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Accuracy of the Babinski sign in the identification of pyramidal tract dysfunction

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ABSTRACT

Background: The extensor plantar response described by Joseph Babinski (1896) indicates pyramidal tract dysfunction (PTD) but has significant inter-observer variability and inconsistent accuracy. The goal of this study was to determine the accuracy of the Babinski sign in subjects with verified PTD.
Methods: We studied 107 adult hospitalized and outpatient subjects evaluated by neurology. The reference standard was the blinded and independent diagnosis of an expert neurologist based on anamnesis, physical examination, imaging and complementary tests. Two neurologists elicited the Babinski sign in each patient independently, blindly and in a standardized manner to measure inter-observer variability; each examination was filmed to quantify intra-observer variability.
Results: Compared with the reference standard, the Babinski sign had low sensitivity (50.8%, 95%CI 41.5–60.1) but high specificity (99%, 95%CI 97.7–100) in identifying PTD with a positive likelihood ratio of 51.8 (95%CI 16.6–161.2) and a calculated inter-observer variability of 0.73 (95%CI 0.598–0.858). The intraevaluator reliability was 0.571 (95%CI 0.270–0.873) and 0.467 (95%, CI 0.019–0.914) respectively, for each examiner.
Conclusion: The presence of the Babinski sign obtained by a neurologist provides valid and reliable evidence of PTD; due to its low sensitivity, absence of the Babinski sign still requires additional patient evaluation if PTD is suspected.

1. Introduction

According to Goetz, [1] the Babinski sign or extensor plantar response was first described in 1896 by the French neurologist of Polish ancestry Joseph F.F. Babinski to indicate the presence of pyramidal tract dysfunction (PTD). Subsequently, Babinski named his sign *le signe de l'éventail* (a hand-held fan) and characterized it by "fanning of the toes followed by slow extension of the big toe." This sign allowed him to separate organic hemiplegia from hysterical paralysis [2]. The Babinski sign probably results from loss of inhibition of the spinal flexor reflex modulated by the supplementary motor area [3]. Plantar stimulation, i.e., rubbing of the lateral aspect of the sole of the foot lineally from the heel forward with a blunt object, causes dorsiflexion and abduction of the toes, contraction of the extensor hallucis longus with extension of the big toe, and foot dorsiflexion from activation of anterior tibialis and tensor fasciae latae muscles. Isolated extension of the big toe without the abduction of toes may occur predominantly with cortical pyramidal tract lesions. The signs described by Chaddock [1], Gordon, and Oppenheim, are complementary but can occur independently.

The performance of the Babinski sign is variable, due in part to non-compliance with methodological STARD requirements for the studies of diagnostic accuracy [4]; reported sensitivity range is 35–90%, specificity 77–99%, inter-observer agreement 0.09 to 0.3 and intra-observer concordance 0.59. The purpose of our study was to determine the diagnostic accuracy of the Babinski sign to detect PTD.

Abbreviations: PTD, pyramidal tract dysfunction; CI, confidence interval; Bp, Babinski sign present; Ba, Babinski sign absent; STARD, Standards for the reporting of diagnostic accuracy studies.

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2. Material and methods

2.1. Patient selection

After signing informed consent we recruited 107 consecutive patients ≥18 years-old suspected of having PTD due to limb weakness of any degree, acute or chronic, and admitted to University Hospital, Medellin, between 2009 and 2010. Inclusion criteria required recent neuroimaging of brain or spinal cord. Exclusions included coma, lower limb amputation, foot wounds, plantar hyperesthesia or hyperkeratosis. The study was approved by the University Hospital Ethics Committee. Patients' privacy, data confidentiality and security were guaranteed. Sample size was calculated at N = 107 (supplementary electronic data). Each patient was assigned a random identification number for blinding purposes. Two junior neurologists, independently, without knowledge of clinical history or test results elicited the Babinski sign using the standardized method described below. No clinical changes occurred between these 2 evaluations separated by <6 h. The subject was placed in a cubicle, with curtains and linens set so that only the lower limbs were visible up to the knee with the patient in a supine, relaxed position, with the head centered and the knees extended. The Babinski sign was elicited using the tip of a Queen Square hammer to stimulate the external aspect of the sole starting at the heel and following a transverse arch line along the sole with a single and firm movement without touching the hallux base. Examiners could repeat the stimulation up to 3 times. The response of the hallux and toes was recorded as extensor, flexor or neutral for each foot. The presence of an extensor response was considered a Babinski sign; and neutral and flexor responses were considered as absent Babinski sign. The procedure was filmed from the neurologist's visual angle; this film did not have audio and was identified with the patient's random number. Each neurologist reviewed the film at least 30 days later to judge the plantar response, which was then compared with the initial one. A senior neurologist (C.S.U.) examined every patient ≤6 h later to provide the diagnostic reference standard independently, blinded from the previous results, and based on history, examination, laboratory tests and neuroimaging; this evaluation concluded on the absence or presence of PTD and the side(s) affected.

2.2. Data analyses

We analyzed patients' age, gender, diagnosis, reference standard for PTD (N = 214), Babinski sign present (Bp), absent (Ba), neutral or flexor, for the 3 neurologists and for each side (N = 428). We used Kolmogorov–Smirnov test to define age distribution and Mann–Whitney's *U* test when age distribution was not normally distributed by gender and PTD. Other variables were presented as proportions; Chi^2 or Fisher's test were used for the comparison between groups of patients with or without PTD. The ROC curve and area under the curve were calculated. For inter-/intra-observer agreement we used the Kappa index (≤0.4 = minimal agreement, 0.4–0.6 = average, ≥0.6–0.8 = moderate, ≥0.8 = excellent concordance). A statistical significance level of $p < 0.05$ was used and we calculated 95% confidence intervals (CI). SPSS for Windows (version 15 SPSS Inc.) and EPIDAT 3.1 were used.

3. Results

The subjects' mean age was 56 years (75% >40 years) with men predominance (59%) but women were older than men ($p = 0.007$). Most (80%) were admitted to neurology, 16% to neurosurgery, and 4% to medicine; 57% had no PTD and the remainder had unilateral or bilateral injury. Table A.1 summarizes the diagnoses of the patients in the study. Table B.1 shows the performance of the Babinski sign. We compared the reference standard with Babinski sign results obtained by either neurologist 1 or 2 (Table C.1 in supplementary electronic data). Kappa index indicated a moderate concordance (Table C.2 in supplementary electronic data).

The Babinski sign's sensitivity was 54.1% and 47.5% for each neurologist indicating modest probability of correctly classifying an individual without PTD but the specificity was very high (98.7% and 99.3%); i.e., Babinski sign present indicates high probability of correctly classifying an individual with PTD. The positive likelihood ratio was 41.4 and 72.1 indicating higher likelihood of obtaining Babinski sign in the presence of PTD than in someone without PTD (Table B.1).

The Babinski sign has low sensitivity but very high specificity indicating a high accuracy to properly classify an individual with PTD when the sign is present. The positive likelihood ratio was high, i.e. more likely to occur in PTD; positive predictive value was higher than its negative predictive value, i.e., there was high certainty of having PTD in the presence of Babinski sign.

The accuracy of the Babinski sign by each examining neurologist was similar in the area under the ROC curve, for neurologist #1 was 0.764 (CI 95% 0.681–0.846) and for neurologist #2 was 0.734 (CI 95% 0.649–0.820). The accuracy of the sign to discriminate patients with PTD or not had moderate area under the ROC curve 0.749 (CI 95% 0.690–0.809). Comparison of areas under the curve shows no major difference according to the neurologists' experience.

4. Discussion

We demonstrate that the Babinski sign has a very high specificity (99%) and low sensitivity (51%) with reliable confidence intervals indicating that these results are accurate. In compliance with methodological STARD requirements we calculated the sample size for specificity; therefore, other results must be assumed with care (supplementary electronic data). Higher sensitivity was reported by Miller and Johnston (56%) in only 10 subjects, [5] and by Ghosh and Pradhan (75%) in spastic children [6]. Using a standardized method we obtained better concordance values than Miller and Johnston [5], Maher et al. [7] and Singerman and Lee [8], probably due to undefined examiners' experience, and lack of blinding and standardization in those studies. Our intra-observer variability was similar to that of Maher et al. [7]. We found higher positive likelihood ratio than Cook et al. [9], in cervical myelopathy patients.

5. Conclusions

Our study is based on acute, hospitalized patients; therefore, the performance of the Babinski sign in chronic conditions and in other age groups should be considered separately. When obtained in a standard manner by a neurologist, the Babinski sign is a valid, accurate, and reliable test for the diagnosis of PTD. However, if the Babinski sign is absent, the patient may have PTD and requires additional diagnostic evaluation.

Conflicts of interest

The authors report no conflicts of interest and no industry sponsor. Accuracy of the Babinski sign in the identification of pyramidal tract dysfunction.

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Appendix A. Tables

Table A.1
Diagnosis of the patients in the study.

Cause	Proportion (%)
Ischemic stroke (there were more women than men p = 0.04)	35.4
Seizures and epilepsy (most frequently symptomatic for metabolic disorders, neurocysticercosis, alcohol suppression, systemic infection, aftermath of trauma or meningitis)	18.6
Tumors (meningiomas and metastases)	10.1
Neurological infections	6.4
Dementia and delirium (vascular dementia most frequently; delirium most frequently urinary infection)	12.9
Others (firearm bullet wounds, hydrocephalus, chronic lithium intoxication, acute disseminated encephalomyelitis, trigeminal neuralgia, Guillain–Barré syndrome, spinal and cranial trauma)	16.6

Table B.1
Performance of the Babinski sign.

Characteristics of Babinski sign	Values for neurologist 1 (95% CI)	Values for neurologist 2 (95% CI)	Total value (95% CI)
Sensitivity	54.1% (40.77–67.42)	47.54% (34.19–60.89)	50.82% (41.54–60.10)
Specificity	98.69% (96.57–100)	99.35% (97.74–100)	99.02% (97.75–100)
Positive predictive value	94.29% (85.17–100)	96.67% (88.58–100)	95.38% (89.51–100)
Negative predictive value	84.36% (78.76–89.96)	82.61% (76.87–88.36)	83.47% (79.51–87.43)
Positive likelihood ratio (LR+)	41.39 (10.25–167.17)	72.14 (10.13–522.23)	51.84 (16.59–161.99)
Negative likelihood ratio (LR−)	0.47 (0,35–0,61)	0.53 (0.42–0.67)	0.5 (0.41–0.60)
Validity index	85.98 (81.1–90.87)	84.58 (79.51–89.65)	85.28 (81.81–88.75)
Reliability (kappa index)	0.5714 (0.2701–0.8728)	0.4667 (0.0194–0.9139)	0.728 (0.5984–0.8580)
Area ROC curve (Receiver operating characteristic)	0.764 (0.681–0.846)	0.734 (0.649–0.820)	0.749 (0.690–0.809)

Appendix B. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.jns.2014.05.028.

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