Performance of components of Year 3 Luton GCA FTP compared to final diagnosis at >6 months*

Diagnostic Tool	No. pts**	Sensitivity %	Specificity %	PPV %	NPV %
Rheumatologist CP GCA (High)***	90	60	98.3	93.8	86.1
Rheumatologist CP GCA (Moderate & High)***	90	88	63.5	48.9	93
Any halo on TAUS	85	81.5	91.4	81.5	91.4
Bilateral positive TAUS	85	59.3	98.3	94.1	83.8
TAB	28	69.2	100	100	78.9
LVI (CT or CTPET)	12	80	100	100	87.5
Combined diagnostic tests in the whole FTP (bilateral positive TAUS +/- or positive TAB +/- or positive LVI)	88	92	98.3	95.8	96.7

*using patient record to Oct 2019 **2 patients passed through twice ***before imaging/TAB

Conclusion: Unlike Yr 2, the higher secure diagnosis rate in Yr 3 could not be attributed to shorter time on prednisolone or better equipment. The increase was likely due to several factors including further improved sonographer skill and increased confidence to withdraw steroids in insecure cases with low/moderate CP-GCA. This approach did not increase sight loss. Further reduction in TAB rate financially justified a 3rd FTP slot/wk created in Yr 3.

Each component of the FTP was an inadequate diagnostic tool. Combinations of diagnostic tools are needed to obtain the highest sensitivity and specificity for GCA diagnosis. FTPs limit tests to the minimum required for secure diagnosis. The "gold standard" diagnostic test for GCA is the whole FTP combined.

References:

- [1] Schäfer et al *Rheum (Ox)* 2017;56(9):1479-83
- [2] van der Geest et al *ARD* doi: 10.1136/annrheumdis-2019-216343

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SAT0272

THE VALUE OF CLINICAL AND LABORATORY FEATURES TO PREDICT EXTENT OF LARGE VESSEL VASCULITIS ON PET CT

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Background: Giant cell arteritis (GCA) is an idiopathic vasculitis affecting large and medium-sized vessels. The pattern of arterial involvement is heterogeneous with two overlapping categories recognised: classical cranial GCA and extra-cranial GCA (or large vessel vasculitis – LVV) that predominantly affects the aorta and its proximal branches. Although LVV is present in around 80% of patients with cranial GCA, and around one third will develop large vessel complications, there are no guidelines for which patients should be screened for it (1). We sought to investigate whether clinical and laboratory features were a useful guide to the severity of LVV on FDG PET-CT.

Objectives: To retrospectively analyse whether baseline patient characteristics are able to predict the extent of large vessel vasculitis on PET-CT.

Methods: Clinical data for 65 patients referred for a PET-CT scan by Rheumatology at the Freeman Hospital, Newcastle between January 2015 and May 2018 were retrospectively analysed. The most recent full blood count and inflammatory markers prior to the scan were used. Scans were reviewed by a consultant radiologist and trainee. The arterial network was split in to ten potentially involved territories (aortic arch, thoracic aorta, abdominal aorta, iliac vessels, axillary, brachiocephalic, subclavian, carotid, vertebral and femoral arteries. Both the value of highest standardised uptake value (SUV max) and the territory affected was recorded for each positive scan.

Results: In the period analysed 65 PET-CT scans were requested, mostly (77%) as baseline investigations for symptoms with LVV in the differential diagnosis. Of these 22 (34%) were positive for LVV and in that group the majority of patients (64%) were female. In those with a negative scan, 47.5% were on concurrent steroid treatment compared to 9% with a positive scan. Regression analysis suggested that the number of systemic features (weight loss, pyrexia, polymyalgia) was weakly correlated with the number of affected territories ($p=0.04$). In contrast there was no correlation between laboratory tests ((CRP ($p=0.91$), ESR ($p=0.46$), Hb ($p=0.44$), platelets ($p=0.74$)) and the number of territories affected. The aortic arch (47%) was most commonly the territory with the highest degree of FDG

uptake (SUV max) followed by the abdominal aorta (21%) and thoracic (10%) and femoral arteries (10%). There was no correlation between SUV max and laboratory tests ((CRP ($p=0.55$), ESR ($p=0.89$), Hb ($p=0.82$), platelets ($p=0.17$)) or the number of systemic features ($p=0.7$). There was no significant difference in the number of territories affected between those on steroid treatment at the time of the scan and steroid-naïve patients, albeit the number of positive scans in those on steroid treatment was low ($n=5$).

Conclusion: These results suggest that clinical and laboratory features are a poor guide to predicting the maximal severity and extent of disease on FDG PET-CT.

References: [1]Koster MJ, Matteson EL, Warrington KJ. Large-vessel giant cell arteritis: diagnosis, monitoring and management. *Rheumatology (Oxford)*. 2018;57(suppl_2):ii32-ii42.

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SAT0273

PROGNOSTIC FACTORS FOR POOR OUTCOME IN BEHÇET'S SYNDROME WITH NEUROLOGICAL INVOLVEMENT: RESULTS FROM A CLINICAL LONG-TERM FOLLOW-UP OF A SINGLE CENTRE

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Background: Behçet's Syndrome (BS) is a vasculitis of unknown cause. Neurological disease is a type of its serious involvement and causes morbidity and mortality.

Objectives: In this study, we aimed to describe the clinical characteristics of neuro-Behçet Syndrome (NBS) and to define prognostic factors that were associated with poor outcome.

Methods: Among 2033 patients with BS, we performed a retrospective analysis of 94 patients (52.1% male; mean age 36.1 (11.9) years), who fulfilled the ISG-1990 for BS. We divided patients into two subgroups, either parenchymal (p-NBS) or non-parenchymal (np-NBS). Clinical, laboratory, and imaging characteristics of the patients were defined. We described the poor outcome as a modified Rankin score (mRS) ≥ 3 at last follow-up and/or death, and assessed the predictor factors associated such kind of outcome.

Results: In total, 52 (55.3%) patients presented with p-NBS, of whom 15 (28.8%) had progressive course, and 42 (44.7%) presented with np-NBS (Table.1). Ocular involvement was more common in p-NBS than np-NBS (59.6% vs. 39.1% respectively, $p=0.04$), whereas vascular involvement excluding sinus vein thrombosis was more frequent in patients with np-NBS (15.4% vs. 50.0%, respectively, $p=0.001$). Headache and papilledema were frequently observed in np-NBS ($p<0.001$), whereas paresis, sensory symptoms, pyramidal and cerebellar signs were significantly common in p-NBS (<0.001). Twenty-five patients (26.6%) experienced at least one relapse. After a median of 54 (IQR 79.4) months, 11 patients (11.7%) had disabling

Table 1. Clinical characteristics of the patients in each subgroup

	All n= 94	Parenchymal NBS n= 52	Non parenchymal NBS n= 42	p value
Gender (male, %)	49 (52.1)	29 (55.8)	20 (47.6)	0.43
Age at Behçet diagnosis	29.2 $\pm$ 9.52	29.93 $\pm$ 9.49	28.26 $\pm$ 9.60	0.40
Age at NBS diagnosis	36.1 $\pm$ 11.9	37.02 $\pm$ 11.43	35.00 $\pm$ 12.61	0.42
Initial mRS ≥ 3	33 (35.1)	28 (53.8)	5 (11.9)	<0.001
Oral aphthae	94 (100.0)	52 (100.0)	42 (100.0)	-
Genital ulcer	71 (75.5)	40 (76.9)	31 (73.8)	0.73
Skin involvement (PPE+EN)	72 (76.6)	35 (67.3)	37 (88.1)	0.018
Musculoskeletal involvement	46 (48.9)	28 (53.8)	18 (42.8)	0.29
Ocular involvement	47 (50)	31 (59.6)	16 (38.1)	0.038
Vascular involvement (except SVT)	29 (30.9)	8 (15.4)	21 (50)	<0.001
Gastrointestinal involvement	5 (5.3)	2 (3.8)	3 (7.1)	0.65
HLA B51 positivity (n=32)	14 (43.8)	6/12 (50)	8/20 (40)	0.58
Pathergy positivity (n=51)	26 (51.0)	13 (54.2)	13 (48.1)	0.67
Clinical characteristics				
Headache	73 (77.7)	32 (61.5)	41 (97.6)	<0.001
Fever	6 (6.4)	5 (9.6)	1 (2.4)	0.22
Paresis	26 (27.7)	24 (46.2)	2 (4.8)	<0.001
Pyramidal signs	34 (36.2)	31 (59.6)	3 (7.1)	<0.001
Sensory symptoms	34 (36.2)	30 (57.7)	4 (9.5)	<0.001
Cerebellar signs	19 (20.2)	18 (34.6)	1 (2.4)	<0.001
Cranial nerve involvement	13 (13.8)	9 (7.3)	4 (9.5)	0.28
Papilledema	24 (25.5)	3 (5.8)	21 (50)	<0.001
Relapse	25 (26.6)	16 (30.8)	9 (21.4)	0.31
Total follow-up time (mo)	54.01 (IQR: 79.4)	64.8 (IQR: 90.2)	44.0 (IQR: 68.1)	0.12
Last mRS ≥ 3	16 (16.8)	15 (28.8)	1 (2.4)	0.001
Death	5 (5.3)	5 (9.6)	0 (0)	0.06

mRS: Modified Rankin Score, PPE: Papulopustular eruption, EN: Erythema nodosum, SVT: Sinus Vein Thrombosis