Supplementary Table S1 Summary of clinical and genetic aspects of the studied sample with childhood-onset demyelinating CMT ($n = 32$)

Pt (subtype; gene)	Age at onset (y)	Gender	Age at genetic diagnosis (y)	Age at NCS diagnosis (y)	Current age (y)	Pathogenic variants	Consanguinity	Clinical course	Severity (CMTNsV2)
Pt1 (CMT1A; *PMP22*)	4	M	15	11	18	*PMP22* duplication	No	P	15
Pt2 (CMT1A; *PMP22*)	7	M	31[a]	33	34	*PMP22* duplication	No	S	19
Pt3 (CMT1A; *PMP22*)	4	M	36[a]	37	39	*PMP22* duplication	No	S	20
Pt4 (CMT1A; *PMP22*)	1	M	25	25	28	*PMP22* duplication	No	P	18
Pt5 (CMT1A; *PMP22*)	4	M	11	8	14	*PMP22* duplication	No	S	11
Pt6 (CMT1A; *PMP22*)	6	F	41	39	44	*PMP22* duplication	No	P	20
Pt7 (CMT1A; *PMP22*)	3	F	63	60	66	*PMP22* duplication	No	P	24
PPt8 (CMT1A; *PMP22*)	7	M	9[a]	10	12	*PMP22* duplication	No	P	20
Pt9 (CMT1A; *PMP22*)	17	F	21[a]	22	24	*PMP22* duplication	No	P	9
Pt10 (CMT1A; *PMP22*)	5	M	19	19	21	*PMP22* duplication	No	S	17
Pt11 (CMT1A; *PMP22*)	3	M	15[a]	16	18	*PMP22* duplication	No	S	16
Pt12 (CMT1A; *PMP22*)	1	M	6	5	9	*PMP22* duplication	No	P	20
Pt13 (CMT1A; *PMP22*)	1	F	26[a]	27	29	*PMP22* duplication	No	P	16
Pt14 (CMT1A; *PMP22*)	4	M	25[a]	29	29	*PMP22* duplication	No	P	19
Pt15 (CMT1A; *PMP22*)	8	F	13[a]	14	16	*PMP22* duplication	No	P	20
Pt16 (CMT1A; *PMP22*)	7	F	45[a]	46	48	*PMP22* duplication	No	S	27
Pt17 (CMT1A; *PMP22*)	6	F	53[a]	54	56	*PMP22* duplication	No	S	22
Pt18 (CMT1A; *PMP22*)	1	M	18	5	21	*PMP22* duplication	No	P	19
Pt19 (CMTX1; *GJB1*)	14	M	30	30	33	c.281A > G; p.His94Arg	No	P	23
Pt20 (CMTX1; *GJB1*)	5	M	68[a]	70	71	c.494T > A; p.Leu165Gln	No	S	28
Pt21 (CMTX1; *GJB1*)	5	M	46	48	49	c.164C > T; p.Thr55Ile	No	S	22
Pt22 (CMTX1; *GJB1*)	6	F	71[a]	73	74	c.494T > A; p.Leu165Gln	No	S	23
Pt23 (CMTX1; *GJB1*)	5	M	42[a]	43	45	c.37G > A; p.Val13Met	No	S	25
Pt24 (CMT1B; *MPZ*)	2	F	31	30	34	c.188_190delCCT; p.Ser64del	No	P	34
Pt25 (CMT1B; *MPZ*)	3	M	15	13	18	c.393C > G; p.Asn131Lys	No	P	26
Pt26 (CMT1B; *MPZ*)	14	F	48[a]	51	51	c.699_702delTGAG; p.Ser233Argfs*18	No	S	32
Pt27 (CMT4J; FIG4)	5	M	17[a]	18	20	c.1141C > T (p.Arg381*); c.122T > C (p.Ile41Thr)	No	P	24
Pt28 (CMT4J; *FIG4*)	15	F	58	58	61	c.122T > C (p.Ile41Thr); c.262C > T (p.Arg88*)	Yes	P	16

Supplementary Table S1 (*Continued*)

Pt (subtype; gene)	Age at onset (y)	Gender	Age at genetic diagnosis (y)	Age at NCS diagnosis (y)	Current age (y)	Pathogenic variants	Consanguinity	Clinical course	Severity (CMTNsV2)
Pt29 (CMT4J; FIG4)	13	F	51	51	54	c.122T > C (p.Ile41Thr); c.262C > T (p.Arg88*)	Yes	P	21
Pt30 (CMTRIC; *PLEKHG5*)	12	F	20	20	23	c.985–2A > G; splicing acceptor	No	P	27
Pt31 (CMT4C; *SH3TC2*)	5	F	38[a]	39	41	c.2860C > T (p.Arg954*); c.3511C > T (p.Arg1171Cys)	No	P	19
Pt32 (CMT4F; *PRX*)	1	F	52[a]	54	55	c.3286_3356del71 (p.Ile1096Trpfs*18); c.3685C > T (p.Arg1229*)	No	S	21

Abbreviations: CMT, Charcot–Marie–Tooth's disease; CMTNsV2, CMT neuropathy score,?second version; NCS, nerve conduction studies; P, progressive; Pt, patient; S, stable.
[a]Some patients had genetic diagnosis prior to definite neurophysiological diagnosis both due to low-quality previous NCS examinations and previous known genetic basis of CMT in the family.
[b]Patients with autosomal recessive CMT subtypes were compound heterozygous or homozygous with biallelic disposition of their variants in the patients, confirmed by analyses of their relatives.

