การแพทย์จีโนมิกส์สู่การวินิจฉัยและการรักษาที่แม่นยำ

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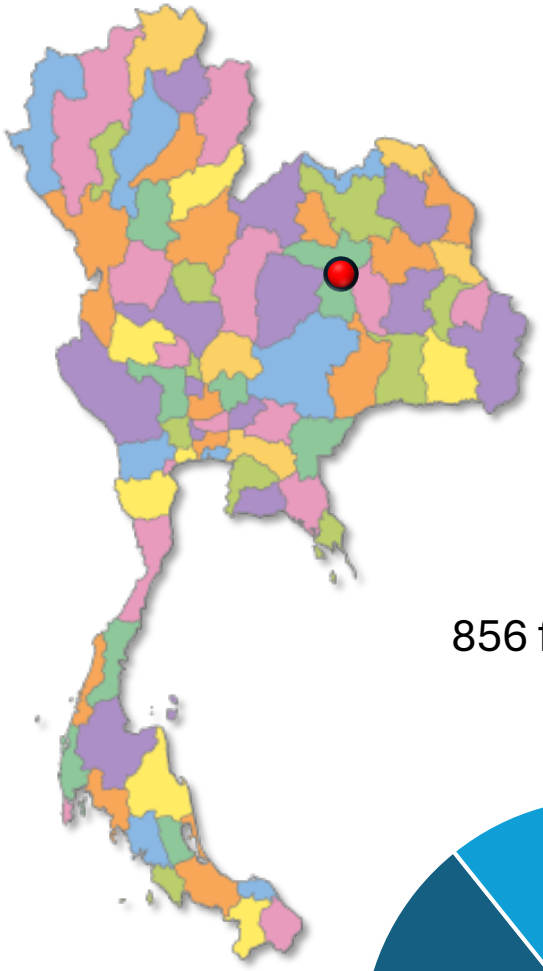
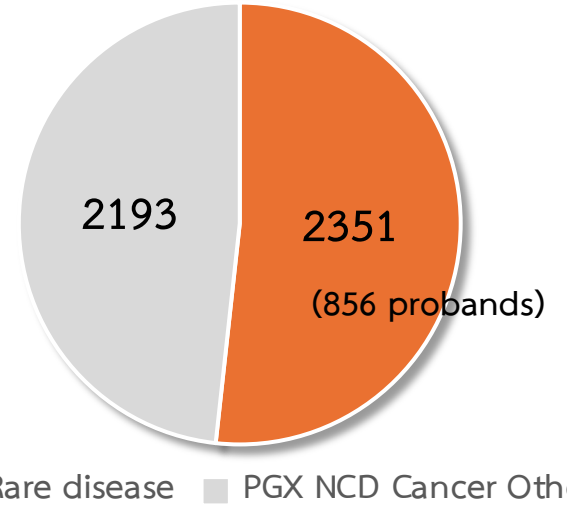


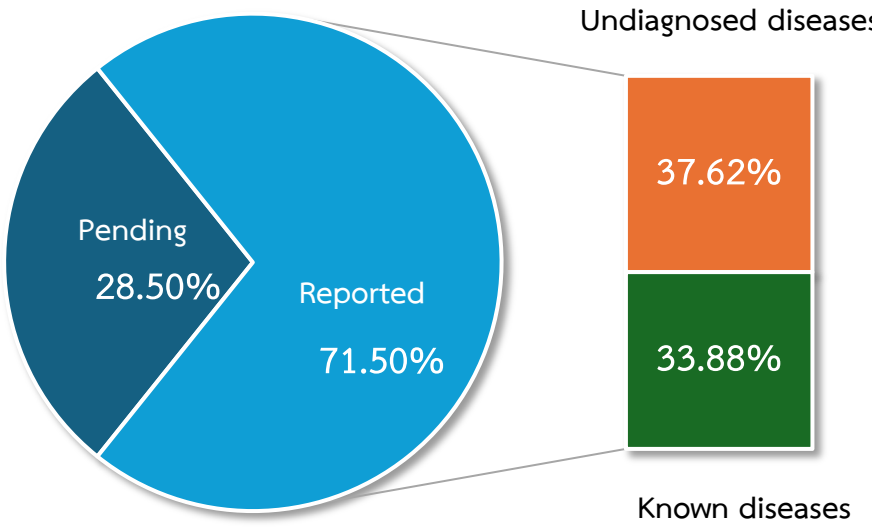


June 2020 to February 2024

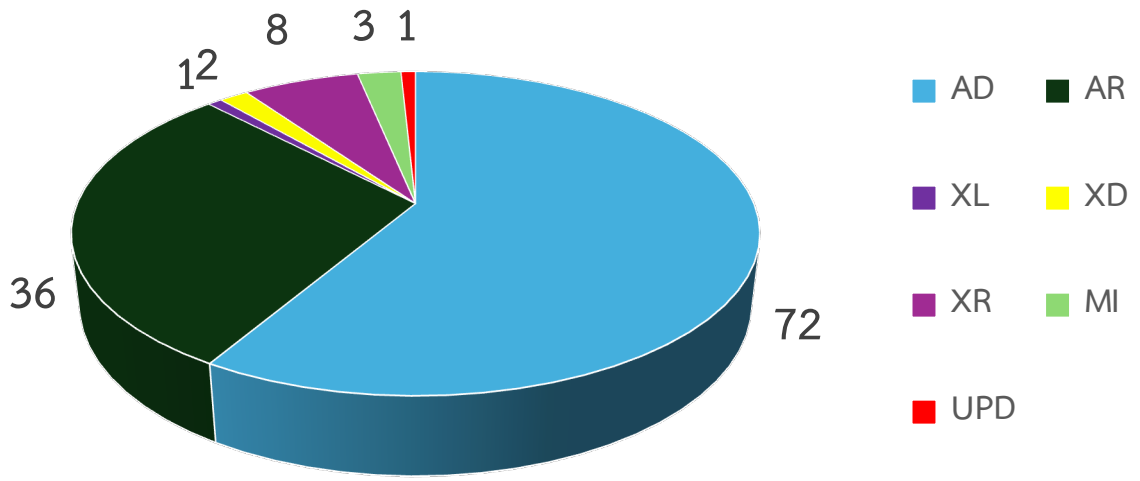
856 families (2351 participants) 51.74%

	Number of patients	%	Positive results	%
Sex				
male	178	55.28	58	32.58
female	144	48.57	65	45.14
Singleton WGS	47	14.60	16	34.04
Duo WGS	63	19.57	24	38.10
Trio WGS	190	59.00	72	37.89
Large family WGS	22	6.83	11	50.00
All (all participants)	322 (834)		123	38.08

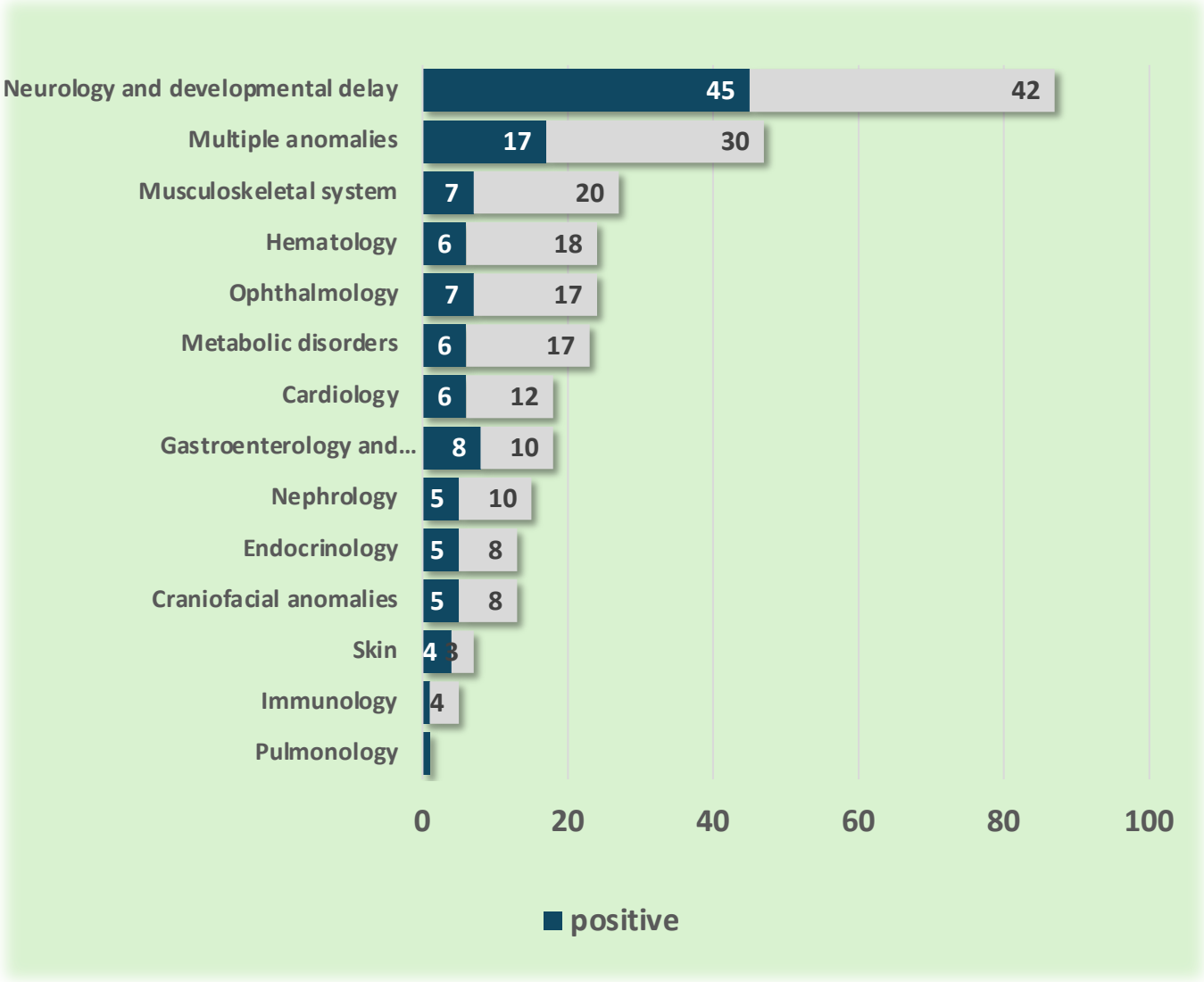
Diagnostic Yield 38.08%



Mode of inheritance

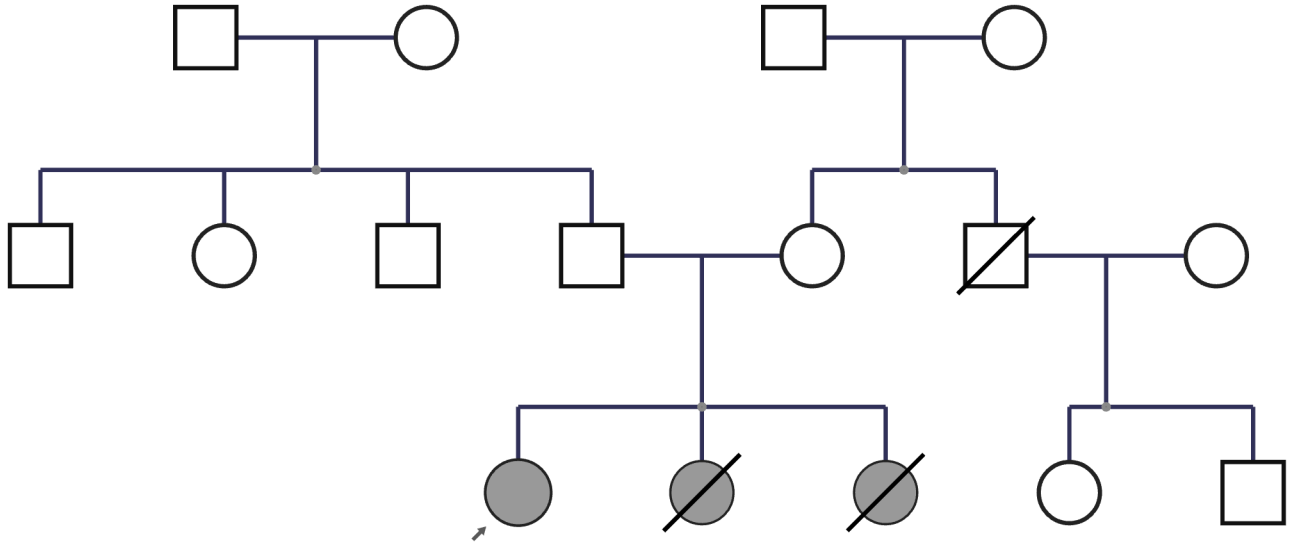


Diagnostic yield of WGS across various phenotypic categories

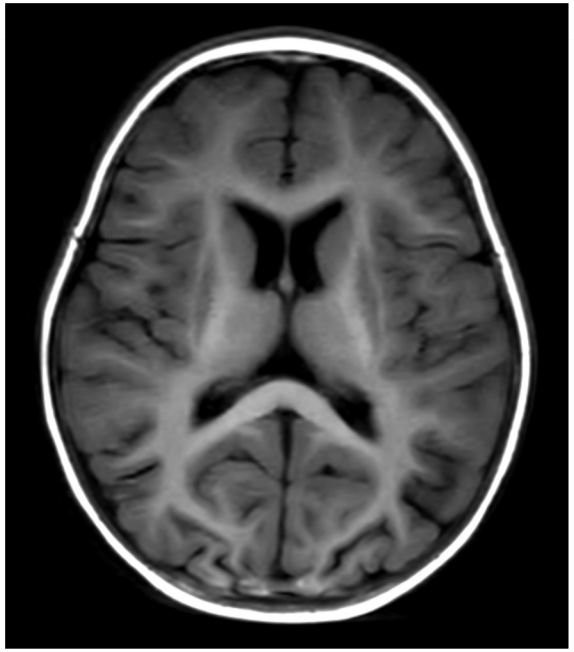


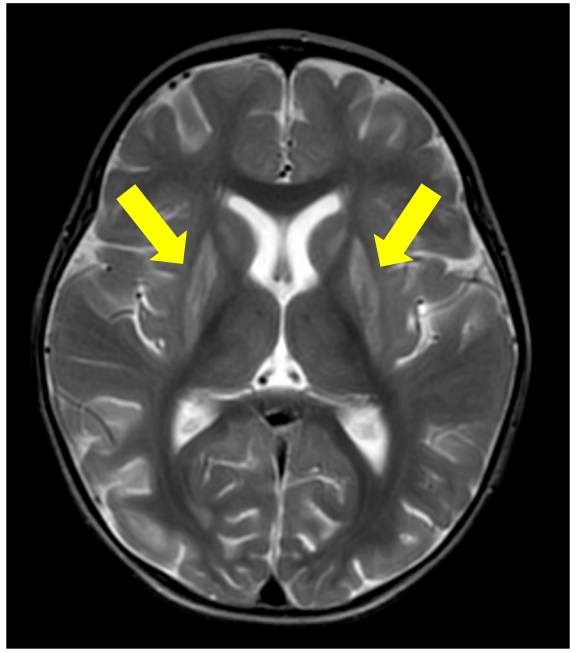
Phenotypic categories	number of patients	positive results	%positive
Pulmonology	1	1	100.00
Skin	7	4	57.14
Neurology and developmental delay	87	45	51.72
Gastroenterology and hepatobiliary system	18	8	44.44
Craniofacial anomalies	13	5	38.46
Endocrinology	13	5	38.46
Multiple anomalies	47	17	36.17
Nephrology	15	5	33.33
Cardiology	18	6	33.33
Ophthalmology	24	7	29.17
Metabolic disorders	23	6	26.09
Musculoskeletal system	27	7	25.93
Hematology	24	6	25.00
Immunology	5	1	20.00

Case I Recurrent infantile onset encephalopathy



- G1 เสียชีวิตตอน 3 เดือนเป็น influenza A + acute necrotizing encephalitis
- G2 เสียชีวิตตอน 7 เดือน acute encephalopathy MRI brain: bilateral symmetrical basal ganglia lesion
- G3 ทารกแรกเกิดปกติ แต่ตรวจพบ lactate 34.8 mg/dl





MRI brain at 8 months: Symmetric abnormal increased SI on T2/FLAIR at bilateral lentiform nuclei (more prominent at putamina), caudate nuclei, thalami, and periaqueductal region. Diffusion restriction involving bilateral putamina and caudate nuclei are seen

Back

NBTID: 73744757

GeTHlyzer | Discovery | PHENOTIPS

Filtering | Candidate | Hide 0

Selected | Predicted | GEL | GeneSet | ACMG | 10/11 | Search HPO ... | Search Gene1,Gene2 ...

1 chr2_227699286_G_T SLC19A3 Homozygous GeneCards OMIM GeneReviews

varsome | IGV Compound | IGV | Add | Hide

ACMG Classsification	ACMG Prediction	Probability of Pathogenicity
PVS1 PM2	likely pathogenic	

Feature	Variant	Protein	Impact	Consequence	Mane Select	Predicted Deleterious	ClinVar Submitter(s)
NM_025243.4	c.429C>A	p.Cys143Ter	HIGH	stop_gained	YES	4	Pathogenic () - Benign ()

More detail

Diagnosis: Biotin-Thiamine-Responsive Basal Ganglia Disease

2 chr2_151519744_T_C NEB Heterozygous

IGV | Add | Hide

ACMG Classsification	ACMG Prediction	Probability of Pathogenicity	gnomADe	gnomADg	ThADe	ThADg
PM1 PM2 BP1 BP4	likely benign		0.00001205 (3/248960)	0.00001396 (2/143310)	0.00228938 (5/2184)	0.00207254 (4/1930)

Feature	Variant	Protein	Impact	Consequence	Mane Select	Predicted Deleterious	ClinVar Submitter(s)
NM_001164508.2	c.22504A>G	p.Lys7502Glu	MODERATE	missense_variant	YES	2 4	Pathogenic () - Benign ()

More detail

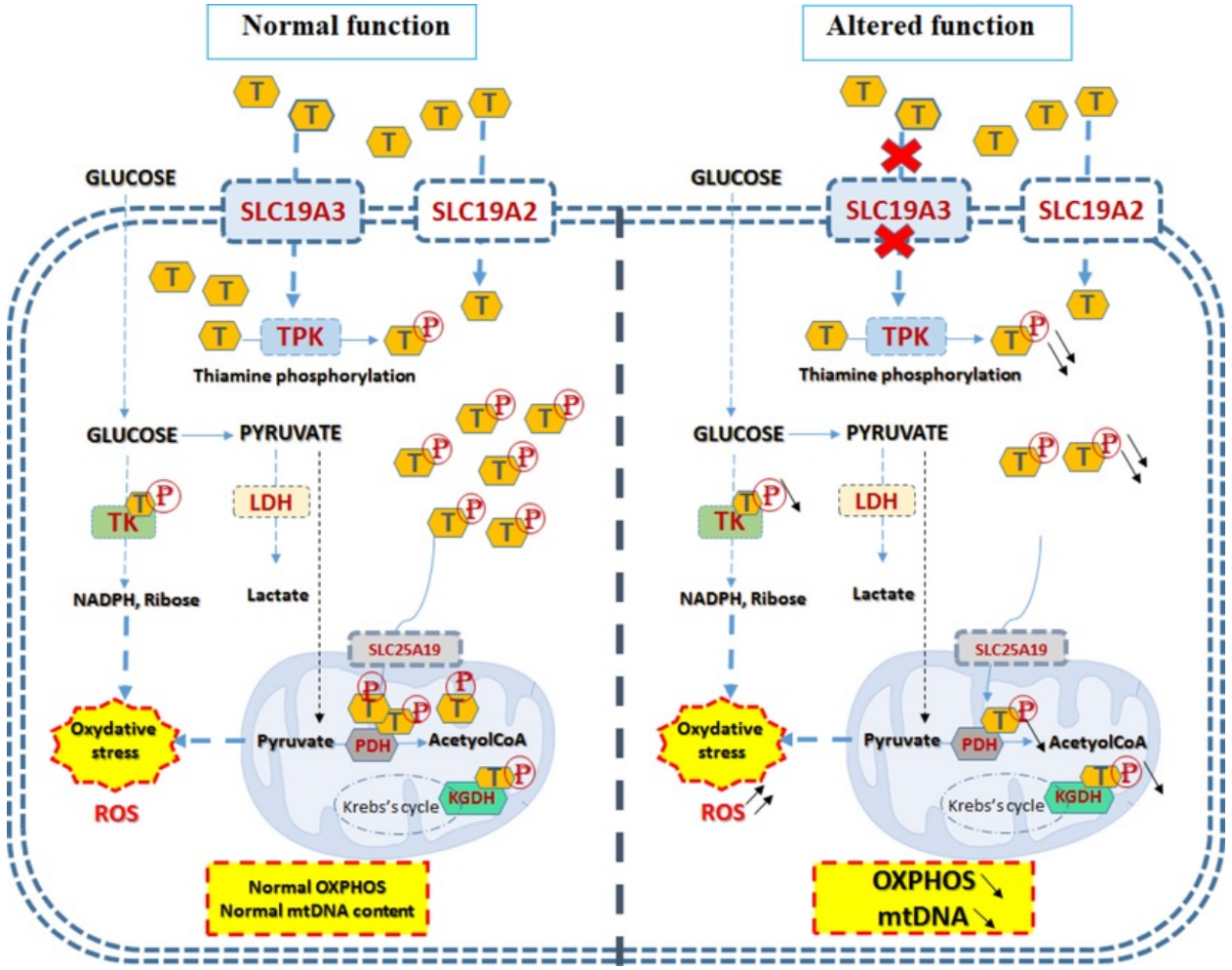
3 chr14_31561510_C_T NUBPL Heterozygous GeneCards OMIM GeneReviews

varsome | IGV Compound | IGV | Add | Hide

ACMG Classsification	ACMG Prediction	Probability of Pathogenicity	gnomADe	gnomADg	ThADe	ThADg	ExAC
PM1 PM2 BS1 BP4	likely benign		0.00005962 (7/117416)	0.00002093 (3/143318)	0.00505515 (11/2176)	0.00362694 (7/1930)	0.00006679 (8/119770)

Feature	Variant	Protein	Impact	Consequence	Mane Select	Predicted Deleterious	ClinVar Submitter(s)
NM_025152.3	c.71C>T	p.Pro24Leu	MODERATE	missense_variant	YES	14	Pathogenic - Benign

Biotin-Thiamine-Responsive Basal Ganglia Disease



Treatment of Manifestations in Individuals with BTBGD

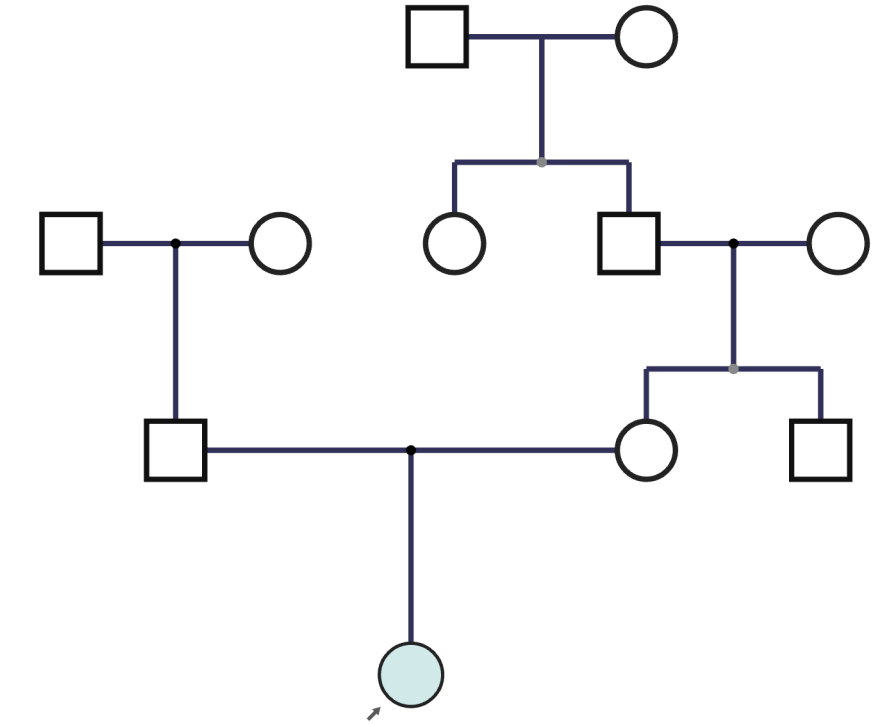
Manifestation/ Concern	Treatment	Considerations/Other
Acute encephalopathy	ICU care incl treatment of seizures & ↑ intracranial pressure	Empiric treatment w/antimicrobial/antiviral agents recommended until infectious causes of acute/subacute encephalopathy are ruled out
Neurologic disorder	Both biotin & thiamine oral therapy: • Biotin: 5-10 mg/kg/day • Thiamine: ≤40 mg/kg/day; max of 1500 mg/day	• Note: Some persons respond only to thiamine. • Lifelong treatment w/biotin & thiamine required • During acute decompensation thiamine may be ↑ to 2x regular dose & given intravenously.
	Fever control	Fever exacerbates the disease.
Seizures	Anti-seizure medication to control seizures	Avoid sodium valproate.
Dystonia	Symptomatic treatment incl trihexyphenidyl or L-dopa	
Developmental delays	Rehab, PT, OT, speech therapy, & educational programs adapted to individual needs	
Social	Education of family re importance of lifelong compliance w/medical therapy	

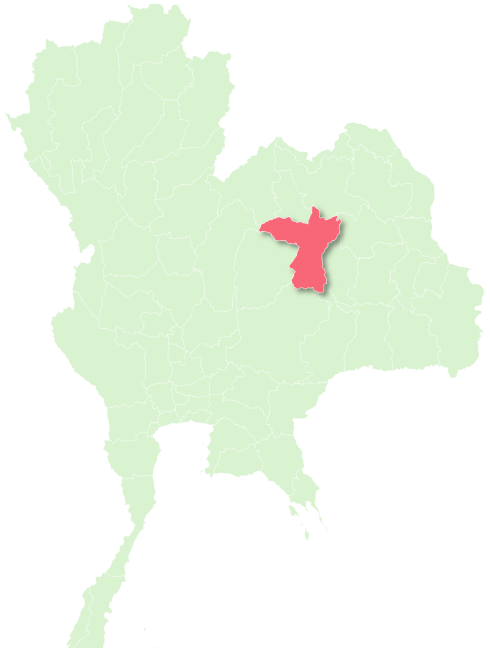
Felhi R, Sfaihi L, Charif M, Frikha F, Aoiadni N, Kamoun T, Lenaers G, Fakhfakh F. Vitamin B1 deficiency leads to high oxidative stress and mtDNA depletion caused by SLC19A3 mutation in consanguineous family with Leigh syndrome. Metab Brain Dis. 2023 Oct;38(7):2489-2497. doi: 10.1007/s11011-023-01280-w. Epub 2023 Aug 29. PMID: 37642897.

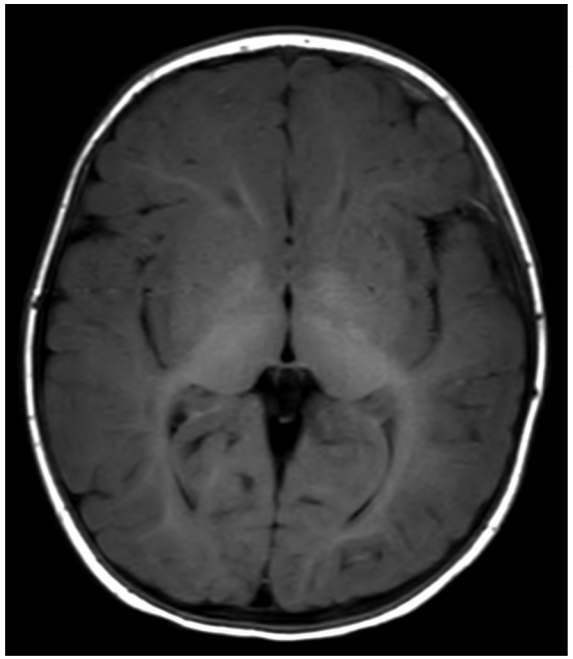
Tabarki B, Al-Hashem A, Alfadhel M. Biotin-Thiamine-Responsive Basal Ganglia Disease. 2013 Nov 21 [Updated 2020 Aug 20]. In: Adam MP, Feldman J, Mirzaa GM, et al., editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2024. Available from: https://www.ncbi.nlm.nih.gov/books/NBK169615/

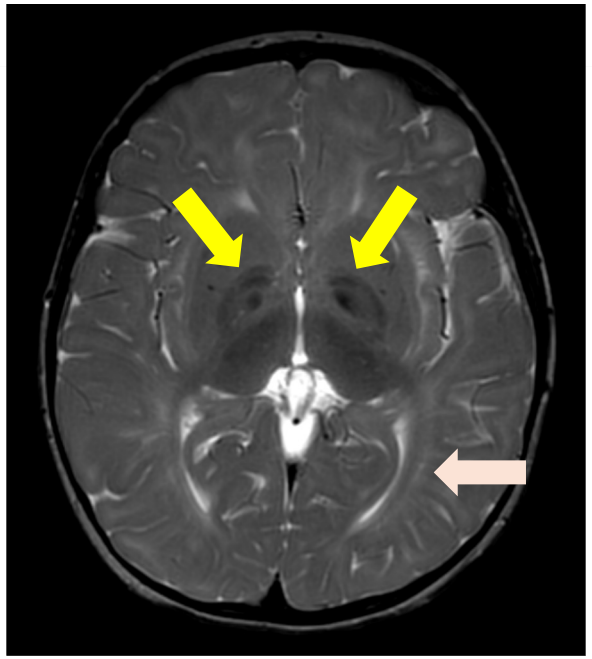
Case II

- เด็กหญิงอายุ 1 ปี 8 เดือน มีพัฒนาการช้ารอบด้าน นั่งได้ตอน 7-8 เดือน คลานได้ตอน 1 ขวบ เกาะยืน 1.2 ปี พูดเป็นคำ ๆ ได้ 5-6 คำ
- ตรวจร่างกายไม่พบความผิดปกติ
- Chromosome study : 46,XX

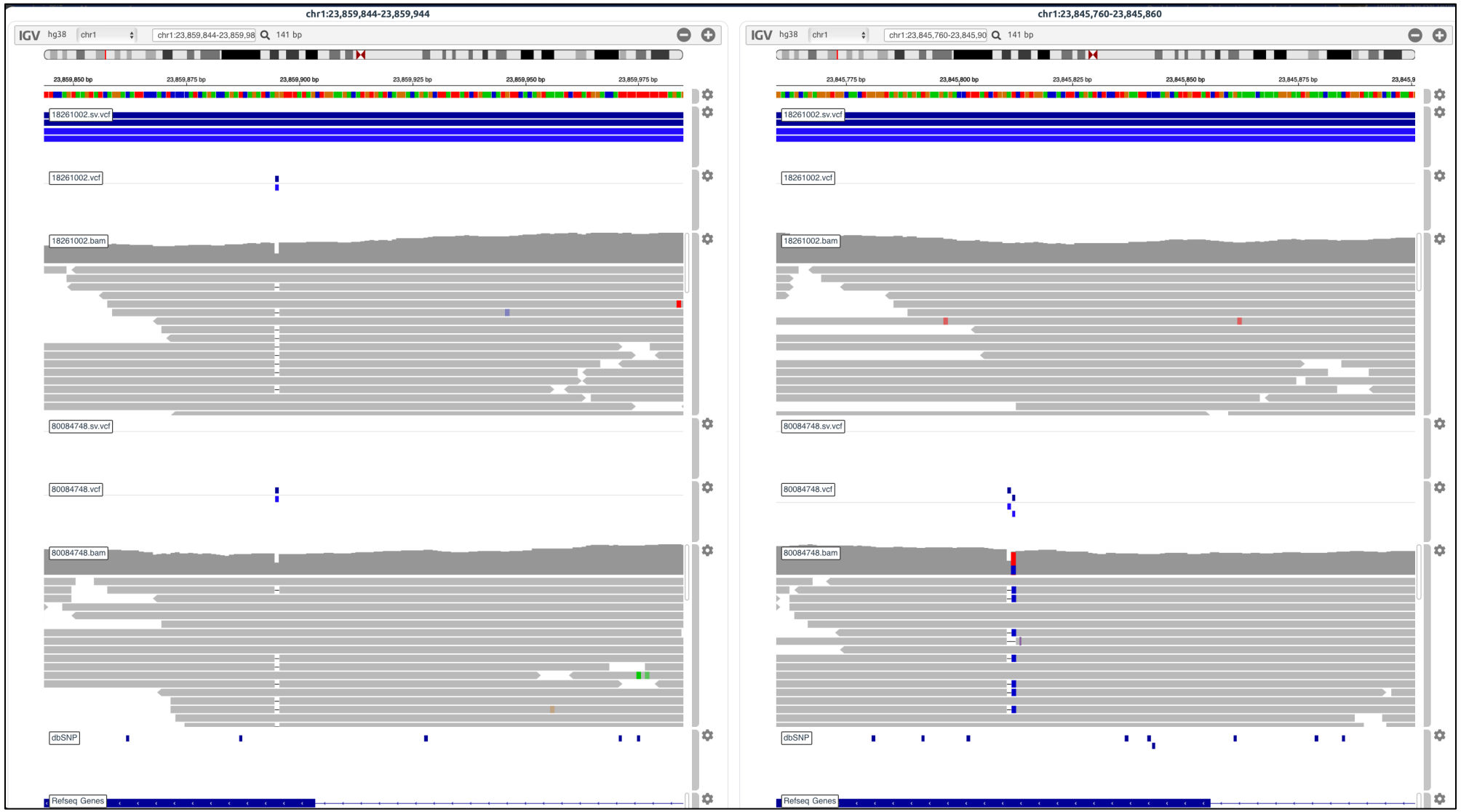








MRI brain: Diffuse high SI in T2W involving subcortical, deep white matter of bilateral cerebral hemispheres and posterior limb internal capsule. There is abnormal high SI in T1W and low SI in T2W involving both thalami.
DDx 1) gangliosidoses 2) Krabbe disease



- Compound heterozygous c.671del, p.Pro224LeufsTer3 (pathogenic) and c.1304_1305delinsC, p.Asp435Alafs*26 (likely pathogenic) in *FUCA1*
- Diagnosis: Fucosidosis

Fucosidosis

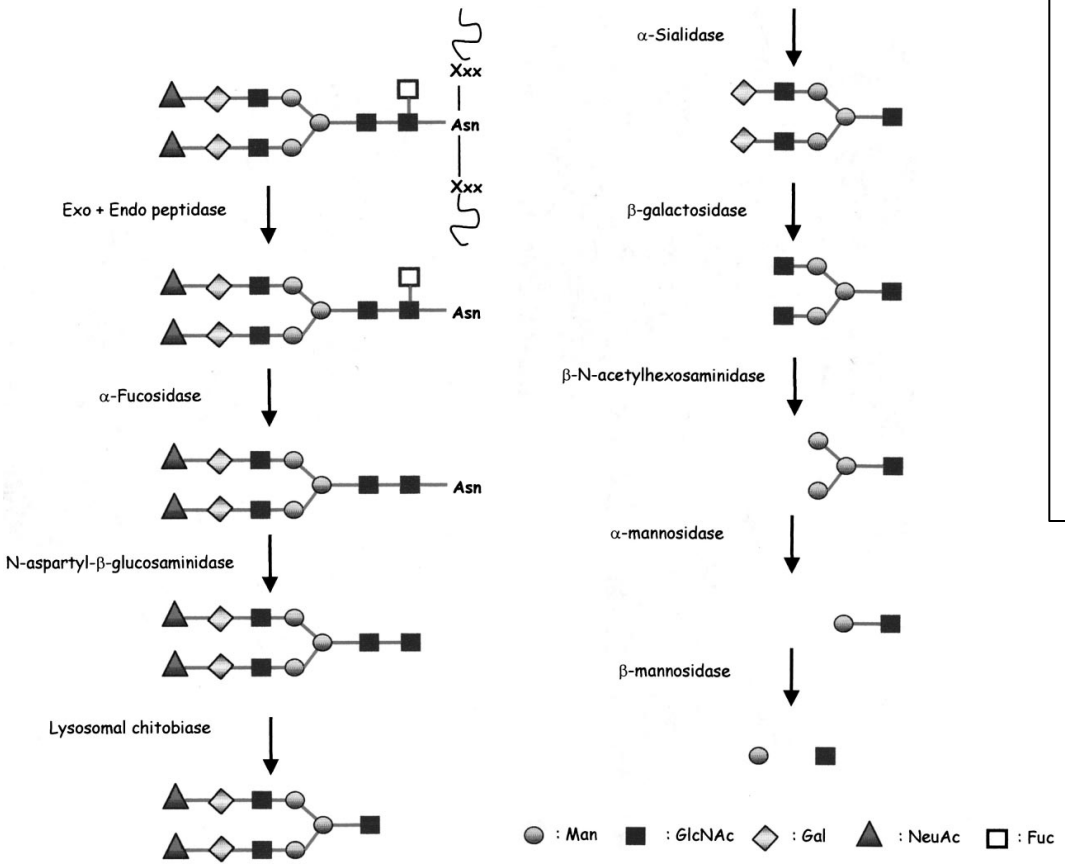


Fig. 1. The bi-directional catabolic pathway for *N*-acetyllactosaminic glycoproteins.

Michalski JC, Klein A. Glycoprotein lysosomal storage disorders: alpha- and beta-mannosidosis, fucosidosis and alpha-N-acetylgalactosaminidase deficiency. Biochim Biophys Acta. 1999 Oct 8;1455(2-3):69-84. doi: 10.1016/s0925-4439(99)00077-0. PMID: 10571005.

Table I. Lysosomal storage diseases with defects in proteases or catabolism of glycoproteins

Disorder	Enzymic defect
Fucosidosis	α-L-fucosidase (E.C. 3.2.1.51)
Sialidosis (mucolipidosis I)	N-acetyl-α-neuraminidase (E.C. 3.2.1.18) (sialidase)
G_{M1}-gangliosidosis	β-D-galactosidase (E.C. 3.2.1.23)
Sandhoff disease	β-D-hexosaminidase A and B (E.C. 3.2.1.30)
α-Mannosidosis	α-D-mannosidase (E.C. 3.2.1.24)
β-Mannosidosis	β-D-mannosidase (E.C. 3.2.1.26)
Aspartylglucosaminuria	*N*-(β-*N*-acetylglucosaminyl)-1-asparaginase or aspartylglucosaminidase (E.C. 3.5.1.26)
Schindler (Kawasaki) disease	α-*N*-acetylgalactosaminidase (α-D-galactosidase B) (E.C. 3.2.1.49)
I-cell disease and pseudo-Hurler polydystrophy (mucolipidosis II and III)	UDP-*N*-acetylglucosamine-1-phosphotransferase leading to multiple enzyme deficiencies
Galactosialidosis	Protective protein/cathepsin A (E.C. 3.4.16.1)
Papillon-Lefevre syndrome	Cathepsin C (E.C. 3.4.14.1)
Ovine and murine ceroid lipofuscinosis	Cathepsin D (E.C. 3.4.23.5)
Pycnodysostosis	Cathepsin K
Infantile neuronal ceroid lipofuscinosis	Palmitoyl protein thioesterase
Late infantile neuronal ceroid lipofuscinosis	Tripeptidyl peptidase 1

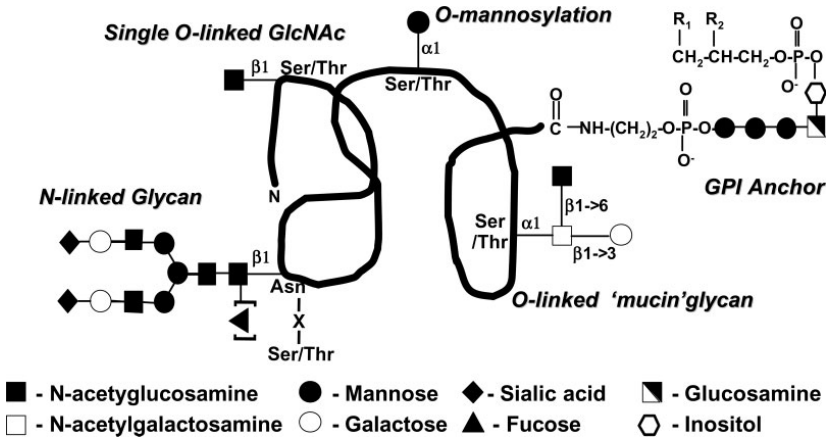


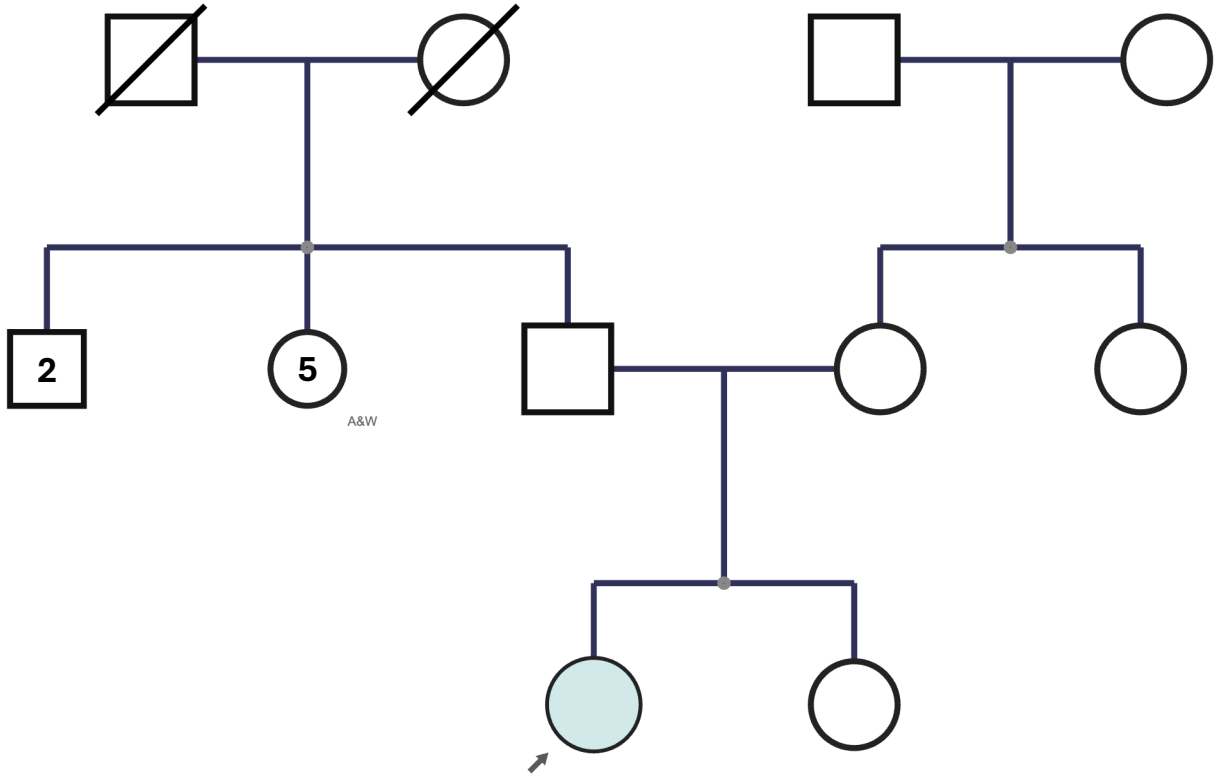
Fig. 2. The main forms of attachment of carbohydrate to protein in mammalian cells.

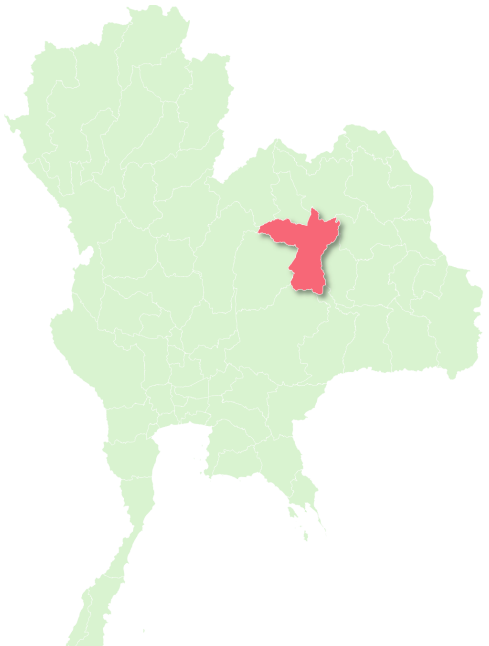
Winchester B. Lysosomal metabolism of glycoproteins. Glycobiology. 2005 Jun;15(6):1R-15R. doi: 10.1093/glycob/cwi041. Epub 2005 Jan 12. PMID: 15647514.

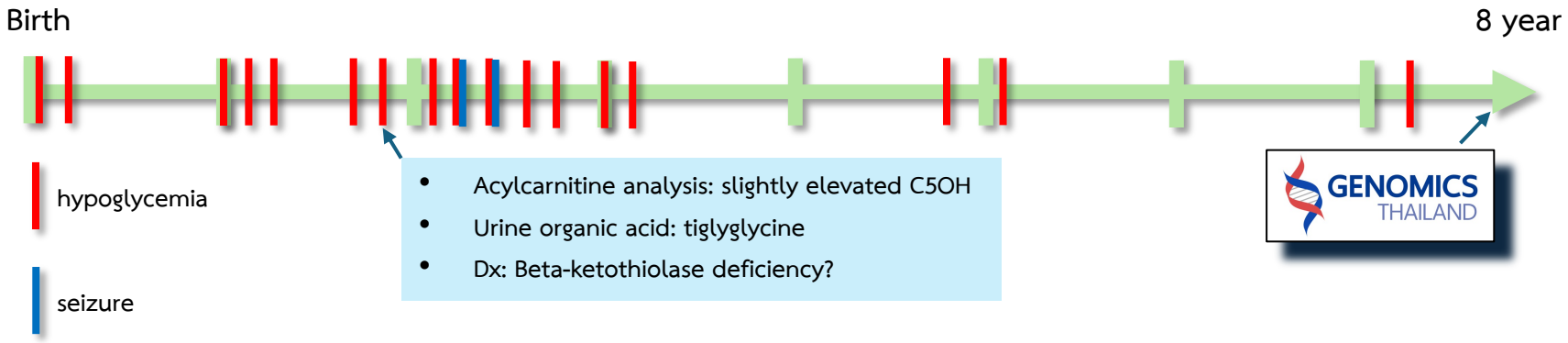
Genotype Set [1]	*FUCA1* Pathogenic Variant(s) and Zygosity		Ethnic Origin (pts)	Gender/No of Cases	Family History	Phenotype			Ref.
						Developmental History	Additional Clinical and Enzymatic Findings	Disease Type	
G6	c.437delC, p.(Pro146Argfs*41)	hmz	Italian (8)			NA			[27]
G7	c.459G > A, p.Trp153*/ entire gene del, loss of protein	htz comp	Japanese (1)	Female	Non-consan-guineous parents	At 23 months: delayed speech and hearing difficulty. At 3 years and 7 months: coarse face, small stature and kyphoscoliosis. At 4 years: angiokeratoma on the palms, bone abnormalities and motor dysfunction gradually progressed. At age 6 years: unable to walk, myoclonic seizures developed. At age 13 years: spasticity and dystonia of all extremities with involuntary, movements, generalized angiokeratoma corporis diffusum, no hepatosplenomegaly or pubertal development.	α-L-fucosidase in leukocytes (0 nmol/h per mg protein compared with 29.1 ± 4.7 nmol/h per mg protein in control).	Chronic, slow progressive	[32]
G8	c.464C > T, p.Ser155Phe/c.790C > T, p.Arg264*	htz comp	Spanish or Portuguese (1)			NA			[33]
G9	c.467_468delAA, p.(Lys156Argfs*11)/ c.1160G > A, p.Trp387*	htz comp	Italian (1)			NA	All patients had negligible enzyme activity and reduced CRIM.	Not classified	[34]
G10	c.525-76_663-163del3282, p.?/c.671delC, p.(Pro224Leufs*3)	htz comp	Thai (1)	Male	Non-consanguineous parents	Until 2 years of age: milestones reportedly attained within normal limits. At age 3 years and 6 months: psychomotor regression (lost his ability to communicate verbally, had spasticity in which the lower extremities were more affected than the upper). Growth parameters and development continued to deteriorate At 7 years: bed-ridden, coarse facial features with macroglossia. At age 9 years: angiokeratomas.	Brain MRI: increasing degrees of cerebral atrophy with significant signal changes in the thalamus. Chest X-ray: oar-like ribs, bullet-shaped vertebrae, and widening of both clavicular heads.	Not classified	[35]
G11	c.564G > A, p.Trp188*	hmz	Austrian (1)			NA			[30]
G12	c.648C > A, p.Tyr216*	hmz	Belgian (1)						[27]

Case III

- เด็กหญิงอายุ 6 ปี มีประวัติ recurrent hypoglycemia lactic acidosis และ ketosis ตั้งแต่ภายหลังคลอด อาการจะดีขึ้นหลังได้รับสารน้ำ
- ช่วงอายุ 1 ปีแรกมีปัญหาน้ำตาลในเลือดต่ำหลายครั้ง ร่วมกับมี severe wide anion gap metabolic acidosis, hyperlactatemia



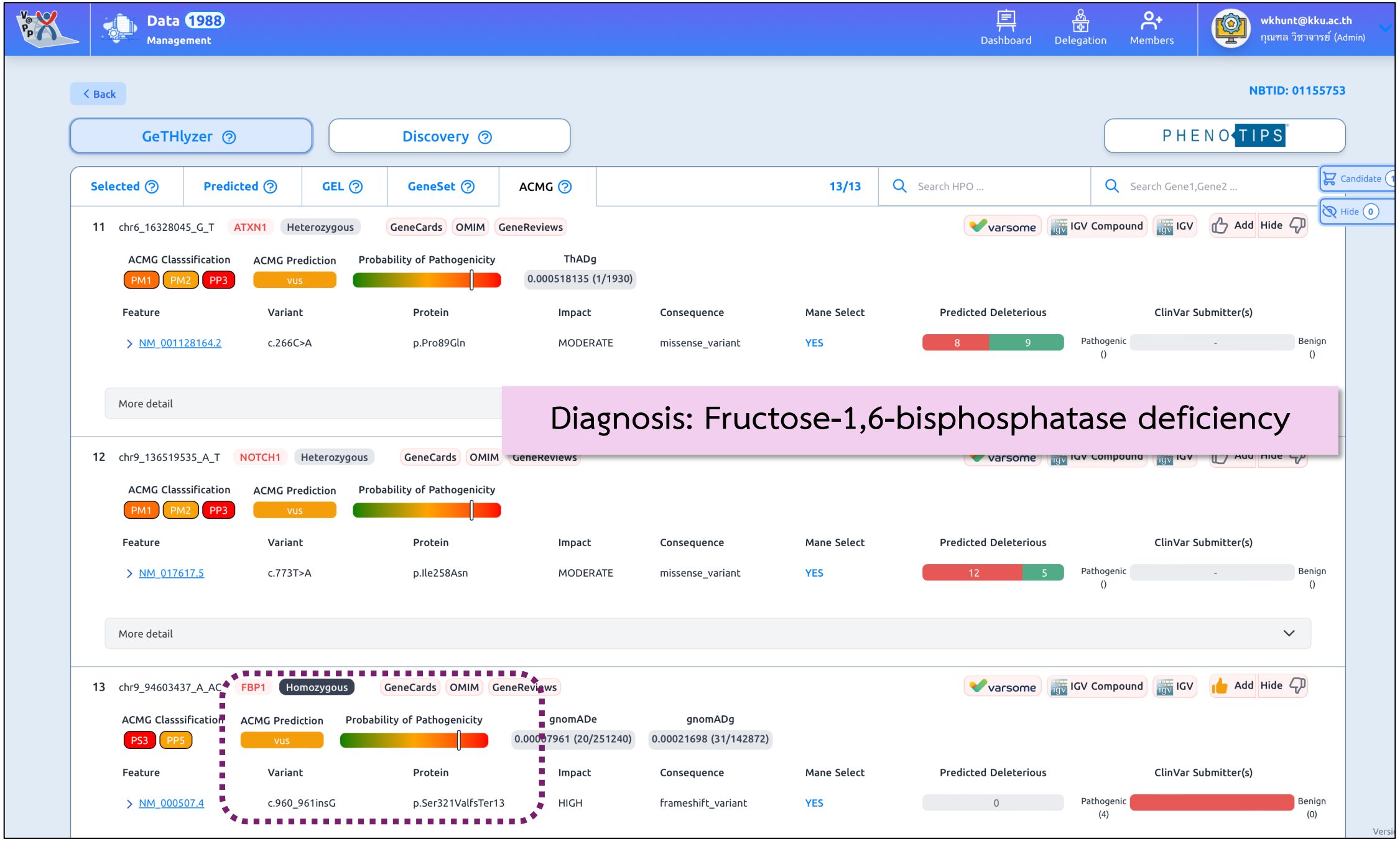




Biochemical Parameters of Disorders in the Differential Diagnosis of Fructose-1,6-Bisphosphatase Deficiency

Biochemical Parameter	FBP1	BKT	HFI	GSDI	PCD	FAOD	Respiratory Chain Defects
↑ lactate	Fasting	During crisis	Fasting	Permanent, also increased w/fasting	Permanent	Fasting	Permanent
Lactate/ pyruvate ratio	20-40	–	–	–	>30	–	>20
Ketosis	↑↑/–	↑↑	↑	↑↑/–	↑↑↑	–	↑↑
Triglycerides	Pseudo-hyper	↑	N	↑	N	N	N
Glucose	L	L	L	N/L	L/N/H	N/L	L
Ammonia	N	↑	N	N	↑↑	↑	N/↑↑
Alanine	↑↑	N	↑↑	N	↑↑	N	↑↑
Citrulline	N	N	N	N	H	N	N
Liver dysfunction (transaminitis)	↑/–	↑	↑	↑	↑	N	↑/–
Uric acid	↑	N	N	↑	N	N	N
Organic acids in urine	Ketonuria, glycerol, glycerol-3-phosphate	2-methylacetocetate, 2-methyl-3-hydroxybutyryl CoA, tiglylglycine	Ketonuria	Ketonuria	Lactate/ ketonuria	↑ C16-C22	Lactate

Bijarnia-Mahay S, Bhatia S, Arora V. Fructose-1,6-Bisphosphatase Deficiency. 2019 Dec 5. In: Adam MP, Feldman J, Mirzaa GM, et al., editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2024. Available from: https://www.ncbi.nlm.nih.gov/books/NBK550349/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Dashboard
Delegation
Members
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GeTHlyzer
Discovery
PHENOTIPS
Selected
Predicted
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GeneSet
ACMG
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Search HPO ...
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Candidate
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Diagnosis: Fructose-1,6-bisphosphatase deficiency
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ACMG Prediction vus
Probability of Pathogenicity
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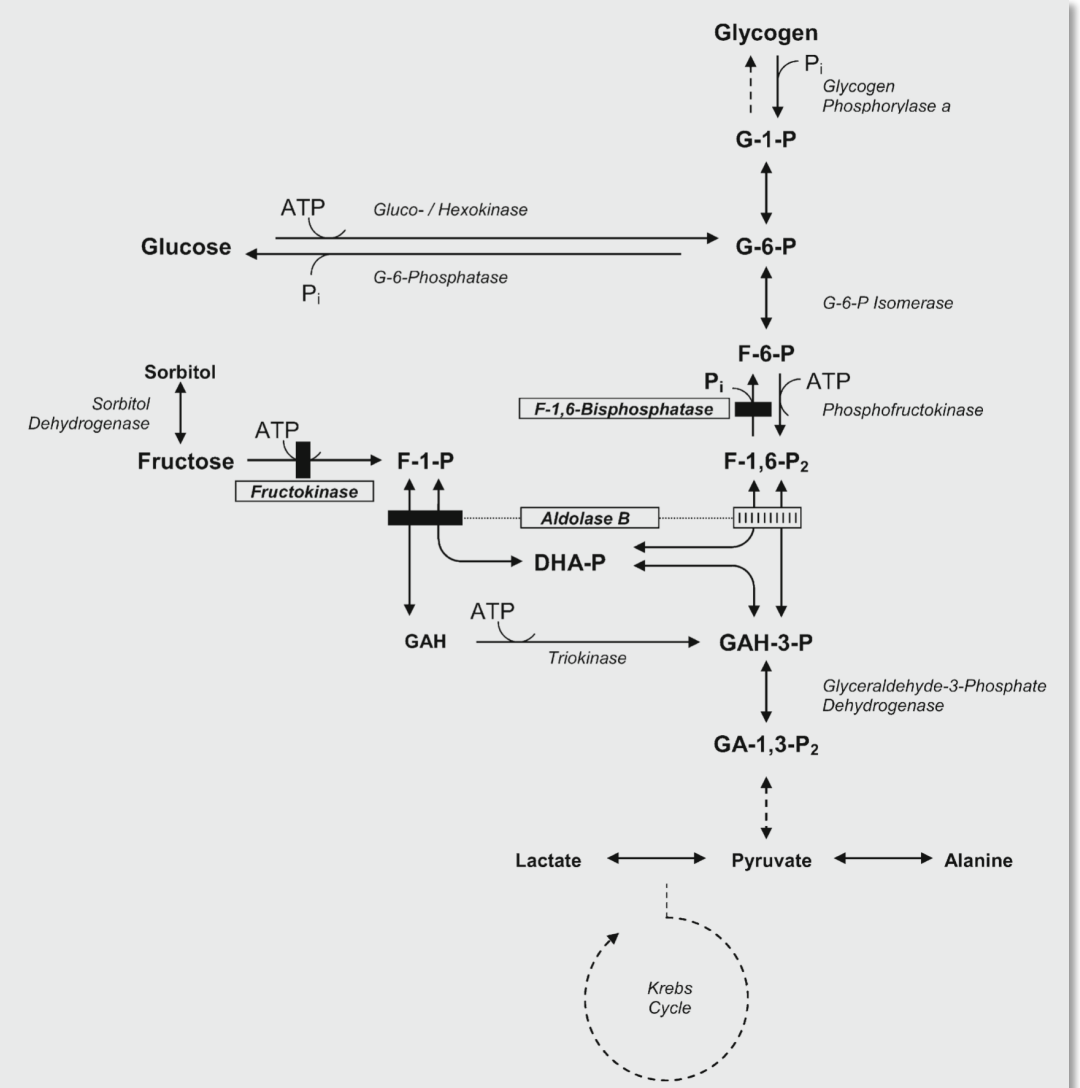


Fig. 9.1. Fructose metabolism. *DHA-P*, Dihydroxyacetone phosphate; *F*, fructose; *G*, glucose; *GA*, glycerate; *GAH*, glyceraldehyde; *P*, phosphate; *Pi*, inorganic phosphate. The three enzyme defects in fructose metabolism are boxed and depicted by *solid bars* across the *arrows*; the diminished activity of aldolase B toward fructose-1,6-bisphosphate is depicted by a *broken bar*

Emergency Outpatient Treatment in Individuals with Fructose-1,6-Bisphosphatase Deficiency

Manifestation/ Concern	Treatment	Considerations
Mildly ↑ catabolism (fever, cough, or cold; loose stools; continues to eat; not assoc w/lethargy)	1. Restriction of fructose, sucrose, glycerol, & sorbitol 2. ↑ frequency of carbohydrate feedings 3. Intake of glucose polymers	• Trial of outpatient treatment at home requires good communication between family & providers. • Frequent reassessment of patient
Ketonuria		An early indicator of impending crisis

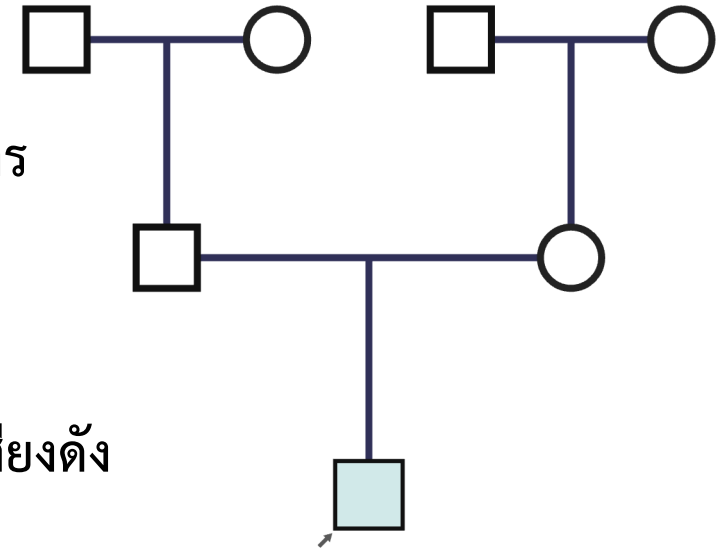
Acute In-Patient Treatment in Individuals with Fructose-1,6-Bisphosphatase Deficiency

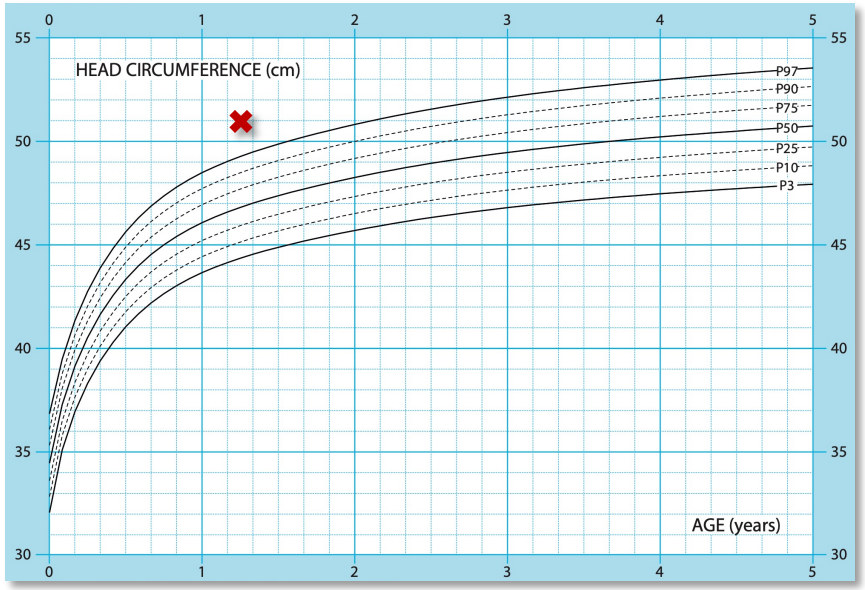
Manifestation/ Concern	Treatment	Considerations
Hypoglycemia	1. IV glucose bolus (2 mL/kg of 10% dextrose) followed by continuous infusion of glucose at high rates (10% dextrose infusion) 2. Transition to oral/enteral feeds as clinically tolerated	The symptoms of acute illness typically subside soon after administration of IV dextrose & child should recover quickly (w/in hrs), usually w/no residual damage.
Metabolic acidosis	1. IV glucose bolus as above 2. If pH remains <7.1 or worsens, administer $NaHCO_3$ as 1/2 the calculated dose over a 30-min period. 3. Restrict fructose, glycerol, sucrose, & sorbitol.	• Acidosis usually corrects quickly w/out $NaHCO_3$ infusion. • No consensus exists re restriction of dietary fructose & sucrose.
Hepatomegaly & ↑ transaminases	None	Transient findings that resolve spontaneously

Bijarnia-Mahay S, Bhatia S, Arora V. Fructose-1,6-Bisphosphatase Deficiency. 2019 Dec 5. In: Adam MP, Feldman J, Mirzaa GM, et al., editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2024. Available from: https://www.ncbi.nlm.nih.gov/books/NBK550349/

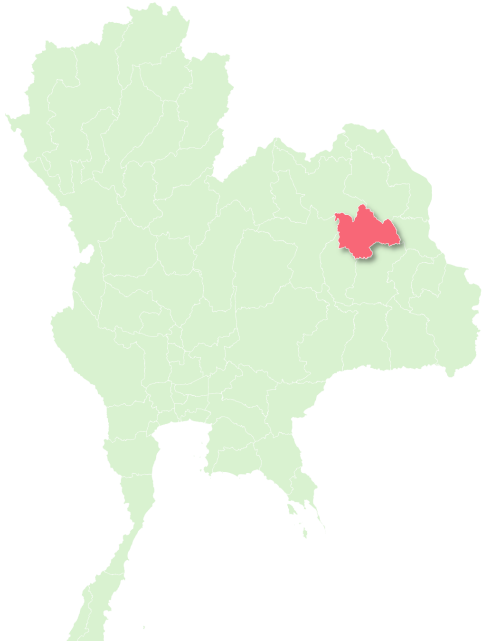
Case IV

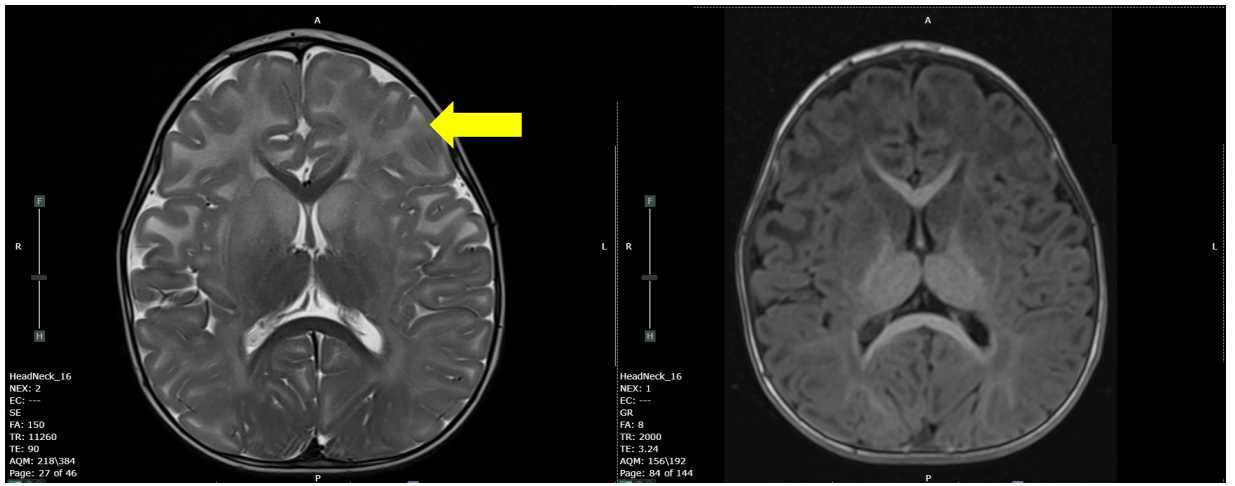
- เด็กชายอายุ 1 ปี มีปัญหาพัฒนาการถดถอย ชัก
- ร่วมกับศีรษะโต HC > P97
- มีอาการสะดุ้งตกใจง่ายเวลาได้ยินเสียงดัง
- ตรวจตา ไม่พบ cherry red spot



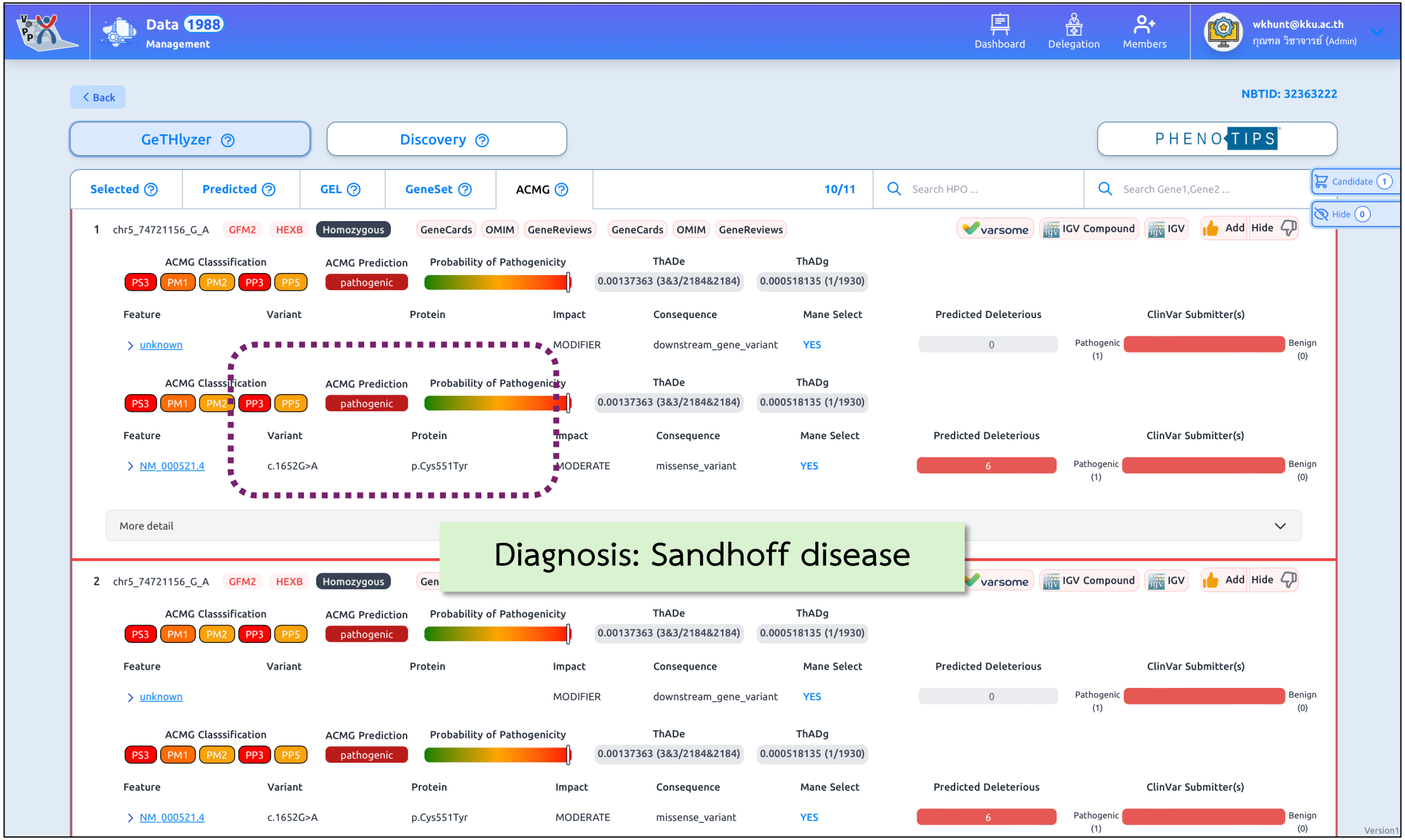


	results	unit	Cut-off value
Hexosaminidase A	0.6	nmol/h	> 0.6
Hexosaminidase total	0.6	nmol/h	> 2.0



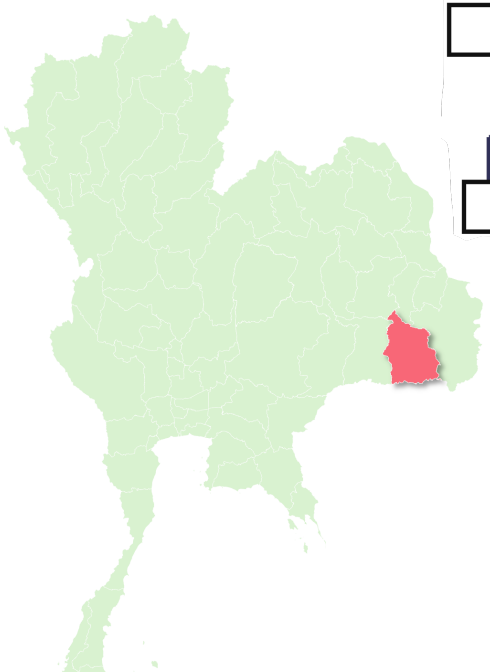


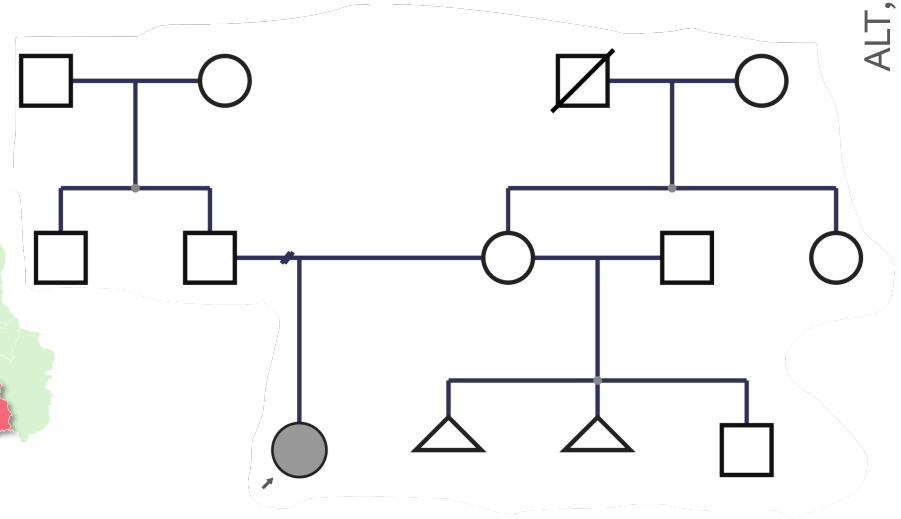
MRI: Symmetrical bilateral hypoS1 T1, hyperT2 change or demyelination at bilateral cerebral white matter and both basal ganglia, involving subcortical U fiber and predominate at both frontal lobe. involve caudate nucleus, internal capsule, putamen involvement, T2 hypotense band at periventricular white matter (ventricular Garlands sign). DDx: Alexander disease

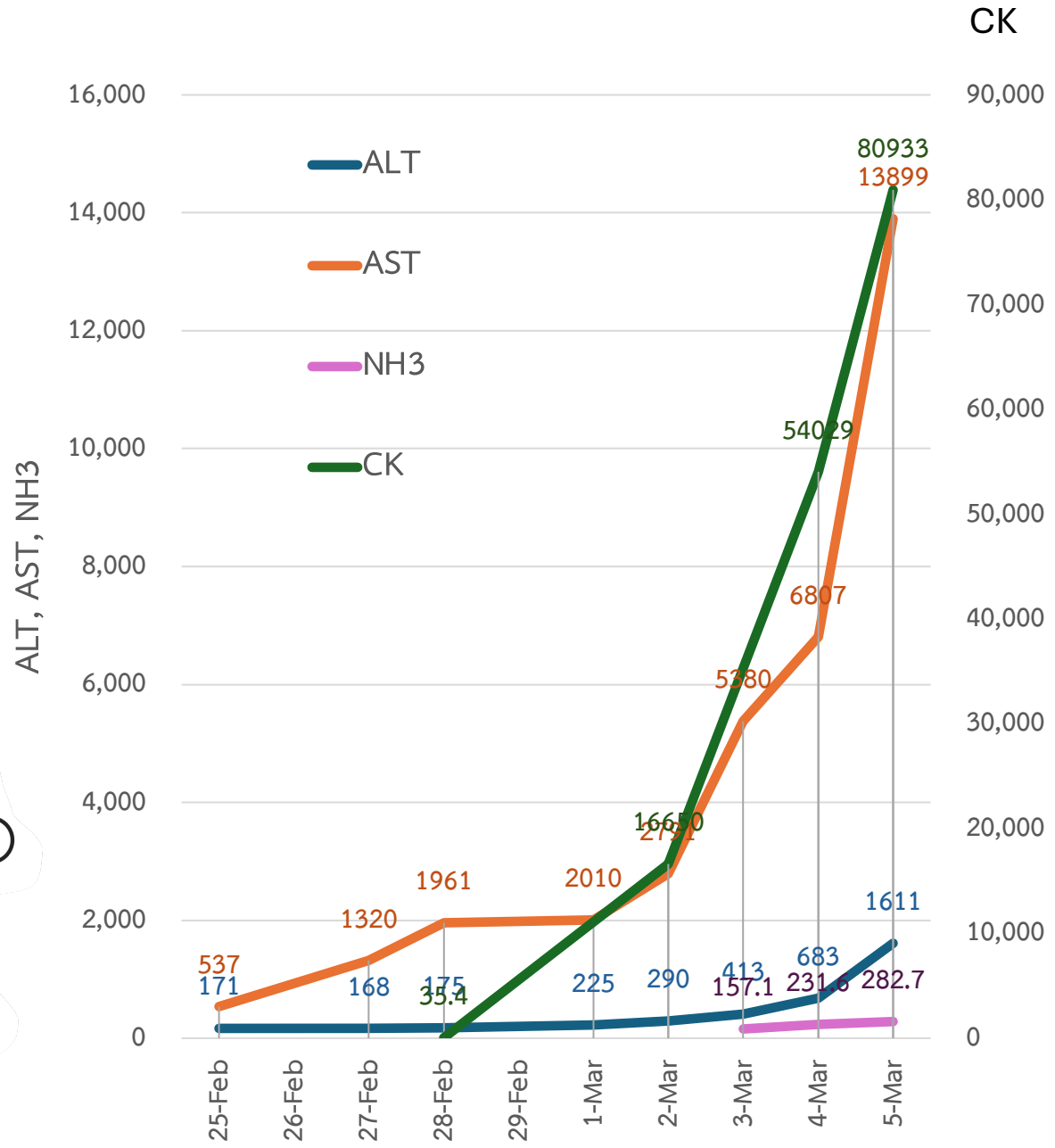

Data 1988
Management
Dashboard
Delegation
Members
wkhunt@kku.ac.th
กุณฑล วิชาจารย์ (Admin)
Back
NBTID: 32363222
GeTHlyzer
Discovery
PHENOTIPS
Selected
Predicted
GEL
GeneSet
ACMG
10/11
Search HPO ...
Search Gene1,Gene2 ...
Candidate 1
Hide 0
1 chr5_74721156_G_A GFM2 HEXB Homozygous
GeneCards OMIM GeneReviews GeneCards OMIM GeneReviews
varsome IGV Compound IGV Add Hide
ACMG Classsification
PS3 PM1 PM2 PP3 PP5
ACMG Prediction
pathogenic
Probability of Pathogenicity
ThADe
0.00137363 (3&3/2184&2184)
ThADg
0.000518135 (1/1930)
Feature Variant Protein Impact Consequence Mane Select Predicted Deleterious ClinVar Submitter(s)
unknown
MODIFIER
downstream_gene_variant
YES
0
Pathogenic (1)
Benign (0)
NM_000521.4
c.1652G>A
p.Cys551Tyr
MODERATE
missense_variant
YES
6
More detail
Diagnosis: Sandhoff disease
2 chr5_74721156_G_A GFM2 HEXB Homozygous
Version1

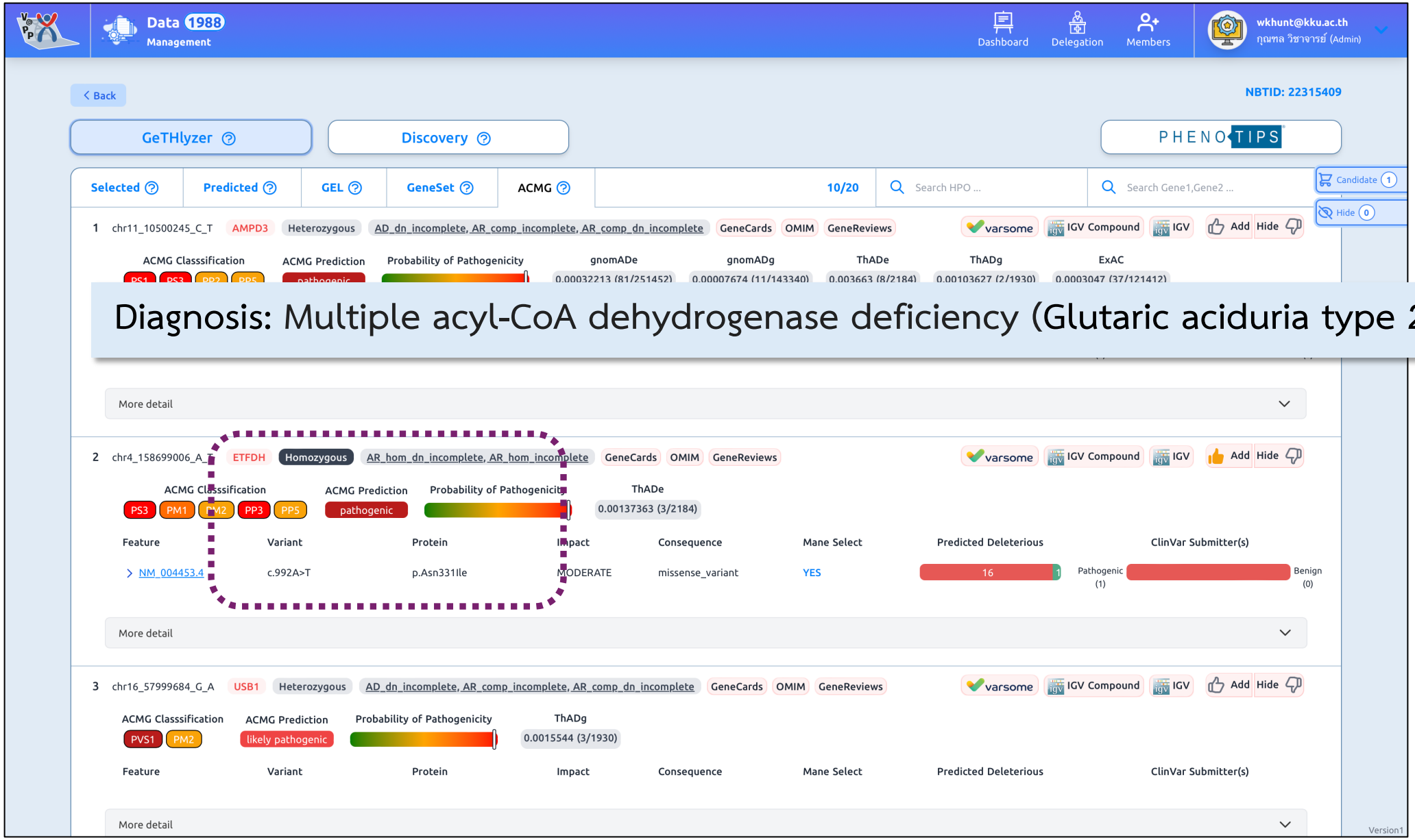
Case V

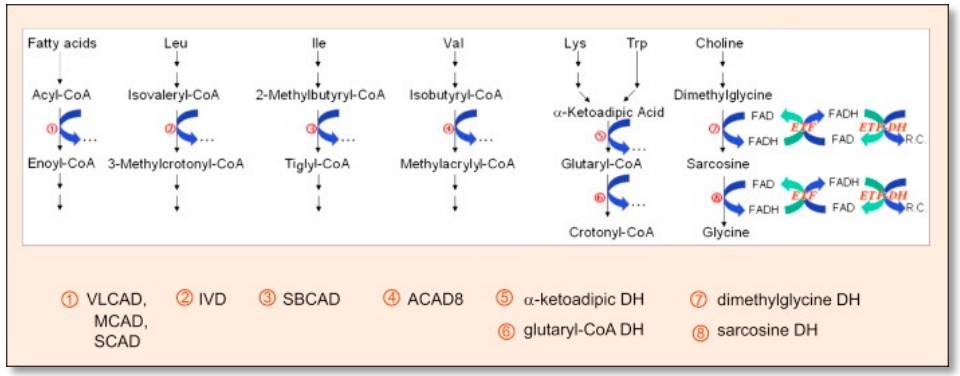
- เด็กหญิงอายุ 14 ปี ไม่มีโรคประจำตัว ถูกส่งตัวมาด้วยปัญหา acute liver failure ไม่ทราบสาเหตุ
- 1 สัปดาห์ก่อนมาโรงพยาบาล มีอาการท้องเสียถ่ายเหลว 4-5 ครั้งหลังกินส้มตำ
- 5 วันก่อนมาโรงพยาบาลมีอาการอ่อนแรง ยกคอไม่ขึ้น ลุกไม่ได้ วินิจฉัย Guillain-Barré syndrome จึง refer มา








Data 1988
Management
Dashboard
Delegation
Members
wkhunt@kku.ac.th
กุณฑล วิชาจารย์ (Admin)
Back
NBTID: 22315409
GeTHlyzer
Discovery
PHENOTIPS
Selected
Predicted
GEL
GeneSet
ACMG
10/20
Search HPO ...
Search Gene1,Gene2 ...
Candidate 1
Hide 0
1 chr11_10500245_C_T AMPD3 Heterozygous AD_dn_incomplete, AR_comp_incomplete, AR_comp_dn_incomplete GeneCards OMIM GeneReviews
varsome IGV Compound IGV Add Hide
ACMG Classsification
ACMG Prediction
Probability of Pathogenicity
gnomADe
gnomADg
ThADe
ThADg
ExAC
0.00032213 (81/251452)
0.00007674 (11/143340)
0.003663 (8/2184)
0.00103627 (2/1930)
0.0003047 (37/121412)
Diagnosis: Multiple acyl-CoA dehydrogenase deficiency (Glutaric aciduria type 2)
More detail
2 chr4_158699006_A_T ETFDH Homozygous AR_hom_dn_incomplete, AR_hom_incomplete GeneCards OMIM GeneReviews
varsome IGV Compound IGV Add Hide
ACMG Classsification
PS3 PM1 PM2 PP3 PP5
ACMG Prediction
pathogenic
Probability of Pathogenicity
ThADe
0.00137363 (3/2184)
Feature
Variant
Protein
Impact
Consequence
Mane Select
Predicted Deleterious
ClinVar Submitter(s)
NM_004453.4
c.992A>T
p.Asn331Ile
MODERATE
missense_variant
YES
16
1
Pathogenic (1)
Benign (0)
More detail
3 chr16_57999684_G_A USB1 Heterozygous AD_dn_incomplete, AR_comp_incomplete, AR_comp_dn_incomplete GeneCards OMIM GeneReviews
varsome IGV Compound IGV Add Hide
ACMG Classsification
PVS1 PM2
ACMG Prediction
likely pathogenic
Probability of Pathogenicity
ThADg
0.0015544 (3/1930)
Feature
Variant
Protein
Impact
Consequence
Mane Select
Predicted Deleterious
ClinVar Submitter(s)
More detail
Version1

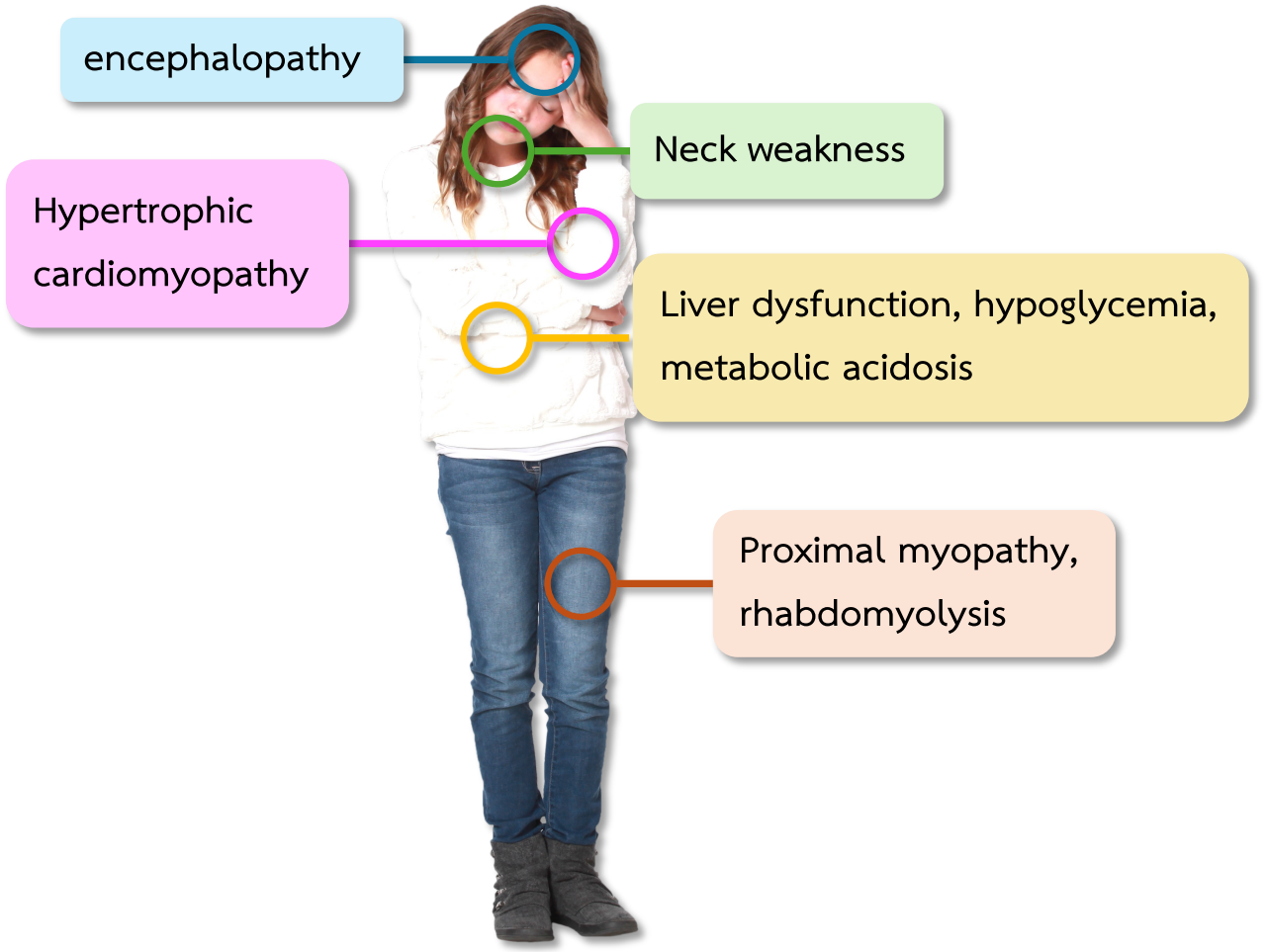


El-Gharbawy A, Vockley J. Chapter 14 - Nonmitochondrial Metabolic Cardioskeletal Myopathies. In: Jefferies JL, Blaxall BC, Robbins J, Towbin JA, editors. Cardioskeletal Myopathies in Children and Young Adults [Internet]. Boston: Academic Press; 2017. p. 265–303. Available from: https://www.sciencedirect.com/science/article/pii/B9780128000403000145

Table 1 Clinical information on 350 patients with late-onset MADD published in the literature

Sex	Female n = 144, male n = 177, not reported n = 29
Identified by newborn screening	2.0% (7/350) patients
Identified by family screening	1.4% (5/350) patients
Acute symptoms	33.1% (111/335) of patients
Chronic symptoms	85.3% (291/341) of patients
Acute and chronic symptoms	20.4% (68/333) of patients
Mean age at onset of symptoms	19.2 years (n = 273)
Mean diagnostic delay	3.9 years (0-29 years)
Asymptomatic	2.6% (9/349) of patients
Deceased patients	5.2% (18/349) of patients
Riboflavin responsiveness	98.4% (256/260) of patients

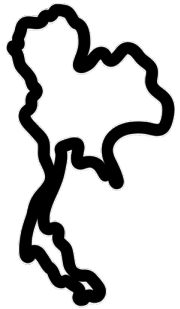
Grünert SC. Clinical and genetical heterogeneity of late-onset multiple acyl-coenzyme A dehydrogenase deficiency. Orphanet J Rare Dis. 2014 Jul 22;9:117. doi: 10.1186/s13023-014-0117-5. PMID: 25200064; PMCID: PMC4222585.



~98% of persons with late-onset MADD respond to riboflavin (100-300 mg/day).

Prasun P. Multiple Acyl-CoA Dehydrogenase Deficiency. 2020 Jun 18. In: Adam MP, Feldman J, Mirzaa GM, et al., editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2024. Available from: https://www.ncbi.nlm.nih.gov/books/NBK558236/

Challenges in implementing WGS from the Genomics Thailand project into healthcare services in Thailand.

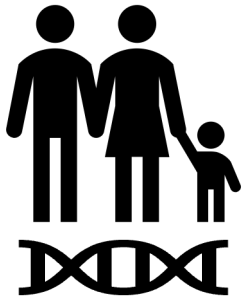


Coverage

Cost

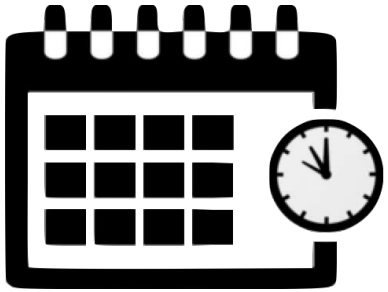
Privacy

Ethics



Genetic counseling

Analysis



TAT

Thank you


GENOMICS
THAILAND


สวทช
NSTDA


มหาวิทยาลัยขอนแก่น
๒๕๑๕
คณะแพทยศาสตร์


HSRI
สถาบันวิจัยระบบสาธารณสุข


กระทรวงสาธารณสุข
MINISTRY OF PUBLIC HEALTH
กรมวิทยาศาสตร์การแพทย์
Department of Medical Sciences