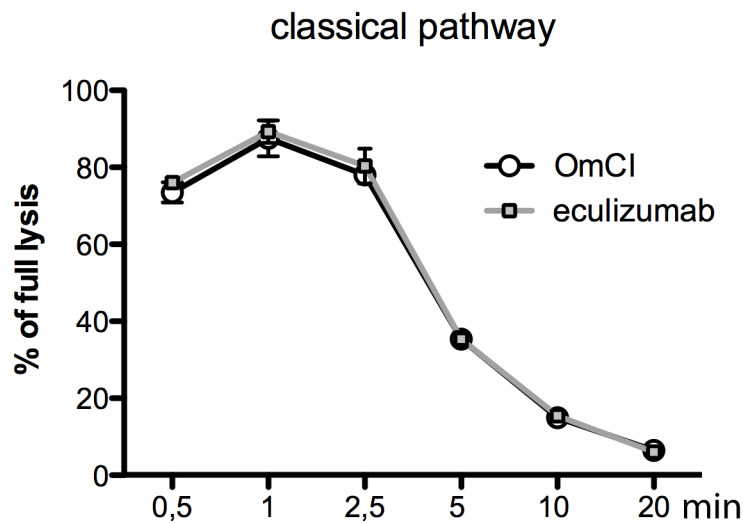
Table 1. Detailed characteristics of clinical samples used in the study.

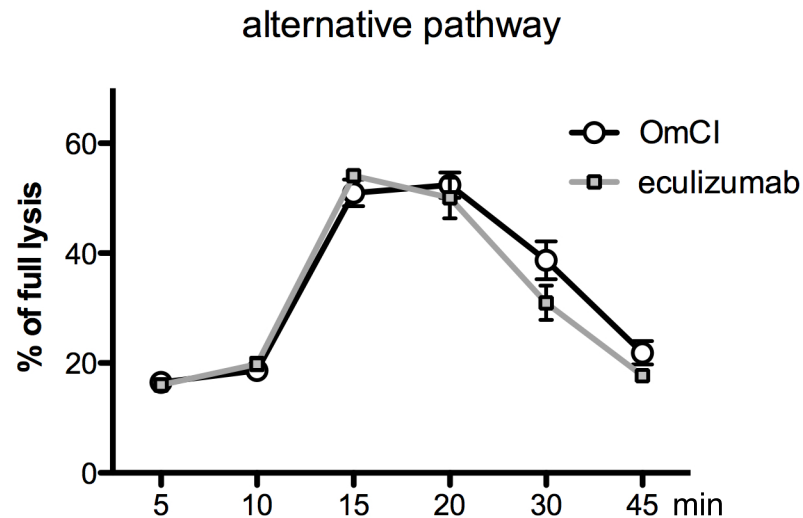
Sample id	Diagnosis	Sex (M/F)	Age	FHaAbs content	Hemolysis in modified Rother's assay [2]	Mutation in complement proteins
GN23	DDD	F	14 years	-	39%	-
GN32	DDD	F	17 years	-	50%	-
GN39	MPGN type I	F	35 years	-	42%	-
IND4	Renal cortical necrosis, meningococcal meningitis	M	19 years	-	26%	-
#1	aHUS	F	11 years	+ 0.482 [1]	-	-
#2	aHUS	M	8 years	+ 1.920 [1]	-	-
#3	aHUS	M	5 years	+ 0.518 [1]	-	-
#4	aHUS	F	9 months	+ 0.415 [1]	-	-
#5	aHUS	M	16 years	+ 0.444 [1]	-	C3 p.Lys65Gln
#6	aHUS	M	13 years	+ 0.177 [1]	-	-
#7	aHUS	M	59 years	-	-	C3 p.Arg161Trp
#8	aHUS	F	46 years	-	-	FH p.Arg1206Cys
#9	aHUS	F	21 years	-	-	FB p.Lys323Glu
#10	aHUS	M	11 years	-	-	FH p.Arg1215Gln
#11	aHUS	F	21 years	> 1000 r.u. [3]	-	C3 p.Lys633Arg

[1] ELISA to detect anti-FH autoantibodies was based on the previously described method (reference 36). Mean and SD of the optical density values of 16 healthy negative controls were calculated. Then, from each patient sample mean+2SD of the controls was subtracted. Patients with results of the calculation >0 were considered positive for anti-FH autoantibodies. Analysis was replicated in at least three independent experiments.

[2] assay described in reference [10]

[3] assay described in reference [38]





intra-assay CV (%)

OmCI	3.59	5.34	2.21	3.38	8.47	4.05	8.78	4.09	4.71	4.39	8.93	9.79
eculizumab	0.99	3.20	5.67	5.22	4.70	6.02	6.59	5.67	1.87	7.39	10.03	2.78

Supplementary Fig.1 Intra-assay (intra CV) coefficient of variation of Tmax curves. Tmax curve for classical pathway (left panel) and alternative pathway (right panel) was calculated from four repetitions. Classical pathway convertase was formed from 0.5% NHS blocked with 12.5 nM OmCI or 2 nM eculizumab. Alternative pathway convertase was formed from 5% NHS blocked with 100 nM OmCI or 60 nM eculizumab.