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ORIGINAL COMMUNICATION

Diagnostic accuracy of acute vestibular syndrome at the bedside in a stroke unit

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Abstract Acute vestibular syndrome may be due to vestibular neuritis (VN) or posterior circulation strokes. Bedside ocular motor testing performed by experts is superior to early MRI in excluding strokes. We sought to demonstrate that differentiation of strokes from VN in our stroke unit is reliable. During a prospective study at a tertiary hospital over 1 year, patients with AVS were evaluated in the emergency department (ED) and underwent admission with targeted examination: gait, gaze-holding, horizontal head impulse test (hHIT), testing for skew deviation (SD) and vertical smooth pursuit (vSP). Neuroimaging included CT, transcranial Doppler (TCD) and MRI with MR angiogram (MRA). VN was diagnosed with normal diffusion-weighted images (DWI) and absence of neurological deficits on follow-up. Acute strokes were confirmed with DWI. A total of 24 patients with AVS were enrolled and divided in two groups. In the pure vestibular group ($n = 20$), all VN ($n = 10/10$) had positive hHIT and unidirectional nystagmus, but 1 patient had SD and abnormal vertical smooth pursuit (SP). In all the strokes ($n = 10/10$), one of the following signs suggestive of central lesion was present: negative hHIT, central-type nystagmus, SD or abnormal vSP. Finding one of these was 100% sensitive and 90% specific for stroke. In the cochleovestibular group ($n = 4$) all had normal DWI, but 3 patients had central ocular motor signs (abnormal vertical SP and SD). Whilst the study is small, classification of AVS in our stroke unit is reliable. The sensitivity and specificity of bedside ocular motor testing are comparable to those previously reported by expert neuro-otologists. Acute cochleovestibular loss and normal DWI may signify a labyrinthine infarct but differentiating between different causes of inner ear dysfunction is not possible with bedside testing.

Keywords Acute vestibular syndrome · Vestibular neuritis · Brainstem stroke · Ocular motility, nystagmus, skew deviation, vertigo · Neurological examination

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Introduction

Vertigo is common, accounting for 2.5% of the emergency department (ED) presentations [16] and has 5% 1-year prevalence in the community [26]. Acute vestibular syndrome (AVS) is characterized by acute prolonged rotatory vertigo (>24 hours) with vegetative symptoms, without presence of other brainstem signs [12]. The differential diagnoses lies between vestibular neuritis (VN) and posterior circulation stroke. Although fortunately stroke accounts for less than 1% of isolated vertigo presenting to ED [15], the distinction has therapeutic implication. It may be difficult to differentiate between the two at the bedside and no clinical test in isolation has been found to be reliable in distinguishing stroke from VN [4], although the horizontal head impulse test (hHIT) is 100% sensitive for stroke but only about 90% specific [27]. A three-step test performed by neuro-otologists, incorporating hHIT, skew deviation (SD) and determination of directionality of nystagmus, has been shown to be more sensitive than early diffusion-weighted imaging (DWI) in excluding stroke whilst maintaining a high specificity [14]. We hypothesise that classification of AVS is accurate in our stroke unit despite relative inexperience in neuro-otology.

Method

The study was conducted over 1 year at a single tertiary hospital with approximately 700 stroke unit admissions per year, and was approved by the Institutional Review Board. Patients who presented with acute isolated vertigo to the ED were identified by referral. The indications for referral were uncertain diagnosis, presence of vascular risk factors (smoking, hypertension, diabetes, dyslipidaemia, atrial fibrillation and recent neck trauma) and failure of symptom improvement for safe discharge. Those with acute prolonged rotatory vertigo associated with nausea and/or vomiting, without other brainstem signs, were included. Exclusion criteria are presented in Table 1. All patients underwent bedside vestibular testing by one of two examiners (LC or WL) within 12 hours of referral using a 4-step ocular motor signs examination (hHIT, directionality of nystagmus, SD and vertical smooth pursuit). Examiners' exposure to formal neuro-otology teaching consisted of a 3-hour video-based lecture and a 1-hour small group tutorial, covering a broad range of topics including demonstration of hHIT. If a patient could stand from lying down without the aid of an assistant then this was defined as standing unassisted. Head impulses were delivered as described previously [10] with the patient sitting up. SD was detected with alternate cover test and Maddox rod. A stroke was diagnosed at the bedside if any one of the four central ocular motor signs was present: a negative hHIT, "central type" nystagmus, SD or abnormal vertical smooth pursuit. A negative hHIT was defined as absence of observable catch-up saccade, and a positive hHIT the presence of unequivocal observable catch-up saccade. Nystagmus was assessed in the primary position and eccentric gazes with visual fixation. "Central type" nystagmus included direction-changing, gaze-evoked nystagmus and purely vertical/purely torsional nystagmus. Unidirectional nystagmus always beat in the same direction regardless of direction of gaze. CT results were available to examiners at time of referral, but all MRI were performed as inpatients and results were masked until after completion of bedside vestibular testing. All patients underwent MRI according to the in-house stroke protocol (1.5 T and 5 mm slice, DWI, T1, T2, fluid-attenuated inversion recovery and time-of-flight MRA). The reference standard for the diagnosis of an acute stroke was a positive DWI, and for VN, a negative DWI with absence of other neurological signs on daily examination until discharge. Not all patients underwent formal vestibular function testing because of difficulty with timely access. TCDs were performed by experienced sonographers and reported by stroke neurologists with neurosonology expertise (BRC and HMD).

Table 1 Exclusion criteria

Tinnitus
Antecedent viral illness
Prior diagnosis of or attacks suggestive of Meniere's disease
Vestibular migraine (Neuhauser's criteria [25])
Corticospinal tract dysfunction
Appendicular and truncal cerebellar signs
Hemianopia or other visual field defect
Horner's syndrome
Sensory disturbance (facial or limb)
Facial palsy
Bulbar dysfunction and dysarthria
Dense ocular motor signs—3, 4 or 6th nerve palsy, internuclear ophthalmoplegia, gaze palsy

Results

We identified 36 patients and excluded 12 after clinical evaluation (2 vestibular migraine [25], 7 benign paroxysmal positional vertigo, 2 patients with Horner's syndrome and lower cranial nerve palsies and 1 patient with bedside evidence of stroke with negative early DWI). Of 24 patients, 20 reported no hearing loss and 4 presented with deafness. The patient characteristics are summarized in Table 2a, b.

The study consisted of 63% men with a mean age of 64 years (SD 13 years; range 42–83 years). In the stroke group, 40% had less than one risk factor, and 70% in the VN group had more than two risk factors. In every VN patient, the vertigo was the first episode and developed over minutes to hours, but in the stroke group, 20% ($n = 2/10$) had recurrent isolated vertigo over 12–18 months without accompanying neurological deficit during or between attacks. The vertigo was characterised by a sudden onset, lasting 10–20 min with complete resolution each time. All patients presented within 72 hours of vertigo onset, with 71% ($n = 17/24$) presenting within 24 hours.

In the vestibular group, most (17/20, 85%) were imaged within 5 days of symptom onset. No patients had unexplained signs on serial examination and none required a second MRI for this reason. Gait disturbance was present in all (broad-base stance or tandem walk difficulty) with 50% in each group unable to stand unassisted. All patients with VN had unidirectional nystagmus, and unequivocal positive hHIT. Canal paresis was confirmed on caloric testing in 3/3. All strokes had abnormal DWI in the posterior fossa, except one with vertical nystagmus and SD whose acute infarct was in the left periventricular region. None had imaging evidence of mass effect. Pontocerebellar (PC) infarcts ($n = 4$) were the commonest mimicker of VN, followed by inferior cerebellar (IC) ($n = 3$) and lateral

Table 2 Summary of clinicoradiological findings

Patient/Age (y)/Sex	Risk factors	First/ recurrent	Stand unassisted	Nystagmus	hHIT	Vertical SP	SD	TCD	Sx onset to MRI (d)	DWI	MRA
(a) Vestibular presentation											
1/52/F	2	First	Yes	No	Neg	Normal	No	Normal	9	L inf cbm	L VA
2/53/M	1	First	Yes	Static positional CCWTN	Neg	Normal	No	Normal	8	L lat medulla, inf cbm	L PICA
3/60/M	1	First	Yes	Unidirectional LBHN 1°, L	Pos to R	Abnormal	SD	Normal	3	R MCP	Normal
4/45/F	2	Recurrent	Yes	RBHN to R LBHN to L	Neg	Abnormal	No	BA, L VA	3	R MCP, L pons	BA, L VA
5/48/M	1	First	Yes	Unidirectional RBHN to R	Neg	Normal	SD	Normal	2	L lat medulla	Normal
6/79/F	4	First	No	RBHN to R LBHN to L Up/ downbeat	Neg	Abnormal	SD Head tilt	Normal	3	L perivent	Bilat PCA
7/62/M	2	First	No	RBHN to R LBHN to L	Pos to R	Abnormal	No	BA, L VA	5	L pons, cbm/occ	BA, L VA
8/78/M	1	First	No	Unidirectional RBHN to, R	Neg	Abnormal	Head tilt only	Normal	1	L inf vermis, nodulus	Normal
9/83/M	2	First	No	RBHN to R LBHN to L	Neg	Abnormal	No	Normal	5	L pons, inf vermis	Normal
10/70/M	3	Recurrent	No	Unidirectional RBHN 1°, R	Neg	Abnormal	Head tilt only	R VA	5	R inf vermis	R VA
11/59/M	2	First	Yes	Unidirectional	Pos	Abnormal	SD Head tilt	Normal	3	Normal	Normal
12/71/M	3	First	No	Unidirectional	Pos	Normal	No	Normal	5	Normal	Normal
13/42/M	2	First	No	Unidirectional	Pos	Normal	No	Normal	3	Normal	Normal
14/53/M	2	First	No	Unidirectional	Pos	Normal	No	Normal	4	Normal	Normal
15/65/F	2	First	Yes	Unidirectional	Pos	Normal	No	Normal	5	Normal	Normal
16/78/M	3	First	No	Unidirectional	Pos	Normal	No	Normal	6	Normal	Normal
17/64/M	1	First	Yes	Unidirectional	Pos	Normal	No	Normal	5	Normal	Normal
18/47/M	1	First	Yes	Unidirectional	Pos	Normal	No	Normal	2	Normal	Normal
19/77/F	2	First	No	Unidirectional	Pos	Normal	No	Normal	3	Normal	Normal
20/76/F	1	First	Yes	Unidirectional	Pos	Normal	No	Normal	3	Normal	Normal

Table 2 continued

Patient/Age (y)/Sex	Risk factors	First/ recurrent	Stand unassisted	Nystagmus	hHIT	Vertical SP	SD	TCD	Sx onset to MRI (d)	DWI	MRA
(b) cochleovestibular presentation											
21/64/F	1	First	No	Unidirectional RBHN 1°, R	Pos to L	Abnormal	No	Normal	3	Normal	Normal
22/69/M	3	First	Yes	Unidirectional RBHN 1°, R	Pos to L	Abnormal	No	Normal	3	Normal	R PCA
23/67/F	2	First	Yes	Unidirectional LBHN 1°, L	Pos to R	Abnormal	SD Head tilt	Normal	3	Normal	Normal
24/70/F	2	First	Yes	Unidirectional LBHN 1°, L	Pos to R	Normal	No	Normal	7	Normal	Normal

CCWTN Counter-clockwise (from patient's perspective) torsional nystagmus, *LBHN* left beating horizontal nystagmus, *RBHN* right beating horizontal nystagmus, *HSN* head-shaking nystagmus, *lat* lateral, *inf* inferior, *cbm* cerebellum, *MCP* middle cerebellar peduncle, *perivent* periventricular, *occ* occipital, *BA* basilar artery, *VA* vertebral artery

medullary (LM) ($n = 2$). Many (70%) with VN have chronic ischaemic changes with multiple T2 lesions in the periventricular region.

Although the sensitivity of each individual ocular motor sign is variable (Table 3), finding one of the four central ocular motor signs had 100% sensitivity and 90% specificity (Table 4). One patient with VN was mislabeled as a stroke because of the presence of SD. SD was present in 3 patients with stroke (2 PC and 1 LM). Both PC infarcts had positive hHIT, but abnormal vSP, together with either SD or central-type nystagmus differentiated stroke from VN. Two patients underwent audiovestibular function testing. Patient 3 (PC infarct) had positive hHIT but negative vertical canal HIT. Audiogram, canal and otolith functions were normal. Patient 4 (bilateral pontine infarcts) had recurrent vertigo, negative hHIT but positive posterior canal HIT bilaterally. The audiogram was normal but there was bilateral mild canal paresis and absent cervical vestibular-evoked myogenic potential on the right.

Transcranial Doppler (TCD) and MRA were normal in all VN, but TCD missed 70% and MRA 40% of strokes. However, none of the basilar artery or multiple vascular stenoses were missed on TCD. In 3 patients, the MRA revealed vascular stenoses that were not present on TCD. Vertebral artery stenosis ($n = 4$) was the commonest MRA abnormality, followed by basilar artery stenosis ($n = 2$). MRA was normal in 4 patients, with the pons and cerebellum the commonest infarct location ($n = 2$).

In the cochleovestibular group, all presented with sudden onset of vertigo and unilateral deafness over minutes and were imaged within 7 days and had normal DWI. All patients had unidirectional nystagmus and unequivocally positive hHIT, but in 3 patients abnormal smooth pursuit and skew deviation were present. Patients 23 and 24 had pure tone audiogram which showed severe sensorineural hearing loss and this remained unchanged at 3-month follow-up.

Discussion

The distinction of VN from strokes in AVS remains a challenge for frontline clinicians [30] and even general neurologists. In our stroke unit differentiation of stroke from VN was reliable, reproducing previously reported sensitivity and specificity [14].

Studies have suggested that PC-labyrinthine, LM and IC infarcts can mimic VN closely and traditional neurological examination would miss such strokes [14, 21, 27, 32]. A positive hHIT has been suggested as a surrogate for peripheral lesion [2, 9, 31]. It is now known that approximately 10% of posterior circulation strokes have positive hHIT, mostly PC-labyrinthine and IC [27], thought to be

Table 3 Properties of individual ocular motor sign

	Sensitivity			Specificity		
	Current study (%)	Cnyrim et al. [4] (%)	Kattah et al. [14] (%)	Current study (%)	Cnyrim et al. [4] (%)	Kattah et al. [14] (%)
"Central type" nystagmus[a]	56	56	20	100	83	100
Negative hHIT	80	60	93	90	93	100
SD and head tilt	50	–	–	90	–	–
SD only	30	40	25	90	100	96
Vertical SP	70	88	–	90	80	–

[a] Excludes patient 1 who had no nystagmus. "Central type" nystagmus was defined as direction-changing, gaze-evoked nystagmus, or purely vertical/purely torsional nystagmus

Table 4 Properties of multi-stepped ocular motor testing

	Current study		Kattah et al. [14]	
	Normal DWI (%) ($n = 10$)	Abnormal DWI (%) ($n = 10$)	Normal DWI (%) ($n = 25$)	Abnormal DWI (%) ($n = 76$)
"Central" signs[a]	10 ($n = 1$)	100 ($n = 10$)	4 ($n = 1$)	100 ($n = 76$)
"Peripheral" signs[b]	90 ($n = 9$)	0 ($n = 0$)	96 ($n = 24$)	0 ($n = 0$)

[a] Central finding was defined as finding any one of "central type" nysatgmus, skew deviation, negative hHIT or impaired vertical SP in our study, and any one of direction-changing nystagmus, skew deviation or negative hHIT in Kattah et al. [14]

[b] Peripheral finding was defined as finding all of unidirectional nystagmus, absence of skew deviation, positive hHIT and normal vertical SP

due to labyrinthine or root entry zone ischaemia. The presence of SD can correctly predict hHIT-positive AVS as central in nature, though about 3/51 (8%) had the combination of absent SD and positive or untestable hHIT [14]. In our study other signs such as impaired vSP and central-type nystagmus were helpful in the diagnosis of strokes.

Individual canal impulsive testing is valuable in detecting peripheral vestibular loss [5]. Dissociated individual canal impulsive test findings in the face of normal or mildly reduced caloric response may suggest a central localization. In patient 3 vestibular findings may be explained by selective vestibular nuclei rather than labyrinthine ischaemia, even though the inner ear maybe more vulnerable to ischaemia [8, 28]. The sparing of vertical but not the horizontal canal in patient 3 on impulsive testing suggests selective labyrinthine ischaemia is less likely, because the anterior and horizontal canals share the same vascular supply via the anterior vestibular artery, a branch of internal auditory artery [24]. Canal paresis in patient 4 is consistent with previous findings that mild caloric abnormalities are seen in lower brainstem lesion [7].

AICA infarct frequently presents with acute cochleovestibular loss but the absence of other brainstem sign is very rare [18, 20]. DWI can be normal initially in cases of cochleovestibular infarct that subsequently developed into AICA infarcts with additional development of cranial nerve or cerebellar signs [17, 19, 34]. In our study all patients in the cochleovestibular group presented with sudden vertigo and deafness with positive hHIT and unidirectional nystagmus in the presence of normal DWI, suggestive of labyrinthine infarct without evolution into AICA infarct. However, bedside ocular motor testing could not distinguish between the causes of labyrinthine dysfunction, although the presence of skew deviation in one (patient 23) might suggest a central aetiology that was not captured on DWI, and better diagnostic test is needed to confirm this.

There were several limitations in our study. The sensitivity and specificity of ocular motor testing in our hands is similar to those previously reported by experts [4, 14] and should be interpreted with caution. The study population consisted of patients with vascular risk factors and it is perceivable that some VN or mild strokes without risk factors would have been discharged directly by ED clinicians. The examiners were not masked to the clinical details and were perhaps biased to diagnose strokes based on presence of vascular risk factors. Conversely, history taking is part of the clinical assessment by expert and non-expert alike and knowledge of the history may have contributed to the excellent sensitivity and specificity of bedside testing in this study. That is, the utility of hHIT may be increased when physicians know the history of the presenting problem.

Interpretation of bedside hHIT can be complicated. Positive hHIT may be related to lack of experience as novice tends to overcall whilst experts tend to undercall corrective saccades in chronic peripheral lesions [13].

Since a negative hHIT is strongly associated with central lesion [27], over-diagnosing a positive hHIT is dangerous as a stroke maybe dismissed. We only performed caloric testing in 30% of VN, all of whom had positive hHIT. Caution is required when comparing hHIT to caloric testing, as each tests a different frequency property of the vestibulo-ocular reflex [29]. Overall hHIT has a sensitivity of 35–100% and a specificity of 95–100% [6, 10] against caloric testing depending on the population studied. A negative hHIT maybe seen in 8–18% of VN [4, 23], and this maybe because more than 50% of canal paresis is needed for hHIT to be positive [11], the presence of inferior VN [1], or covert saccades that are imperceptible to the examiners [33]. The order of examination maybe important, as detecting signs such as SD and central-type nystagmus first may bias the examiners to produce negative hHIT, and conversely seeing a unidirectional nystagmus may tempt the examiners to diagnose a positive hHIT. Quantitative aspect of hHIT, such as covert saccades, has been investigated in chronic vestibular lesions [3, 22, 33], but its property in AVS has not been systematically evaluated. Likewise, quantification of SD, vertical smooth pursuit and characterization of nystagmus could lead to better diagnostic accuracy in AVS, and a prospective study utilizing a combination of video-oculography and scleral search coil is needed.

Our study has practical implication for reducing health care cost, as CT and MRI utilization has risen by more than 1.5-fold in a 10-year period but the rate of central vestibular diagnoses has not [16]. Given AVS was the presentation in 3.4% (24/700) of the stroke admissions in our study, teaching for the frontline clinicians could now include multistep ocular motor testing.

Conflict of interest Drs Luke Chen and Will Lee have nothing to disclose. Associate Professors Brian Chambers and Helen Dewey have nothing to disclose.

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