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Streamlining the Diagnosis of Atypical Facial Palsies: A 5-year Review of 805 Patients

Lok Ka Cheung, MBChB, MRCS*
Ruben Y. Kannan, FRCS (Plast), PhD*
Catriona Neville, BSc (Hons)*
Tamsin Gwynn, BSc (Hons)*
Karen Young, BSc (Hons)*
Rosanna C. Ching, FRCS (Plast)†
Charles Nduka, FRCS (Plast)*

Background: Currently, there are no definitive guidelines in the investigation and management of atypical facial palsies (AFPs). Our aim was to determine the etiology of AFPs presenting to a tertiary facial palsy center and to review the current spectrum of diagnostic and management approaches to these conditions.
Methods: A retrospective cohort analysis of attendees at the Queen Victoria Hospital multidisciplinary facial palsy clinic over a 5-year period from 2016 to 2020 was conducted. Demographic data were collated from the QVH Research and Governance team. Those presenting with classic Bell's palsy or Ramsay-Hunt syndrome were excluded. Anyone with atypical presentations (including multiple recurrences, focal neurological deficits, polycranial neuropathies, autoimmune conditions, hemifacial spasms, hearing/balance issues, weight loss, segmental facial palsies, and gradual onset presentations) were included under the AFP category. These patients were subjected to standard serological and radiological investigations and their follow-ups were reviewed.
Results: A total of 849 patients were identified, and 805 had actual facial palsy presentations. Of these, 172 patients had AFP. The majority of these patients had MRI imaging tests, which were useful, but the remaining serological tests were found to correlate more with symptom clusters and specific questions rather than with random tests for all AFPs.
Conclusions: Although serological and radiological investigations help in the diagnosis of AFP, specific questions and presentations help streamline the diagnosis, without affecting its accuracy whilst reducing unnecessary tests and, thereby, cost and time. We present an algorithm organized by specific questions of presentations in those with AFPs. *(Plast Reconstr Surg Glob Open 2022;10:e4087; doi: 10.1097/GOX.0000000000004087; Published online 9 February 2022.)*

INTRODUCTION

The most common cause of acute lower motor neurone (LMN-type) facial palsy is idiopathic Bell's palsy.[1] This is postulated to be due to herpes-simplex virus (HSV), where 70%–90% of patients recover spontaneously.[2,3] Studies have shown great variations in incidence, although many of the studies cannot be considered representative due to poorly defined area and inclusion of facial palsy patients.[3] Other conditions such as varicella virus, trauma, and autoimmune conditions can cause either more aggressive presentations or increased recurrences of facial palsy.

*From the *Facial Palsy Unit, Queen Victoria Hospital, East Grinstead, UK; and †Oxford Craniofacial Unit, John Radcliffe Hospital, Headley Way, Oxford, UK.*

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Facial palsy is a symptom; more serious conditions including cancer and cerebrovascular accidents can be mistaken for Bell's palsy, with disastrous consequences. Hence, thorough investigation and suitable management plans as recommend by Hohman et al,[4] including blood tests of Lyme titer, HSV-1 & 2 IgG, varicella zoster virus (VZV) IgG & IgM, c-reactive protein (CRP), erythrocyte sedimentation rate (ESR), anti-neutrophil cytoplasmic antibody, angiotensin converting enzyme, rheumatoid factor, antinuclear antibodies (ANA), anti-Sjögren's-syndrome-related antigen A autoantibodies, anti-Sjögren's-syndrome-related antigen b autoantibodies, thyroid-stimulating hormone, glucose, full blood count, antiphospholipid Antibodies (APA), Epstein–Barr virus (EBV), prophyrobilnogen, cytomegalovirus (CMV), and mumps are critical in facial palsy multidisciplinary clinics.[4]

In our institution, we routinely employ the current diagnostic standards for atypical facial palsies (AFPs).[4] As part of a service review, we ask the question as to whether such extensive investigating involving over 20 serological and radiological tests are necessary in all instances, and

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whether or not there is a role for a more streamlined diagnostic algorithm based on specific presentations.

PATIENTS AND METHODS

The Queen Victoria Hospital Business Intelligence Unit database was used to search for first attendances at the Facial Palsy multi-disciplinary clinic over a 5-year period from 2016 to 2020 for this retrospective cohort study. Demographic data (including age, gender, etiology, laterality, and postal code) were collected via a review of medical notes on the EVOLVE medical archiving system. The approval for service review was given by the QVH Clinical Governance Team (Audit No. 508).

AFPs are defined in our clinical practice as those who do not present as classic Bell's palsy patients, that is: (1) acute lower motor neurone type facial palsy involving the entire hemi-face, which resolves within 6 months,[1] (2) those with three or more recurrences of presumed Bell's palsy,[5] (3) those presenting with hemifacial spasms[6] and cranial nerve involvement.[7] The exclusion criteria included documented and/or confirmed causes of facial palsies and those with hemifacial spasms without synkinesis. Data were gathered onto a Microsoft Excel spreadsheet with further exploratory analysis performed using GraphPad PRISM v8.0 (LaJolla, USA). Abnormal serological tests with their respective test sensitivity, specificity, and positive predictive values (PPVs) were performed. Chi-square testing was performed to examine the significant of association between a specific blood test and the subtype of AFP. Fisher's exact test was used when the sample size was small ($n < 5$).

As our hospital is in a Lyme-endemic area,[8] Lyme disease testing is indicated within 6 weeks for anyone with Lyme disease but not for chronic Lyme disease.[9] EBV testing is nonspecific although there is an association with recurrent facial palsies. CMV again is nonspecific and only indicated in those with painless and temporary neck node enlargement.[10] Of the two HSV tests, only HSV-1 is associated with bilateral facial palsies and recurrence (31%). HSV-2 is a nonspecific test with no associations. VZV IgG antibody testing is associated in those with recurrent facial palsies but also has significant overlap with other viruses, as shown in Table 4. The IgM VZV antibody test, although not sensitive in our cohort, is an important test to perform in those with active polyneuritis cranialis.[11] While there is an association of facial palsy with HIV infections, these should be performed for clinical reasons.[12]

Exploratory Analysis

To better stratify the etiologies of facial palsy, the overall causes of facial palsy were divided into four subsections: autoimmune, infectious, metabolic, and structural.

RESULTS

Of a total of 849 patients, 805 patients met the inclusion criteria for facial palsy presentation. The average age of all cases was 51.4 years (range: 1–97 years). There was a female predominance, with 478 female (59.4%) and 327 male patients (40.6%), with the majority of cases being idiopathic Bell's palsy at 61.4%, followed by iatrogenic injury (17.8%), Ramsay-Hunt syndrome (5.8%), traumatic injury (4.5%), congenital (3.8%), cerebrovascular accidents (2.2%), pregnancy (1.9%), vascular loops (1.6%) and Guillain- Barre syndrome (1%). This is shown in Table 1. The number of patients presenting with unilateral symptoms was 772 (95.2%), with 421 patients (52.3%) on the left and 351 patients on the right (43.6%), whereas 33 (4.1%) patients presented with bilateral facial palsy.

Of these, 172 cases (21.4%) presented atypically as AFPs. Over 20 serological tests in addition to Gadolinium131 MRI scans of the brain and internal acoustic meatus were performed in selected cases. One-hundred and forty of these patients (17.4% of the total) had a range of the recommended 22 blood panel tests. Only 39% of these tests ($n = 54$) had abnormal serological tests, with their respective test sensitivity, specificity, and PPV shown in Table 2.

Takeaways

Question: We ask the question as to whether current extensive investigating involving over 20 serological and radiological tests are necessary in all instances.

Findings: Based on the data in our retrospective cohort study, clinical risk factors are better predictors of the underlying cause of atypical facial palsies rather than blood tests alone.

Meaning: A new diagnostic algorithm for atypical facial palsies is put forward based on our findings.

RESULTS

Autoimmune Group

The mean ages of patients in the autoimmune subcohort are generally over 50 years with a female preponderance, which is shown in Table 3. ANA and Extended nuclear antibody (ENA) tests seem to have much overlap, and either

Table 1. Etiology of Facial Palsies Presenting to Our Tertiary Level Facial Palsy Center

Etiology	*n*
Idiopathic/unknown	479
Iatrogenic	143
Ramsay-Hunt syndrome	46
Trauma	36
Congenital	22
Cerebral vascular accident	18
Pregnancy	15
Vascular loop	13
Guillain-Barré syndrome	8
Moebius syndrome	8
Birth trauma	5
Brain lesion	4
Motor neurone disease	2
SLE and autoimmune cranial neuropathy	2
Goldenhar syndrome	1
Duchennes muscular dystrophy	1
Neurosarcodosis	1
Lyme disease	1

Table 2. Statistical Parameters of Sensitivity, Specificity, and PPVs of Each Test Included in the Hadlock Diagnostic Algorithm[4]

Test	Blood Panel	Performed	Sensitivity	Specificity	PPV
1	ANA	60	75%	98%	75%
2	ENA	9	100%	100%	100%
3	RF	39	0%	86%	0%
4	APA	3	0%		
5	ESR	66	100%	93%	73%
6	CRP	63	0%	98.4%	0%
7	cANCA	62	0%	91.9%	0%
8	pANCA	62	0%	89.9%	0%
9	SSa/SSb	8	100%	100%	100%
10	Lyme	18	100%	100%	100%
11	LFT	75	0%		
12	TFT	41	0%		
13	EBV	61	100%	81.2%	27.8%
14	CMV	52	100%	96.2%	33.3%
15	HSV-1	22	55.6%	50%	38.5%
16	HSV-2	21	0%	87%	0%
17	HIV	1			
18	Varicella IgG	18	100%	54.5%	58.3%
19	Varicella IgM	17	0%		
20	ACE	40	100%	92.9%	100%
21	Bone profile	11	0%		
22	Urine Porphyrins	1	0	0	0 %

HIV, human immunodeficiency virus; HSV-1, herpes simplex virus 1 antibody; HSV-2, herpes simplex virus 2 antibody; SSa, Sjogren's syndrome (Ro) antibody; SSb, Sjogren's syndrome (La) antibody; LFT, liver function test; TFT, thyroid function test.

Sensitivity: The ability of this test to correctly identify the subgroup of atypical facial palsy.

Specificity: The ability of this test to correctly identify patients who truly do not have the subgroup of atypical facial palsy tested for.

Positive Predictive Value: The probability that patients with a positive result truly have that subgroup of atypical facial palsy.

one should suffice. In the case of rheumatoid factor tests, these are indicated in bilateral facial palsies or those with a history of suspected rheumatoid arthritis. Erythrocyte sedimentation rate, on the other hand, is a very sensitive test but lacks specificity. It also has a significant overlap with other markers and is redundant, as is CRP, unless looking for an active infection. Both cytoplasmic antineutrophil cytoplasmic antibody (cANCA) and peripheral cytoplasmic antibody (pANCA) tests, though sensitive, significantly overlap with both the ANA and acetylcholine receptor test (AChR) tests. The latter also has some overlap with viral markers such as CMV, HSV, and VZV, which reduces its autoimmune specificity. Testing for ssA/ssB is indicated in those AFP patients with Sjogren's syndrome symptoms (eg, dry eyes, lack of saliva, and so on), while acetylcholinesterase-converting enzyme is a useful generic test in AFPs, as it can identify sarcoidosis effectively. The APA test is only indicated in those with recurrent thrombophilia. The overlap of these autoimmune markers is graphically depicted in Figure 1.

Infectious Group

In this subcohort, the mean age was 57 years with a 2:1 female-to-male predominance. Lyme disease testing is indicated within 6 weeks for anyone with Lyme disease but not for chronic Lyme disease in our hospital. EBV testing is nonspecific although there is an association with recurrent facial palsies. Of the two HSV tests, only HSV-1 is associated with bilateral facial palsies and recurrence (31%). HSV-2 is a nonspecific test with no associations. VZV IgG antibody testing is associated in those with recurrent facial palsies but also has a significant overlap with other viruses, as shown in Table 4. The IgM VZV antibody test, although not sensitive in our cohort, is an important test to perform in those with active polyneuritis cranialis. The overlap between positive viral titers is shown in Figure 2.

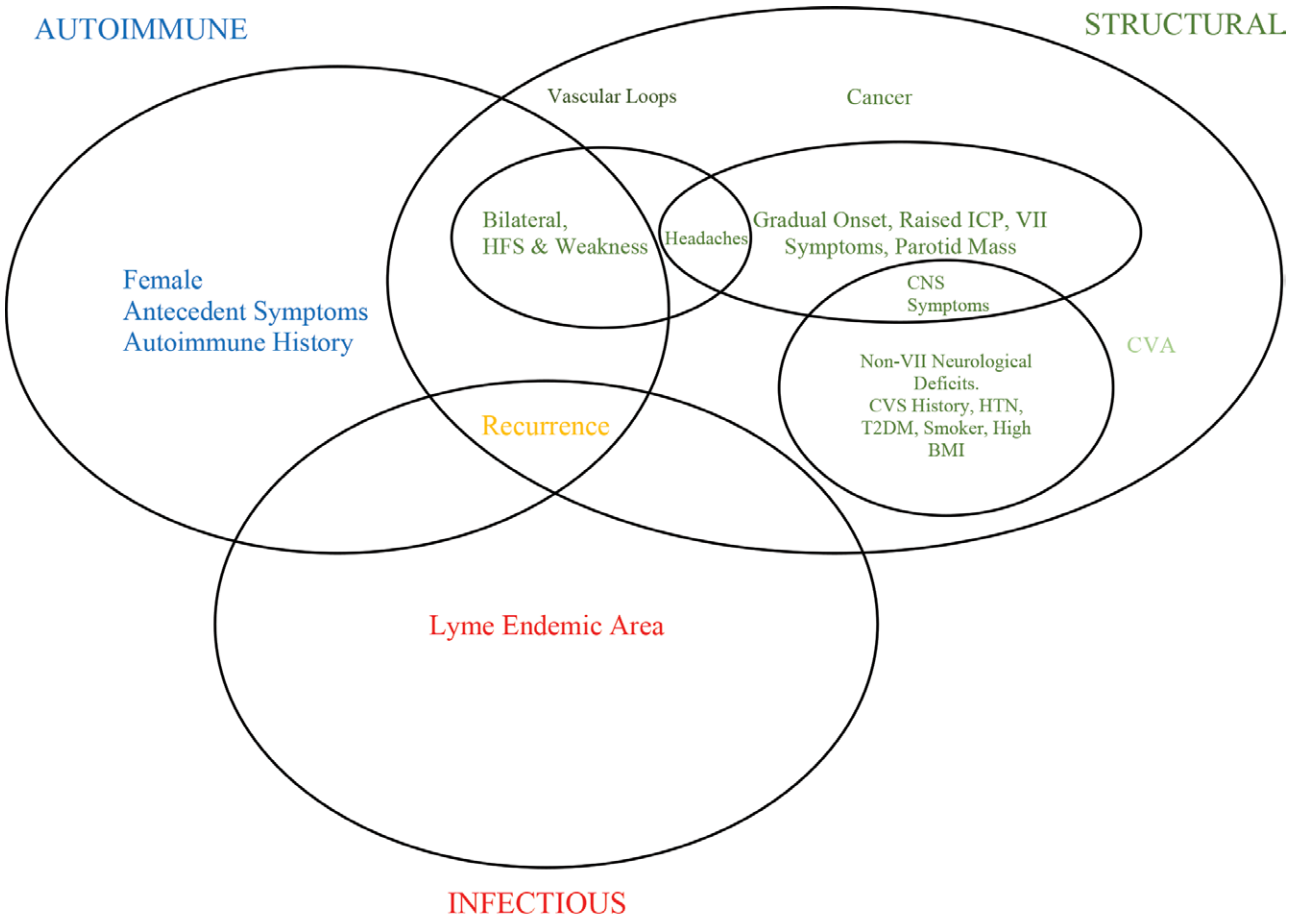
Metabolic Group

The most common indication for performing tests such as the thyroid function test and bone profile is to rule out thyroid disorders and calcium imbalances, respectively.

Table 3. Associations and Overlaps of the Autoimmune and Infectious Markers of the AFP Patients in Our Study

Test	(n)	Positive	Mean Age (y)	F:M	Associations	Overlapping Positive Markers
ANA	60	4	70	1:1	Hashimoto's, DLE, Sjogren's (100%)	ENA, cANCA, AChR Ab (100%)
ENA	9	2	67	2:0	Rheumatoid, Hashimoto's, DLE, Sjogren's (100% of cases)	ANA+ve (100%)
RF	39	5	55	4:1	Rheumatoid, bilateral VII palsy (80% of cases)	ACE & ESR (60% of cases)
ESR	66	15	42	14:1	Hypothyroidism, diabetes, pulmonary sarcoidosis, chronic fatigue syndrome, ankylosing spondylitis, Sjogren's syndrome, ulcerative colitis, antiphospholipid syndrome, and SLE (53% of cases)	RF, ACE, EBV IgM, IgG, CMV IgG, HSV-1 & 2, Varicella Zoster IgG, pANCA and low platelets (73%)
CRP	63	1	35	1:0	Previous surgery	None
cANCA	67	5	53	1:4	Ocular myasthenia (1), brain metastases (1)	ANA, AChR, pANCA (75%)
pANCA	70	7	52	3:4	None	cANCA, CMV, AChR, ANA, HSV-1, VZV IgG, ACE, ESR (100%)
ssA/ssB	9	2	67	2:0	Rheumatoid, Hashimoto's, DLE, Sjogren's (100% of cases)	ANA (100%)
ACE	40	4	52	3:1	Sarcoidosis (50% of cases & a new diagnosis)	RF, pANCA (50%)
Lyme	18	1	35	1:0	Acute Lyme (<6wk)	None
EBV	61	45	53	2:1	50% had viral illnesses associated with 22% RHS and 28% recurrent	Varicella Zoster, Ebnaigg virus, CMV IgG, HSV-1/2, ESR, ACE (78% overlap)
CMV	51	3	47	1:2	Two patients with RHS and one with viral symptoms	pANCA, Varicella Zoster, EBV (100% overlap)
HSV-1	21	13	52	11:2	15% bilateral VII palsy, 31% recurrent palsy	HSV-2, RF, ESR, Varicella Zoster, EBV, pANCA (54% overlap)
HSV-2	20	3	54	3:0	None	HSV-1, RF, ESR, EBV, or VZV IgG (100% overlap)
VZV IgG	18	12	47	5:1	One-third with recurrent facial palsy	ESR, EBV IgM, CMV, EBV, HSV-1, pANCA (83% overlap)

HSV-1, herpes simplex virus-1; HSV-2, herpes simplex virus-2; ssA/ssB, Ro/La Sjogren antibodies.



HFS – Hemifacial spasms
ICP – Intracranial pressure
CVS – Cardiovascular
CNS – Central Nervous System
HTN – Hypertension
T2DM – Type 2 Diabetes Mellitus
CVA – Cerebral Vascular Accident
BMI – Body Mass Index

Fig. 1. Venn diagram showing the cluster presentations of high-value symptoms and signs in AFPs. Using this, clinicians should request targeted investigations.

The latter is only indicated in hemifacial spasm cases. The liver function test was an unnecessary test in our cohort, with zero abnormalities noted in 74 tests.

Structural Group

Contrast-enhanced MRIs were performed in 118 patients, with 50 of them (42% of the subcohort) showing an abnormal result. The MRIs we use were able to detect the underlying cause of AFP in 48 patients. Although 13 of these patients had prominent vascular loops, detected on MRI, only nine of them had correlating symptoms. Otherwise, there were three cases of false negatives. The overall sensitivity, specificity, and positive predictive value of MRIs in AFPs were 87%, 76%, and 40%.

DISCUSSION

AFPs may be defined as any facial palsy condition where there is no well-defined etiology (eg, trauma, oncological resection, or a presumed case of Bell's palsy or Ramsay-Hunt syndrome). AFPs can be broadly classified into autoimmune, congenital, endocrine, infectious, inherited, neoplastic, and neurovascular conditions (eg, vascular loops).[13] As they routinely result in poorer recovery, the clinician must be prudent in investigating and managing such cases to reduce resultant morbidity. If there is no improvement after 3 weeks of standard treatment and incomplete recovery at 5 months after onset of symptoms, then AFPs should be considered.

Table 4. Statistical Analysis of All the Serological Markers Performed for AFP Patients in This Study

Blood Test	Sample Size	*P*	Outcome
ANA	89	0.197	
RF	40	0.331	
ESR	66	0.677	
CRP	63	0.039	Statistically significant
cANCA	62	0.149	
pANCA	63	0.001	Statistically significant
Lyme	18	1	
LFT	74	1	
TFT	41	1	
EBV	61	0.87	
CMV	51	0.99	
HSV-1	21	0.076	
HSV-2	20	0.361	
Varicella IgG	18	0.768	
Varicella IgM	16	1	
ACE	40	0.316	
Bone profile	11	1	

Autoimmune causes of AFP found in this study include Guillain-Barre syndrome, neurosarcoidosis, systemic lupus erythematosus (SLE), and polycranial neuropathy. When relying on these serological tests for a diagnosis, what is apparent from this study is that these tests are more sensitive than specific. The ANA test, for instance, detects SLE, Sjogren's syndrome, and rheumatoid arthritis,[14,15] and the same goes for ENA, rheumatoid factor, and APA. None of these tests are highly specific. As for the angiotensin converting enzyme test, this has been reported to be 41% sensitive and 90% specific, overall.[16] Both CRP and erythrocyte sedimentation rate in our current battery of serological tests are redundant, with CRP only indicated for suspected ongoing infections (eg, otitis media).

Based on our observation of the data, it appears that clinical risk factors are better predictors of the underlying cause of the AFP rather than blood tests alone. A cluster of autoimmune risk factors, including women with antecedent symptoms, previous autoimmune disease, recurrent facial palsies, hemifacial spasms, and bilateral disease, are better pointers of diagnoses than serological markers alone. It appears therefore that the purpose of these tests is merely to strengthen clinical suspicion rather than diagnose.

Conversely in the case of infection-related AFPs, clinical risk factors are nondescript with many overlapping symptoms. Here, greater reliance is placed on pathogen titer analysis. For instance, the sensitivity of antibody-based Lyme tests is proportional to the duration of the infection, meaning that the test will detect chronic Lyme disease better.[9] For acute Lyme disease, the IgM test can be employed but can only be within a month of onset. The probability of a positive test, nevertheless, is higher in a Lyme-endemic area (eg, South East England) and those presenting with erythema migrans.

In the case of viruses (while these tests are sensitive and, in the case of EBV, specific as well)[17], these are best performed in the acute care setting: for example, CMV for those presenting with painless cervical lymphadenopathy,[10] and HSV-1/-2 for those with suspected encephalitis.[18] In our clinical practice, we have found these tests useful to identify nonconventional viruses as the causative factor. In the case of varicella, IgG analysis is helpful in cases of chronic polyneuritis cranialis,[11] whereas IgM tests are more appropriate in the acute encephalitis/meningitis phase.[19]

In those with organic brain conditions such as strokes, tumors, and vascular loops, symptoms such as headache, visual changes, and non-VII-related neurological deficits suggest a structural cause for the AFP, necessitating imaging. Other red flag signs included projectile vomiting (a sign of raised intracranial pressure), dizziness, hearing/balance disorders (vestibular neuromas), and a parotid mass (cancer). Another tell-tale sign of AFP is gradual onset facial palsy, involving a single branch or a segment of the facial nerve. The key with imaging is to request contrast-enhanced brain, internal acoustic meatus, and parotid MRIs. Even so, if negative, the history is so suggestive that an ultrasound-guided core biopsy or an open parotid biopsy is warranted.[20]

An outlier in the structural category of AFPs is vascular loops. Often looked over as being merely an anatomical variant, there is increasing evidence that these loops can cause facial palsy itself or, more frequently, hemifacial spasms.[21] The combination of vessel impingement rather than mere abutment is necessary to diagnose these, alongside the MRI evidence.

Based on a detailed analysis of clinical symptoms in the 176 AFP patients, we have created a Venn diagram, which clusters high-value symptoms to help compartmentalize the different etiologies of AFP into defined subcohorts in the first stage of the diagnostic sieve, before proceeding to a more refined investigation with serological tests. As for imaging modalities, we recommend that imaging be done for all AFP patients, given its efficacy in identifying abnormal pathology.[22]

Once the likely AFP etiological group has been identified after clinical history, more specific test batteries can be requested, as shown in Table 5. The use of symptom clusters based on our analysis of AFP patients in this study should increase both sensitivity and specificity of these investigations with specific probing questions to hone down the possible diagnosis further. This illustrates that the purpose of a facial palsy clinic is to identity the etiological category and to refer these patients to the appropriate specialty. This is a more bespoke approach to AFP diagnosis.

Streamlining the diagnostic process would improve patient experience and outcome, reduce unnecessary investigations and blood tests, and reduce National Health Service costs.

This study also highlights the limitations of the Harvard Hohman[4] et al approach and introduces a layer of questioning. It can also act as a screening tool for atypical facial palsy and point clinicians to refer to the appropriate specialties.

LIMITATIONS

The majority of patients had MRI imaging tests, which were useful, but the remaining serological tests were

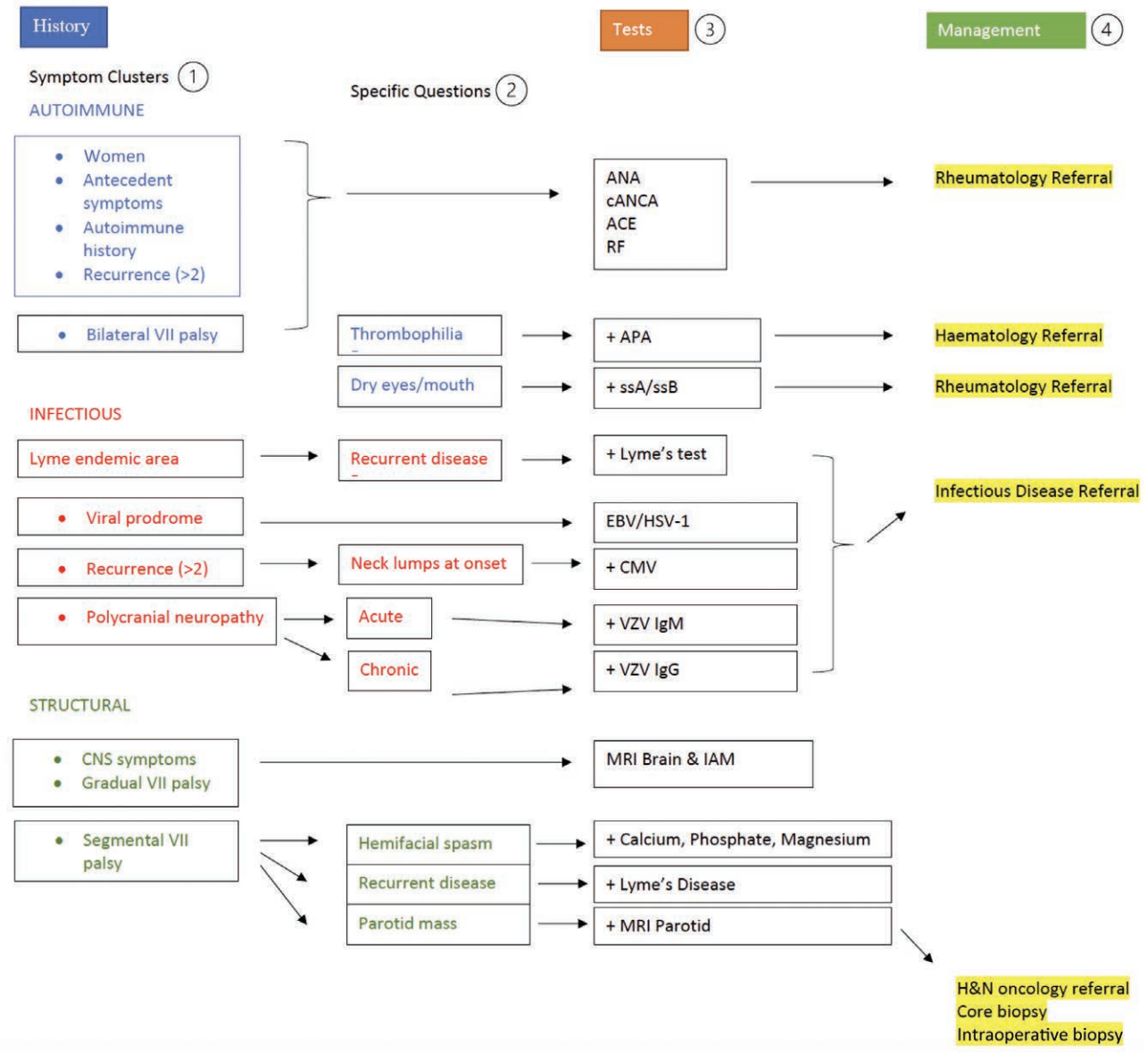


Fig. 2. Proposed algorithm: diagnostic flow chart for atypical facial palsy. Using this, clinicians should request targeted investigations.

found to correlate more with symptom clusters and specific questions rather than random tests for all AFPs. The inconsistent diagnostic testing is also a limitation, as not all patients underwent the same testing throughout the study.

In general, none of the tests for AFPs reached statistical significance, barring both CRP and pANCA, but due to the sample size of these two tests, there is a potential type II error, as not every patient had the full panel or blood tests, which is a limitation of this study. Furthermore, we acknowledge multiple testing as a potential source of positive results. As atypical facial palsy is a nebulous disease, subgrouping was partly determined by the tests, and therefore biased. Hence, it falls on a keen clinician to identify the subtle hints in the history and examination of the patient.

Nevertheless, there remain limitations in our study as, for example, we did not routinely check for trans-thyretin antibodies to rule out amyloidosis as a cause of facial palsy. This cohort usually presents with other systemic manifestations such as cardiac arrhythmias and is particularly prevalent in Northern Europe.[23] Other conditions such as barotrauma-induced facial palsy can be diagnosed purely on history alone.[24]

Table 5. A Clinically-based Algorithm to AFP Diagnostic Testing

Symptom Cluster	Specific Question	Test	Management
Women	None	ANA, cANCA	Rheumatology referral
Antecedent symptoms		ACE	
Autoimmune history		RF	
Recurrence (>2)	Thrombophilia	+ APA	Hematology referral
Bilateral VII palsy	Dry eyes/mouth	+ ssA/ssB	Rheumatology referral
Lyme-endemic area	Recurrent disease	+ Lyme test	Infectious disease referral
CNS symptoms	None	MRI Brain & IAM	
Gradual VII palsy	Hemifacial spasm	+ Ca, P, Mg	
Segmental VII palsy	Recurrent disease	+ Lyme test	
	Parotid mass	+ MRI Parotid	H&N oncology referral Core biopsy Intraoperative biopsy
Viral prodromes	None	EBV, HSV-1	Infectious disease referral
Recurrence (>2)	Neck lumps at onset	+ CMV	
Polycranial neuropathy	Acute	+ VZV IgM	
	Chronic	+ VZV IgG	

CONCLUSIONS

Random testing of all AFPs is not necessary in our opinion but rather, a more structured approach based on symptom clusters and specific questions allows for a smarter and more streamlined approach. The encapsulation of these symptoms and a new diagnostic algorithm for AFPs is put forward based on our findings.

Lok Ka Cheung, MBChB, MRCS
Plastic Surgery Department
Queen Victoria Hospital
Holyte Road
East Grinstead
RH19 3DZ, UK
E-mail: Lokka.cheung@nhs.net

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