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HLA TYPING IN HYPERSOMNOLENCE IN ASSOCIATION WITH REM SLEEP WITHOUT ATONIA AND REM SLEEP BEHAVIOUR DISORDER

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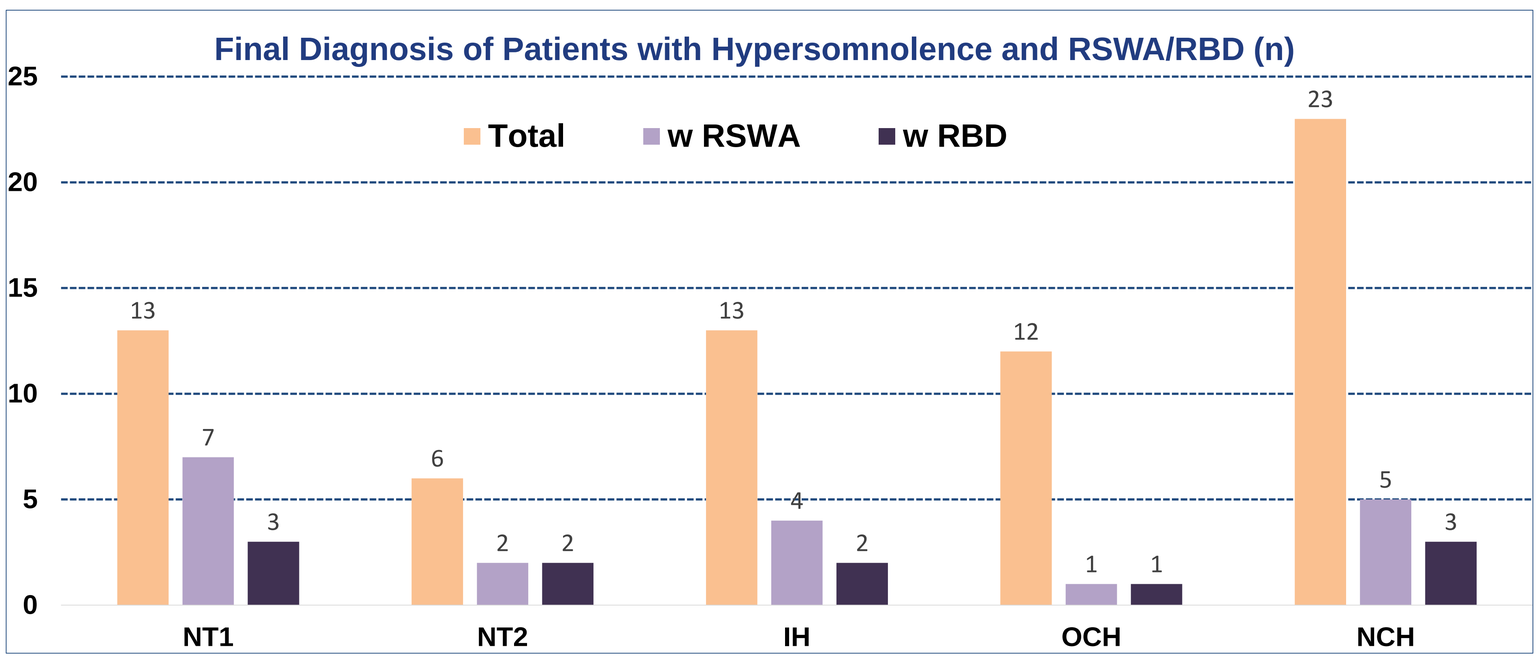
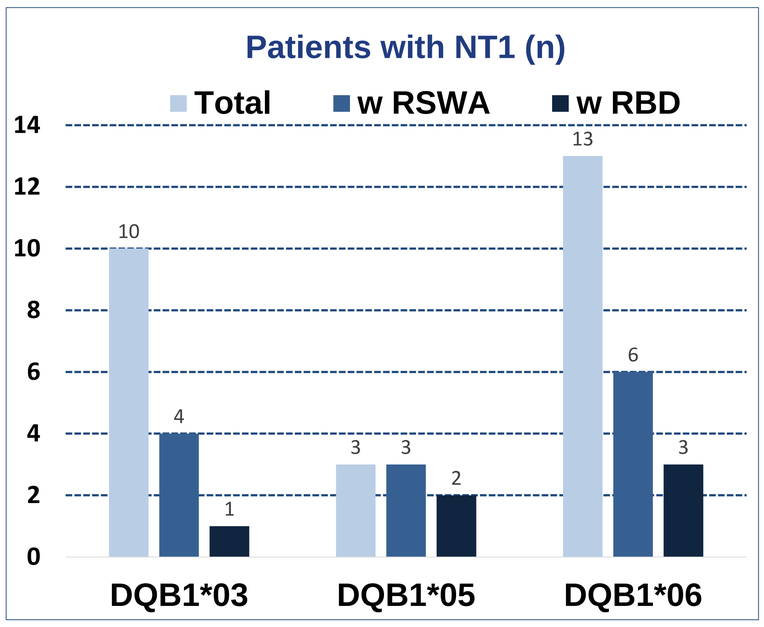
INTRODUCTION & METHOD

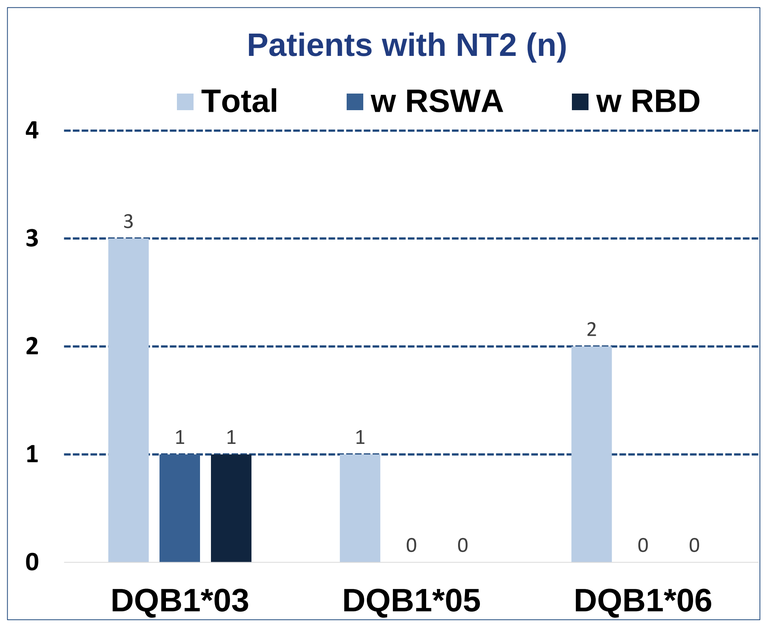
Narcolepsy type 1 (NT1) is caused by the disruption in hypothalamic hypocretinergic system, and associated with human leukocyte antigens (HLA), specifically DQB1 [1]. HLA may play different roles in rapid eye movement (REM) sleep dysregulation besides narcolepsy, and it appears to be associated with HLA class II genes, mainly DQB1*05,06 [2]. REM-sleep without atonia (RSWA) / REM-sleep behavior disorder (RBD) frequently accompany narcolepsy [3]. The significance of HLA locus in RSWA/RBD associated with narcolepsy or other hypersomnolences awaits to be explored. The primary aim of this study to demonstrate the HLA locus distribution in different central disorders of hypersomnolences with and without RSWA/RBD.

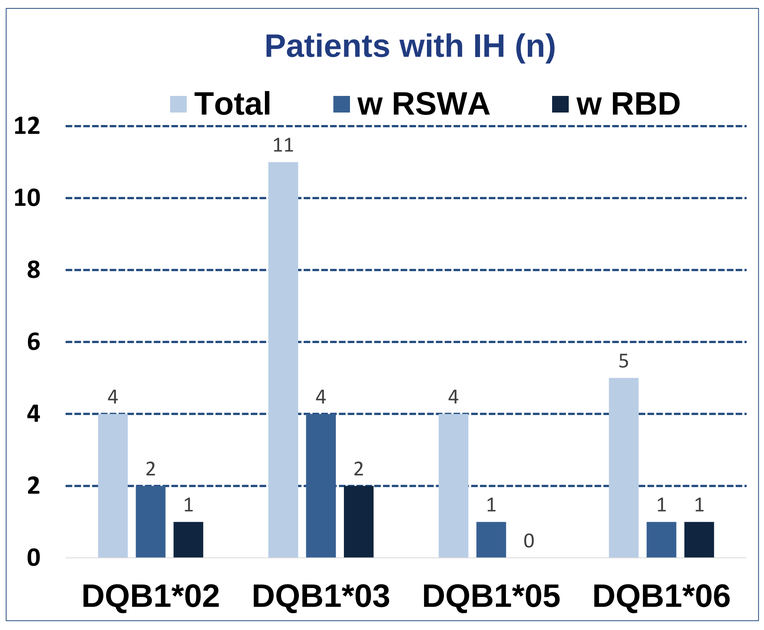
Patients with excessive daytime sleepiness were consecutively enrolled for one year. Demographics, polysomnography, multiple sleep latency test and HLA typing were documented in all and those having missing data were excluded. HLA subtyping included DQB1*02,03,04,05 and 06. The final diagnoses were grouped as NT1, NT2, idiopathic hypersomnia (IH), other central hypersomnias (OCH) and non-central hypersomnias (NCH).

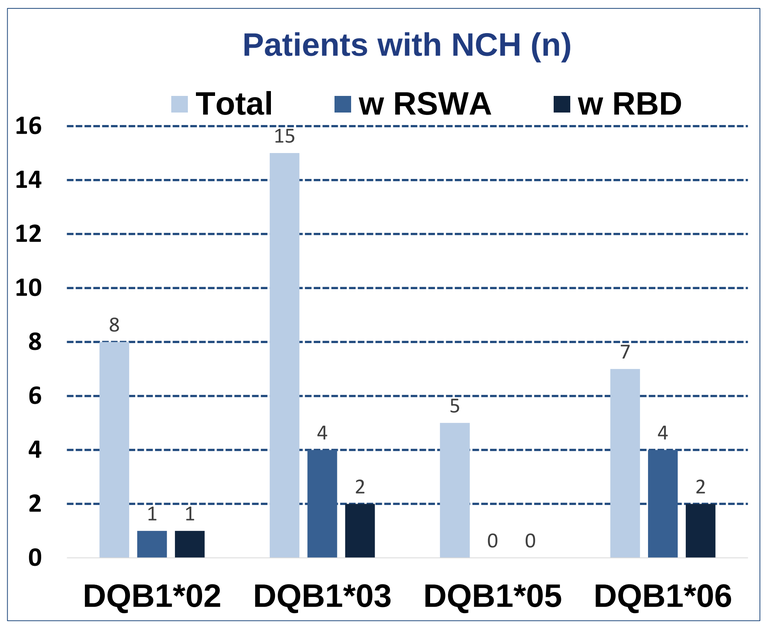
RESULTS











A total of 67 subjects, F/M:42/25, mean age:35.9±12.4 years RSWA was observed in 20/67 (29.8%), and 11/20 had RBD.

HLA distribution in different types of hypersomnias according to the presence/absence of RWA was listed as follows:

- **NT1:** All patients with NT1 had HLA-DQB1*06 locus (with overlapping DQB1*03 and *05 loci). Of these, seven (53.8%) had RSWA (three had RBD).
- **NT2:** Five patients with NT2 had at least one HLA (DQB1*03, DQB1*05, DQB1*06). Two patients with NT2 (33.3%) had RSWA/RBD, and only one had one HLA (DQB1*03).
- **IH:** In patients with IH, interestingly, all had at least one HLA (DQB1*02, DQB1*03, DQB1*05, DQB1*06). Four (30.7%) had RSWA (2 with RBD), and all IH patients with RSWA had at least two HLA loci.
- **OCH:** Except for one patient with RS-who had RBD and DQB1*03, none of the patients with OCH had RSWA/RBD.
- **NCH:** In NCH group, five patients had RSWA (3 with RBD), and all of them had at least one HLA (DQB1*02, DQB1*03, DQB1*06).

Nine patients (45%) out of 20 patients with RSWA had DQB1*03 and DQB1*06 loci together.

Three patients had DQB1*05 and DQB1*06, and another three had DQB1*03 and *05 loci together

CONCLUSION

Preliminary results of our study revealed that HLA loci were frequently present in hypersomnolences. RSWA/RBD was also common in these patients. Although the presence/absence of RWA/RBD did not cause a significant difference on HLA distribution in central hypersomnia cases in this study population; possible shared pathogenesis seems to be related to HLA class II genes, which have a potential association with RSWA/RBD.

REFERENCES & ACKNOWLEDGEMENTS

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