assessment or for monitoring therapy response. It must not be used to evaluate treatment response, persistence of disease, or clearance of disease over time.

3.5.5 CMA cannot show the exact mechanism of a chromosomal imbalance (e.g., whether it is a duplication, insertion, or marker chromosome). Additional cytogenetic or FISH testing may be needed.

3.5.6 CMA does not reliably detect polyploidy (e.g., tetraploidy). SNP-based platforms may suggest it, but confirmation is required.

3.5.7 CMA cannot detect very small DNA changes such as single nucleotide variants (SNVs), small insertions/deletions, or epigenetic modifications; therefore, a normal CMA result does not rule out all genetic conditions at the tested locus.

3.5.8 In urgent cases where a quick answer is needed, other methods (e.g., PCR or rapid sequencing) may be more appropriate.

3.5.9 Genotyping arrays are limited to a set list of predefined variants and are not reliable for diagnostic CNV detection.

3.5.10 Genotyping arrays cannot identify unexpected or novel CNVs and should not be used as a replacement for CMA in diagnosis.

3.5.11 SNP-based CMA detects regions of homozygosity (ROH) and copy-neutral loss of heterozygosity (LOH) but cannot determine the underlying mechanism of the imbalance (e.g., uniparental disomy versus identity-by-descent). This limits precise recurrence risk assessment for subsequent pregnancies.

3.6 Microarray Requirements

3.6.3 Platform Technology

3.6.1.1 Microarray testing can be performed using two main CMA platforms (Figure 1):

3.6.1.1.1 aCGH (array Comparative Genomic Hybridization): Compares patient DNA to a reference sample to detect deletions and duplications at high resolution.

3.6.1.1.2 SNP Arrays: Use probes for hundreds of thousands of SNPs across the genome. Detect CNVs, regions of homozygosity (ROH), and copy-neutral loss of heterozygosity (CN-LOH).

3.6.1.2 Genotyping arrays use SNP platforms to test predefined variants. They may produce CNV information, but are not validated for diagnostic, genome-wide CNV detection.

3.6.1.3 Platform selection should be based on clinical indication, required resolution, and validation status for the intended sample type. Each laboratory must define the minimum CNV size reported, probe density, and analytical resolution.

3.6.1.4 All platforms must use a standardized genome build (e.g., GRCh37 or GRCh38) with documented annotation sources.

3.6.1.5 CMA and genotyping arrays are reliable within their validated scope, but some results require confirmation by another method (e.g., FISH, qPCR, MLPA, targeted NGS), depending on variant type, size, or complexity (Figure 2).

3.6.1.6 Classification Standards:

3.6.1.6.1 Copy-number variants (CNVs) must be classified using the ACMG/ClinGen 2020 technical standards for CNV interpretation. CNVs shall be evaluated using the point-based scoring framework, with separate evidence frameworks for deletions and for duplications.

3.6.1.6.2 Sequence variants (if reported from genotyping arrays) must be classified using ACMG/AMP standards for sequence variant interpretation.

3.6.1.6.3 All variants must be assigned one of five categories: pathogenic (P), likely pathogenic (LP), variant of uncertain significance (VUS), likely benign (LB), or benign (B). See Table 2.

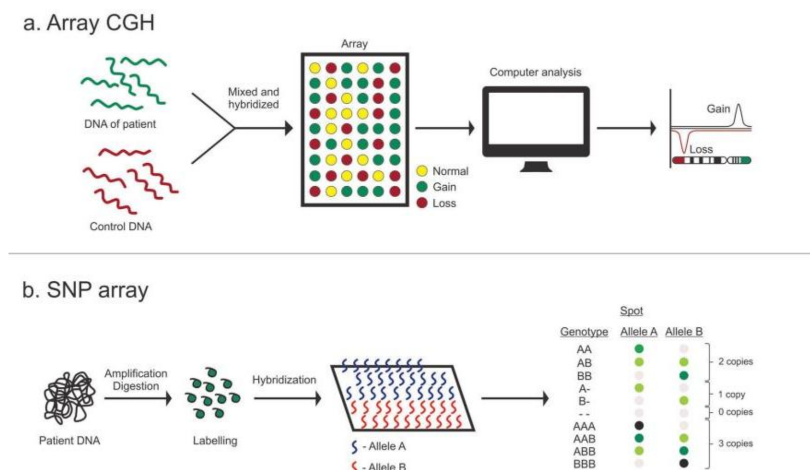


Figure 1: (a) Comparative genomic hybridization array, and (b) Single nucleotide polymorphism array

(Source: Narayanan, D. L., & Girisha, K. M. (2020). Genomic Testing for Diagnosis of Genetic Disorders in Children: Chromosomal Microarray and Next-Generation Sequencing. Indian pediatrics, 57(6), 549–554).

3.6.2 Laboratory Requirements

3.6.2.1 Laboratories must be DoH-licensed, comply with DoH standard for laboratory accreditation for genomic-related services and products, and maintain international accreditation (e.g., ISO 15189, CAP). Participation in external proficiency testing and internal audits is mandatory. [10]

3.6.2.2 Laboratories must have written procedures and a documented quality management program covering all aspects of microarray testing, including pre-analytical, analytical, and post-analytical phases.

3.6.2.3 All laboratory personnel must be licensed by DoH and perform their activities in compliance with DoH Clinical Laboratory Standards, DoH Standard for Clinical Privileging Framework, DoH Healthcare Professionals Manual and Unified Healthcare Professionals Qualification Requirements (PQR). [14,15]

3.6.2.4 Laboratories must also comply with DOH Information Security requirements regarding platform data security.

3.6.2.5 The laboratory must define objective quality control (QC) acceptance criteria as pass/fail thresholds for every run and every patient sample.

3.6.2.6 Any sample or run that does not meet the laboratory's predefined QC thresholds must be flagged as "limited," repeated, or rejected, and this limitation must be documented in the final report if results are still issued.

3.6.2.7 Each workflow must be validated with HTA approval before clinical use, covering accuracy, sensitivity, specificity, reproducibility, and mosaicism detection limits, using well-characterized control samples (e.g., Coriell reference materials or other defined positive/negative controls) with known copy-number changes.

3.6.2.8 Reports must follow international standards (e.g., ACMG, ClinGen), ensuring clear, clinically relevant interpretation for the ordering physician.

3.6.2.9 Reports must include the referral reason and clinical indication.

3.6.2.10 Prenatal CMA carries a higher rate of VUS and uncertain genotype–phenotype correlation; these limitations must be clearly communicated in reports and counseling.

3.6.2.11 Turnaround Time (TAT):
3.6.2.11.1 Postnatal CMA reports should be issued within 2–4 weeks.
3.6.2.11.2 Prenatal CMA should prioritize expedited reporting due to time-sensitive clinical decisions.

3.6.2.12 Laboratories must define policies for the retention of DNA samples and array data and should specify minimum retention periods.

3.6.3 Interpretation & Reporting

3.6.3.1 CNV interpretation must consider:

- Genomic coordinates and size of the CNV
- Platform probe coverage / backbone density
- Dosage-sensitive gene content and gene function
- Inheritance (de novo vs inherited)
- Penetrance and variable expressivity of implicated loci
- Constraint and population frequency data
- Correlation with the patient's clinical presentation / phenotype

- Mode of inheritance of the implicated gene(s)/locus (autosomal dominant, autosomal recessive, X-linked, other)

3.6.3.2 Sequence Variants (Genotyping Arrays): Interpretation is limited to predefined SNPs/variants and must consider allele frequency in appropriate ancestral populations, known clinical validity, actionability/management relevance, and published evidence.

3.6.3.3 Interpretation of all variants must reference curated databases (ClinGen, DECIPHER, DGV, ClinVar, OMIM, gnomAD-SV, gnomAD-CNVs, PubMed).[16−22] The specific gnomAD version must be documented in the patient report.

3.6.3.4 Laboratories are encouraged to submit novel or reclassified findings to public databases (e.g. ClinVar, DECIPHER) to support consistency of interpretation across the health system.

3.6.3.5 CMA may be performed using BAC arrays, oligonucleotide arrays, and/or SNP-based arrays. The critical requirement is analytical coverage and resolution, not the commercial platform.

3.6.3.6 CMA must provide uniform coverage across the genome sufficient to detect chromosomal imbalances at a resolution that exceeds a standard karyotype (~5 Mb). Typical validated analytical resolution should allow detection of imbalances ≥400 kb genome-wide, with higher-density coverage of clinically significant regions (e.g. 22q11.2).

3.6.3.7 Laboratories may include targeted increased probe density for recurrent microdeletion/microduplication syndromes or other high-yield clinically actionable loci, as defined in their validated platform.

3.6.3.9 Laboratories must have a documented policy on when carrier status for recessive and X-linked conditions is evaluated and reported.

3.6.3.9 Carrier findings must be clearly labeled as carrier status and interpreted in the context of the family history, referral indication, and local policies for cascade testing and reproductive counseling.

3.6.3.10 Parental/Family Studies (Segregation Analysis):

3.6.3.10.1 Laboratories should recommend parental testing to distinguish between inherited and de novo CNVs.

3.6.3.10.2 Segregation analysis should be clearly reported as it significantly impacts interpretation and recurrence risk.

3.6.3.11 VUS Reanalysis:

3.6.3.11.1 Laboratories must maintain a documented policy for periodic re-evaluation of VUS and other uncertain findings.

3.6.3.11.2 VUS and other uncertain CNV calls must be re-evaluated at least every 18–24 months, or sooner if new high-quality evidence emerges (e.g. new gene–disease association, updated ClinGen dosage curation, significant reclassification in ClinVar).

3.6.3.11.3 The laboratory must define clear triggers for reanalysis (e.g. updated genome build, updated pipeline, new ClinGen dosage sensitivity evidence, new penetrance data, new segregation information).

3.6.3.11.4 When a VUS is reclassified to likely pathogenic or pathogenic (or from uncertain to likely benign/benign)

and that change has clinically significant impact, an amended report must be issued. The updated interpretation must be communicated to the referring clinician and, where appropriate, genetic counselor, to enable updated patient counseling and management.

3.6.3.12 Prenatal CMA Reporting Requirements

3.6.3.12.1. MCC:

- The laboratory must assess and document MCC for all prenatal CMA specimens.
- A matched maternal sample shall be used to evaluate MCC.
- The final report must include an MCC statement (e.g. “MCC: not detected” or “MCC detected; results may reflect mixed fetal/maternal DNA”), because MCC can mask or distort fetal copy-number results.

3.6.3.13 Reporting VUS:

3.6.3.13.1 VUS shall not be routinely reported in prenatal CMA results.

3.6.3.13.2 A VUS may be reported only if all criteria below are met:

- The copy-number change is plausibly related to the specific fetal phenotype (for example, the structural anomaly detected on ultrasound); and
- Disclosure may directly inform pregnancy management, counseling, or perinatal planning; and
- The finding has been reviewed and approved through a documented multidisciplinary review that includes (at minimum) maternal–fetal medicine, clinical genetics/genetic counseling, and the laboratory director.

3.6.3.13.3 VUS that are incidental, unrelated to the fetal presentation, or not actionable for pregnancy management must not be included in the prenatal report.

3.6.3.13.4 Adult-onset, carrier status, or secondary/incidental findings with no relevance to fetal prognosis should not be reported unless this was explicitly consented to pre-test or non-disclosure would pose an immediate and serious preventable risk to the parents or fetus.

3.6.3.14 Secondary/Incidental Findings:

3.6.3.14.1 Only variants classified as pathogenic or likely pathogenic and deemed clinically actionable may be reported as secondary/incidental findings.

3.6.3.14.2 Findings unrelated to the primary indication must be clearly labeled as secondary/incidental in the report.

3.6.3.14.3 Patient consent and preferences regarding secondary findings must be documented and respected.

3.6.3.14.4 Reports should recommend referral to a clinical geneticist or genetic counselor.

3.6.3.15 All reports must use standardized nomenclature:

- HGVS for sequence-level descriptions

- ISCN / cytogenetic notation for structural and copy-number events
- Chromosomal coordinates mapped to the stated genome build

3.6.3.16 Each reported CNV must, at minimum, include:

- Cytogenetic location (chromosome number and bands).
- Genomic coordinates with CNV size, with the reference genome build explicitly specified (e.g. hg19/GRCh37 or hg38/GRCh38).
- Copy-number state (deletion or duplication; specify 0, 1, 3, or more copies when known).
- Total number of RefSeq- and/or ClinGen-curated genes within the interval.

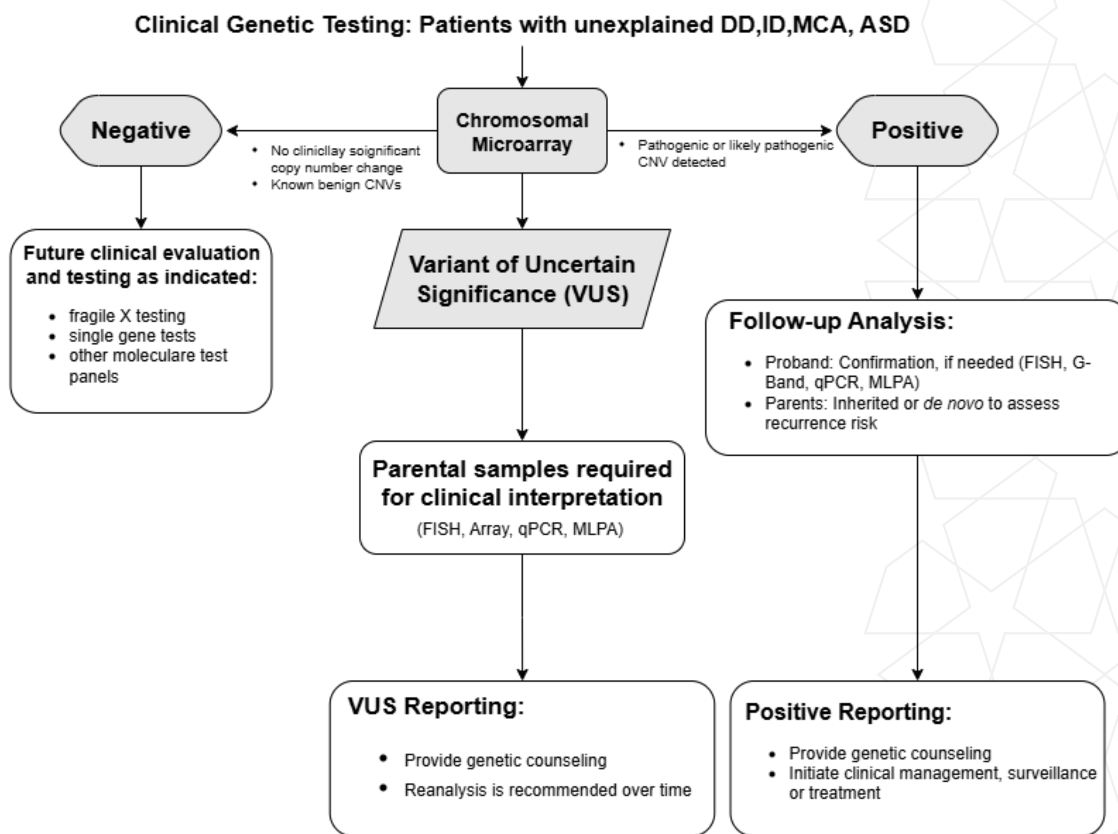
3.2.2.17 Gene content should be summarized as follows:

- For pathogenic/likely pathogenic CNVs or well-established syndromes, it is acceptable to list the syndrome name (with OMIM number when applicable) and/or the most clinically relevant dosage-sensitive gene(s).
- For VUS or regions under periodic re-evaluation, all RefSeq or ClinGen-curated genes fully or partially encompassed by the CNV should be listed when feasible, to facilitate future reanalysis and literature monitoring.
- Each report must clearly describe the limitations of the assay.

Table 2: Classification of Variants & Reporting

Type of CNV	*Criteria*	*Clinical Reporting Guidance*
Pathogenic (P)	Clear, well-established evidence that the variant is disease-causing	Always report with full details (gene(s), size, type, inheritance, associated disorder, and relevant references).
Likely Pathogenic (LP)	Strong but not conclusive evidence of pathogenicity	Report with a statement that additional evidence may be required to confirm pathogenicity; recommend family studies or follow-up testing if appropriate.
VUS	Conflicting or insufficient evidence regarding clinical significance	Report cautiously; clearly state the uncertainty and recommend re-analysis or segregation studies when relevant.
Likely Benign (LB)	Evidence suggests minimal or no clinical impact	Generally not reported unless relevant to clinical question or phenotype under investigation.
Benign (B)	Strong evidence supporting lack of disease association	Not reported in clinical results. May be documented internally for quality tracking.

Figure 2: Clinical Pathway of CMA Testing in Patients with Unexplained DD, ID, MCA, and ASD



* Excludes patients with recognizable syndrome (e.g. Down syndrome), family history of a chromosomal rearrangement or multiple miscarriages

Genetic Counseling

3.6.4.1 In accordance with Federal Law by Decree No. (49) of 2023 Regulating the Use of the Human Genome, genetic counseling is mandatory for all patients undergoing microarray testing. Counseling must be performed by a DoH-licensed clinical geneticist or genetic counselor to ensure patients and families understand the purpose, limitations, and potential implications of the test results.

3.6.4.2 Pre-test counseling must cover:
- 3.6.4.2.1 The purpose and scope of the screening.
- 3.6.4.2.2 Potential physical and psychological effects of results.
- 3.6.4.2.3 Risks of the test and, if relevant, the need for repeat or periodic screening.
- 3.6.4.2.4 The analytical and clinical limitations of microarray testing and how these may be mitigated by additional established methodologies when clinically indicated.

3.6.4.3 Post-test counseling must cover:
- 3.6.4.3.1 Clear explanation of the results (including CNV classification, inheritance, and residual risk).
- 3.6.4.3.2 Potential clinical implications, including preventive or therapeutic interventions.
- 3.6.4.3.3 Recommendations for additional testing or screening, including use of complementary methods to address limitations of the microarray assay when needed.
- 3.6.4.3.4 Referral to specialist physicians when indicated.

4.Relevant References Documents

No.	Reference Date	Reference Name	Relation Explanation / Coding / Publication Links
1	2010	American Journal of Human Genetics	Consensus statement: Chromosomal microarray as first-tier diagnostic test. Am J Hum Genet. 2010;86(5):749–764. https://doi.org/10.1016/j.ajhg.2010.04.006
2	2021	ACMG Technical Standards	Chromosomal microarray analysis, 2021 ACMG technical standard. Genet Med. 2021;23(11):1818–1829.https://doi.org/10.1038/s41436-021-01214-w
3	2020	ACMG / ClinGen	ACMG/ClinGen standards for CNV interpretation. *Genet Med.* 2020;22(2):245–257. https://doi.org/10.1038/s41436-019-0686-8
4	2013	ACMG Statement	Clinical utility of chromosomal microarray testing. Genet Med. 2013;15(6):505–522. https://doi.org/10.1038/gim.2013.129
5	2016	ACOG Committee on Genetics & SMFM	ACOG Committee Opinion No. 682: Microarrays & NGS in obstetrics. *Obstet Gynecol.* https://www.acog.org/clinical/clinical-guidance/committee-opinion/articles/2016/12/microarrays-and-next-generation-sequencing-technology-the-use-of-advanced-genetic-diagnostic-tools-in-obstetrics-and-gynecology
6	2015	Canadian College of Medical Geneticists (CCMG)	Guidelines for genomic microarray testing in Canada. Canadian College of Medical Geneticists. https://www.ccmg-ccgm.org/wp-content/uploads/2022/04/CCMG_Guidelines_for_Genomic_Microarray_Testing_FINAL.pdf
7	2021	WHO CNS Tumor Classification	2021 WHO classification of CNS tumors: summary. Acta Neuropathol. 2021;141(3):1–17. https://doi.org/10.1007/s00401-021-02384-y
8	2024	U.S. Food and Drug Administration	MicroArray/Sequencing Quality Control (MAQC/SEQC) Project. U.S. Food and Drug Administration. https://www.fda.gov/science-research/bioinformatics-tools/microarraysequencing-quality-control-maqcseqc-project
9	2025	Update - Mandatory DoH Prior Approval for the Use of Modern Health Technologies and Therapeutic Practices in Abu Dhabi Emirate.	USO/140/2025 https://www.doh.gov.ae/-/media/FD843DFB44754CAEB66B2AA646A56478.ashx

10	2023	Standard for Laboratory Accreditation for Genomic-Related Services and Products	DOH/RIC/ST/LAGRSP/V1/2023 https://www.doh.gov.ae/-/media/7E35F99573FD488DBD6F6AC3F8835465.ashx
11	2024	College of American Pathologists	CAP GEN.43800 / GEN.43850: College of American Pathologists. Laboratory General Checklist. CAP Accreditation Program. https://www.cap.org/laboratory-improvement/accreditation/accreditation-checklists
12		Clinical Genome Resource	ClinGen Dosage Sensitivity Map: ClinGen Dosage Sensitivity Map. Clinical Genome Resource (ClinGen). https://dosage.clinicalgenome.org/
13	2015	ACMG Standards and guidelines for the interpretation of sequence variants	Standards and guidelines for the interpretation of sequence variants: a joint consensus recommendation of the American College of Medical Genetics and Genomics and the Association for Molecular Pathology https://doi.org/10.1038/gim.2015.30
14	2011	HAAD Clinical Laboratory Standards	Health Policy and Regulation Health Authority Abu Dhabi Clinical Laboratory Standards. https://www.doh.gov.ae/-/media/ECC2A193AF84440AA795437BC9CCDA48.ashx
15	2025	Professional Qualification Requirement (PQR)	The Health Regulatory Authorities in the United Arab Emirates (UAE) represented by: Professional Qualification Requirement (PQR) https://www.doh.gov.ae/en/pqr
16	2025	ClinGen	https://clinicalgenome.org/
17	2025	DECIPHER	https://www.deciphergenomics.org/
18	2025	Database of Genomic Variants	https://dgv.tcag.ca/
19	2025	ClinVar	https://www.ncbi.nlm.nih.gov/clinvar/
20	2025	OMIM	https://www.omim.org/
21	2025	gnomAD	https://gnomad.broadinstitute.org/
22	2025	PubMed	https://pubmed.ncbi.nlm.nih.gov/