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THE IMPACT OF PROLONGED HUNGER STRIKE: CLINICAL AND LABORATORY ASPECTS OF TWENTY-FIVE HUNGER STRIKERS

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Background – Hunger strike is a very serious entity which may lead to severe diseases and death.
Aims – The aim of this study is to document the clinical, neurophysiological, neuroradiological, and neuropsychological aspects of prolonged hunger strike.
Material and method – We investigated the clinical and laboratory characteristics of 25 hungerstrikers hospitalized during refeeding process. One sample t-test, independent samples t-test, Mann–Whitney and Pearson correlation tests were used for statistical analyses.
Results – Twelve of them had a continuous hunger strike ranging between 190–366 days. The other 13 had quitting intervals for various reasons with a continuous hunger ranging between 65–265 days with a total hunger duration of 153–382 days. The mean loss of body mass index (BMI) was 40.98±9.3%. Imbalance, sleep disorders, somatosensory disturbances, and adynamia were the most common complaints. At admission, one third experienced ophtalmoparesis, about half of them had paresis, one quarter had truncal ataxia. At discharge 16% had persistant ophtalmoparesis and 36% nystagmus. Only four patients (16%) could walk independently. There was no serious MRI, EEG findings. Most prominent EMG findings were the decrease in median and sural nerve cnap, median and fibulary cmap, and fibulary ncv values. They showed mild impairment in MMTS and most of them had attention deficit and frontal type memory impairment.
Conclusion – It can be concluded that vitamin B intake, independent of the quantity and timing, lowers the morbidity and mortality of hunger strikers.

Keywords: *starvation, hunger strike, death fast, Wernicke–Korsakoff syndrome*

AZ ELHÚZÓDÓ ÉHSÉGSZTRÁJK HATÁSAI: 25 ÉHSÉGSZTRÁJKOLÓ KLINIKAI ÉS LABORATÓRIUMI EREDMÉNYEI

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Háttér – Az éhségsztrájk nagyon súlyos körülmény, amely súlyos betegségekhez és halálhoz vezethet.
Célok – A szerzők célja az volt, hogy dokumentálják az elhúzódó éhségsztrájk klinikai, neurofiziológiai, neuroradiológiai és neuropszichológiai vonatkozásait.
Anyagok és módszer – A szerzők 25, újratáplálás miatt kórházba felvett éhségsztrájkoló klinikai és laboratóriumi jellemzőit vizsgálták. A statisztikai elemzéshez egymintás t-próbát, független mintás t-próbát, Mann–Whitney- és Pearson-féle korrelációs tesztet használtak.
Eredmények – A vizsgálati alanyok közül 12-en 190–366 napig folyamatosan éhségsztrájkoltak. A többi 13 vizsgálati alany különböző okok miatt ezt időnként megszakította, a folyamatos éhezési időszakok 65–265 napig tartottak, összesen 153–382 napot tettek ki. A résztvevők a testtömegindexüknek átlagosan a 40,98±9,3%-át veszítették el. Leggyakoribb panaszként egyensúlyzavar, alvászavar, szomatoszenzoros zavarok és adinámia jelentkeztek. A felvételkor a betegek egyharmadában volt jelen ophthalmoparesis, felében paresis, egynegyedében törzsataxia. Az elbocsátáskor 16%-ban volt jelent perzisztáló ophthalmoparesis és 36%-ban nystagmus. Csak négy beteg (16%) tudott önállóan járni. Nem észleltek súlyos MR- és EEG-eltéréseket. Az EMG-leletek közül a legszembetűnőbb a nervus medianus és suralis cnap-, a nervus medianus és fibularis cmap- és a nervus fibularis ncv-értékeinek a csökkenése volt. Enyhén károsodott az MMTS, a betegek többségében figyelemhiány és a frontális típusú memória károsodása volt megfigyelhető.
Következtetés – A következtetések szerint a B-vitaminbevitel a mennyiségétől és időzítésétől függetlenül csökkentheti az éhségsztrájkolók morbiditását és halálozását.

Kulcsszavak: *éhezés, éhségsztrájk, éhhalál, Wernicke–Korsakoff-szindróma*

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Hunger strike is a very serious entity which may lead to severe neurological disorders and even death. Although it is very widespread due to several reasons; religious, social, economic, political, the literature is quite limited. Over 50 people died due to hunger strikes in recent times and many more were left severly disabled[1]. In 1996 a large number of prisoners underwent hunger strike for 69 days which resulted in 12 deaths in Turkey[2]. Furthermore most of the survivors suffered from Wernicke–Korsakoff's (WK) syndrome, whose correlation to thiamine deficiency is well established.

Although neurological complications of nutritional deficiency due to chronic alcoholism is common and very well known, other conditions such as prolonged hunger strike have not been investigated in detail in the literature. There have been several reports of fasting for a few days, however there are very few reports, mostly case reports on prolonged hunger strike[3, 4]. There are only two large series[5, 6]. First one is a retrospective study of 33 South African political prisoners on hunger strike for up to 28 days[5]. A more recent study is a prospective follow up of 41 Turkish prisoners who fasted for up to 324 days with a mean of 199 days[6]. Several articles have addressed the neurophysiological aspects of hunger strikers[6–9].

The aim of this study is to document the clinical, neurophysiological, neuroradiological, and neuropsychological aspects of prolonged hunger strike. Based on previous experiences, the strikers consumed liquid, sugar, salt, and took daily vitamin B complex supplement with doctor suggestion, in order to prevent WK syndrome. Therefore their survival times were much longer. There is little information on neurological complications as well as the feeding policies after long lasting starvation.

Material and method

Twentyfive patients who willingly stopped hungerstrike were hospitalized immediately at the neurology department, Bakirköy Hospital for Psychiatric and Neurological Diseases between June 2001 and January 2002. At their admittance their complaints, physical and neurological examination findings were recorded. Their initial weight and body mass index (BMI) at admittance were noted.

As soon as their blood samples were taken for biochemical and hematological analyses, they were introduced a standard preprogrammed feeding. In the first day, 100 mg of vitamin B1 was given intravenously before fluid replacement was done. 1000 µg B12 and 20 mg vitamin K were given intramuscularly. Following these, 900 mg vitamin B1 and other B complex vitamins were given in 500 cc 5% dextrose solution. An additional 500 cc isotonic solution was given. The feeding was started with 250 g oligopeptide diet and was increased by 250 g daily, until it reached 1000 grams. Starting from the second day, supplements of oral vitamins A, B, E and folic acid were added to 500 mg parenteral thiamine. Oligopeptide diet was slowly replaced by polypeptide diet within the first week. Normal diet was started as the patients' oral tolerance improved. Parenteral fluid replacement was ended in the second week. They were taken into strict bed rest and given adequate amount of fluids avoiding overloading.

All patients had a cardiological and psychiatric consultation. They were given a Standardized Mini Mental Test (SMMT) and neuropsychological test battery within the first week, as soon as they were ready to comply with this long assessment. The applied tests were Blessed Short Orientation and Memory Test, Digit Span Test, Weschler Memory Scale, modified verbal memory test, and modified praxis test. Language skills were assessed by Boston Naming Test, and modified reading, writing, comprehention tests. Visuospatial skills and construction ability was assessed by drawing tests (cube, flower). For higher cognitive skills, subgroups of the modified Wais Test were applied. Frontal functions were assessed with clock drawing test, Luria consecutive drawing test and verbal fluency test.

Modified Barthel Index was applied to all patients at admission, and discharge in order to test their daily life activities.

All patients underwent cranial magnetic resonance imaging (MRI) operating at 1.5 T within the first week. Imaging was done in T1W, T2W, flair, axial and sagittal planes.

Early stage electroencephalography (EEG) was performed within the first week after the termination of the hunger strike. Scalp EEG recordings were obtained according to the international 10-20 system with a Nihon Kohden digital EEG device.

In 21 of the 25 patients electrophysiological analysis was performed. In 15 of these patients both nerve conduction velocity (NCV) studies and evoked potentials (SEP, VEP and BAEP), in 4 patients only NCV studies, and in 2 patients only evoked potentials were applied. Electrophysiological analysis were done by a Medelec, sapphire 4ME model.

All tests were applied within the first week of hospitalization. Median, ulnar, tibial and peroneal nerve motor conduction velocities as well as

Table 1. *The basic data and the change of BMI in our patients*

Patient no	Sex	Age	Duration days	Quitting (days)	Total (days)	Initial BMI	Final BMI	Loss of BMI %
1	F	19	205	–	205	23.71	12.42	47.6
2	M	24	200	–	200	21.84	9.36	57.1
3	F	24	185	5	197	22.58	11.66	48.4
4	M	24	152	15	172	22.39	11.94	46.7
5	M	25	80	various	153	24.30	14.87	38.8
6	M	26	125	various	185	24.78	15.52	37.4
7	M	28	268	–	268	21.61	13.10	39.4
8	M	28	212	20	242	20.42	13.72	32.8
9	M	28	190	–	190	23.98	17.35	27.6
10	M	29	217	–	217	25.39	16.01	36.9
11	F	29	185	7	196	19.14	11.32	40.9
12	M	30	217	–	217	22.05	13.65	38.1
13	M	30	165	13	185	24.56	16.47	32.9
14	F	32	229	–	229	24.46	15.05	38.5
15	F	32	125	20	213	26.03	16.82	35.4
16	F	33	65	10, 20	245	24.31	13.42	44.8
17	M	36	210	–	210	20.45	16.07	21.2
18	M	36	200	–	200	26.72	10.02	62.5
19	M	43	315	–	315	26.64	17.64	33.8
20	M	27	261	–	261	26.64	13.49	49.3
21	M	31	200	–	200	19.83	12.99	34.5
22	M	32	224	–	224	30.47	16.62	45
23	M	48	366	–	366	24.22	13.84	42.9
24	M	31	170	60	330	21.87	13.28	39.3
25	M	32	265	61	382	23.4	13.42	42.6

BMI: body mass index

median, ulnar, sural sensory conduction velocities were studied unilaterally. Distal motor and sensory latencies, motor and sensory conduction velocities, motor and sensory action potential amplitudes, minimum median and tibial F latencies were analized. Motor conduction velocities were calculated with proximal stimulus. Base to peak amplitudes were measured.

Distal sensory conduction velocities and sensory action potential amplitudes were performed. Sensory nerve conduction studies were performed orthodromically for the upper extremity and antidromically for the sural nerve. Peak to peak amplitudes were recorded.

Bilateral SEP analysis of median and tibial nerves were performed and cortical SEP results were assessed. N20 and P37 latencies for SEP and P100 latencies of pattern reversal VEP were recorded. The results were compared with the normal control values of our laboratory.

Patients were hospitalized for approximately 3 weeks after normal diet was started, and were discharged after their major complaints ceased.

One sample t-test, independent samples t-test, Mann–Whitney test (95% CI, $P<0.05$) and Pearson correlation tests were used for statistical analyses.

Results

Twentyfive patients, 6 females and 19 males, ages ranging between 19-48 (mean 30.3) were documented prospectively. 12 of them had a continuous hunger strike ranging between 190–366 days. The other 13 had quitting intervals for various reasons with a continuous hunger ranging between 65–265 days with a total hunger duration of 153–382 days. The mean loss of body mass index was 40.98±9.3% (ranging between 21.2–62.5%) during the hunger period (**Table 1**.). Six patients took vitamin B only for a very short time (P 2, 3, 4, 11, 12, 18), whereas only 3 had regular vitamin B intake (P 8, 14, 24) . The others had various intake of vitamin B supplements (containing 100-600 mg of vitamin B1) during the strike.

Thirteen patients (52%) experienced a loss of consciousness at least once during the hunger strike around day 143 (ranging between 80–200). Nausea and vomitting were reported by 28% of the hunger strikers whereas diarrea was a little bit more common (36%).

During admission to the hospital all of the patients complained of adynamia. Imbalance (92%) and sleep disturbances (92%) were also common. Complaints of sleep were mostly insomnia, long

Table 2. *The laboratory and electrophysiological findings of our patients*

Patient no	NSS Adm	NSS Disc.	Laboratory findings	MMS	Cog.	IBI	DBI
1	Par, TA	Par, Romb.	Anemia, Na↓, prt↓	24	AD, FMD	20	59
2	CC, D,Par, HH, EA	D, HH, Romb	Anemia, Hep enz↑	26	AD	36	83
3	CC, D, Nist, Par,TA,	D, Nyst, TA, Romb	Anemia, prt↓	26	AD, FMD	30	61
4	O, Par	Nyst	Anemia, Hep enz-, K↓, prt↓	27	NL	54	92
5	O, Par, HH,	Nyst		29	NL	86	97
6	HH,TA,	Par, HH, Romb.		28	NL	72	96
7	Nyst, Par	Nyst, EA, Romb.	Anemia, K↓, Na↓, P↓, prt↓	28	NL	41	51
8	TA			29	AD	74	97
9	CC, DT, D, Par, EA	D, O, Romb.	Hep enz↑, K↓, P↓, prt↓	21	AD, FMD	52	90
10	O*, Par, HH	HH, Romb.	Anemia, prt↓	28	NL	44	89
11	Par	Romb.	Anemia, K↓, prt↓	29	NL	44	71
12	Nyst, TA, EA	Nyst, HH, TA, EA	Anemia, prt↓	29	AD, FMD	20	38
13	NL	NL	Anemia, prt↓	26	NL	97	100
14	Nyst, EA	NL	Anemia, Hep enz↑, K↓, P↓, prt↓, Hypgl.	29	AD	85	94
15	HH,	HH,		27	AD	97	100
16	HH, Psy	Nyst, Romb.	Anemia, Hep enz↑, K↓, P↓, prt↓	28	AD	73	94
17	O	O	K↓, Ca↓, prt↓	23	AD, FMD	100	100
18	CC, D, O, Nyst, Par, HH, TA,	D, Nyst, HH, TA, Romb.	prt↓	28	AD	27	31
19	DT, Par	Par	Anemia, K↓, Ca↓, prt↓	24	AD, FMD	36	47
20	NL	NL	Anemia, Hep enz↑, K↓, Ca↓	26	AD, FMD	94	98
21	O*, Pys.	O, Romb.	Anemia, Hep enz↑, K↓, prt↓	30	AD, FMD	88	91
22	NL	O	Anemia	26	AD, FMD	94	94
23	CC, DT, O, Psy	Nyst	Anemia, Ca↓, prt↓	22	AD, FMD	76	94
24	CC, D, Nyst, Par, HH,EA	D, Nyst, EA, Romb.	Ca↓, prt↓	28	AD	24	34
25	Nyst	NL	K↓, Na↓, prt↓	30	AD	94	98

CC: change of consciousness, DT: disorientation (time), D: dysartria, O: ophtalmoparesis, Nyst: nystagmus, Par: paresis, HH: hemihypoestesia, TA.: truncal ataxia, EA: extremity ataxia, Psy: psychiatric symptoms, Romb: (+) romberg sign, Hep. enz↑: hepatic enzyme elevation, K: potassium, P: phospate, Ca: calcium , Na: sodium, Prt: proteinemia, Hypgl: hypoglycemia, AD: attention deficit, FMD: frontal type memory deficit (recall abnormality), NL: normal, IBI: initial Barthel index, DBI: discharge Barthel index

latency in falling asleep, and frequent awakenings. Somatosensory disturbances (88%) such as tingling, pain and numbness were also common complaints. 88% of the patients had tinnitus, rumbling noise and phonophobia. Other symptoms at admission and their frequency were as follows; lack of concentration (80%), forgetfulness (76%), gait disorder (68%), headache (60%), disorders of taste (mostly hypogeusia) (80%), photophobia (52%), dizziness (52%), autonomic complaints (52%), osmophobia (16%).

On physical examination all of the patients except two were hypotensive. Only one patient (P1) had marked hepatomegaly, and 5 patients (P 4, 8, 10, 15, 17) had abdominal sensitivity, 7 patients (28%) had bradycardia (P 4, 5, 10, 13, 16, 17, 19), and only one had tachycardia (P 23).

Apart from one patient (P 19) with hypoproteinemia and anemia who experienced severe edema during fluid replacement, no serious cardiac problems were experienced.

On admission all patients were alert and only 3 patients (P 9, 9, 23) had time disorientation.

Four patients, 16% (P 2, 3, 9, 18), had dysarthria on admission, and one (P 24) developed dysarthria two days later. 28% of the patients (P 3, 7, 12, 14, 18, 24, 25) had nystagmus, and 28% had ophtalmoparesis (P 4, 5, 10, 17, 18, 21, 22), although only three of them (P 10, 17, 21) were severe. One patient (P 23) developed nystagmus and ophtalmoplegia 2 days after admission to the hospital. About half of the patients (48%) had paresis, however only two of them (P 1, 2) were severe. 32% of the patients (P 2, 5, 6, 10, 15, 16, 18, 24) had hemihypaesthesia. 5 patients (P 2, 9, 12, 14, 24) had minimal anomaly or slowing in cerebellar tests and 6 patients, 24% (P 1, 3, 6, 8, 12, 18), had severe truncal ataxia. Most of the patients could not

Table 3. *Comparison of nerve conduction velocities between two groups*

	Test value (n=21)	Patient's value (n=19)	P* value
Median cnap	31.2±9.4	25.31±8.87	0.01
Median cmap	10.3±2.88	8.25±1.72	0.000
Ulnar cmap	9.57±2.72	8.01±1.72	0.001
Fibular ncv	51.24±3.82	47.14±3.19	0.000
Fibular cmap	5.15±1.66	3.24±1.67	0.000
Sural cnap	25.31±10.64	12.9±6.18	0.000
Median F	23.67±1.89	26.93±3.97	0.029
Tibial F	43±5.96	49.98±3.22	0.000

*One sample t-test.

Table 4. *Comparison of evoked potential values between two groups*

	Test value (n=42)	Patient's value (n=17)	P* value
Median SEP latency (N20)	19.12±1.1	20.04±1.26	0.014
Tibial SEP latency (P37)	38.75±2.03	41.10±3.84	0.023
BAEP III	3.68±0.31	4.05±0.38	0.002
BAEP V	5.6±0.46	5.91±0.30	0.001
BAEP I-III	1.9±0.52	2.36±0.38	0.000
BAEP I-V	3.93±0.46	4.23±0.31	0.001

*One sample t-test.

do tandem walk, and had a positive Romberg sign, however the patients were severely adynamic. No patient had extrapyramidal findings, except one (P 12), who had titubation. None of the patients had bladder or bowel problems.

Two patients (P 21, 23) had hallucinations, 1 experienced deja vu (P 16) and one (P 23) was agitated.

At their discharge only 4 patients had persistant ophtalmoparesis. Nystagmus was more common as a sequele sign, seen in 9 patients. At discharge only four patients (16%) could walk independently. 13 patients had difficulty in tandem walking, 4 needed assistance even for a short distance walk and 4 could not walk at all.

The details of the neurological signs at discharge are summarized at **Table 2**.

The mean BI was 62 (ranging between 20–100) at admission and 80 (ranging between 31–100) at discharge.

Laboratory findings

Most of the patients suffered from electrolyte imbalance mainly of hypokalemia (44%), hypophosphatemia (24%) and hypocalcemia (20%). 64% of the patients had anemia. Hyponatremia was seen only in 3 patients (P 1, 7, 25) and hypoglycemia (P 14) only in 1. Hypoproteinemia was seen in 18 patients (72%). Liver functional tests were elevated in 7 cases (P 2, 4, 9, 14, 16, 20, 21). 17 of the 19 patients (89%) had decreased blood levels of folic acid. All of the 19 patients had either normal or high levels of vitamin B12. Homocysteine was tested in only 17 cases and was elevated in 12 of them (70%). Interestingly, this increase is positively correlated with a lower BMI loss (p=0.003) and a higher iBI (p=0.023).

Cranial MRI was performed in 24 patients. They were all normal except two. One of them (P1) had tuberculomas and the other (P 16) had mild cerebral atrophy.

Standard EEG was normal in 68% of the cases. There were bilateral bioelectrical disorganization in 8 patients; temporal (P 1), frontal (P 20, 24), frontotemporal (P 2, 16, 25) and diffuse (P 4, 23). Only the patient with tuberculomas (P 1) had generalized tonic clonic convulsions. Control EEGs of patients 16, 20 and 24 were normal whereas anomalies persisted in others.

The mean SMMT score was 26.84±2.44 (ranging between 21-30). 5 patients (20%) got scores <24 indicating cognitive impairment. 8 patients (32%) had only attention deficit, and 10 (40%) had frontal type memory impairment along with attention deficit. Seven (28%) patients had no deficits.

Compared with the normal control values of our

laboratory, 11 of the 19 NCV patients and 9 of the 17 EP patients had abnormalities. Most prominent findings were the decrease in median and sural nerve cnap, median and fibulary cmap, and fibulary ncv values were significantly lower than the control values. Median and tibial F latencies were longer than the control group (**Table 3**).

There was no abnormalities in VEP studies. Median and tibial cortical SEP (P 37) latencies were longer than the control group showing long tract involvement. BAEP showed normal I latencies whereas prolonged III and V absolute latencies and I-III and I-V interpeak latencies suggesting lower brainstem involvement (**Table 4**).

The impact of total fasting duration, vitamin B uptake and change of body mass index on electrophysiological tests were assessed using Mann–Whitney and independent samples t-test. Fasting longer than 200 days was associated with decrease in median cmap ($p=0.017$). Slowing in tibial nerve conduction velocity was correlated with ≥40% change in body mass index ($p=0.004$). Vitamin B uptake did not seem to affect the electrophysiological tests.

Discussion

Severe consequences of nutritional disorders of the nervous system, especially Wernicke's encephalopathy (WE) and Wernicke–Korsakoff syndrome (WKS) are well known. Although it had been mostly described in alcoholism, a large number of cases occur due to poor nutritional states, either from inadequate intake, malabsorption, or increased metabolic requirements. This syndrome is the most expected outcome in hunger strikers[10, 11]. Although deaths started after day 60 in the previous 1996 strikers, a four fold survival time in our group is striking. Duration of hunger was ranging between 65–366 days (mean 201 days) which is much longer than reported in the literature except Basoglu's series (mean 199). Similar to this series our patients consumed liquids with glucose and salt along with vitamin B and minimalized physical activity. Due to these precautions diarrhea, nausea and vomiting were seen rarely and lately. Nausea and vomitting was the most common sign at 1996 strike and day 60 was the critical turnpoint of the deaths[2]. However, only one third of our patients experienced nausea and vomiting during hunger strike. In our group, loss of consciousness occured at least once during strike in about half of the patients. However, although the percentage is quiet high, it took place around day 143.

In our group adynamia, imbalance and sleep disorders were the leading complaints. Somatosensory disturbances were also common.

The strikers lost about 40% of their body mass. As the loss of BMI increased, the initial BI decreased (Pearson correlation coefficient $r=0.406$, $p=0.04$,) and there is a significant correlation between the initial and discharge BMI. On the other hand neither the duration of the hunger strike nor age have any statistical effect on initial BI. Besides, there was no correlation between the loss of BMI and Mini Mental Test scores. At 1996 strike it seemed that <25% loss was not dangerous and 30% seemed like the threshold for mortality and morbidity[2]. As our patients lived longer due to vitamin supplementation, although the mean loss was greater, the survival was better. We had only one patient with less than 25% loss (21.2%), and he was the only patient who got full points from BI at admission and discharge, and his ophtalmoparesis was a sequele sign.

As the duration and amount of vitamin B intake was various, it was not possible to perform any statistical comparisons between the groups. However, 2 patients with the shortest duration of vitamin B intake had the highest percentage of BMI loss. Furthermore, the other patients with short vitamin B intake had higher loss. This group also had the lowest initial BI scores along with 2 patients, P1, who had tuberculosis and P 24, who did not receive any vitamin B for 160 days, and was hospitalized because he lost his consciousness, and then went on strike for another 170 days taking vitamin B. Another point is that they mostly took vitamin B complex pills (vitamins B1, B6 and B12) as pure vitamin B1 is not available at our pharmaceutical market. Some of the patients had very high levels of vitamin B12. Interestingly some of these patients had very low levels of folic acid. We do not know if these had any impact on the prognosis.

Hypoproteinemia and anemia were the most prominent findings as expected. Hypoproteinemia seem to play a negative role on the outcome as patients with normal proteins had a better Barthel index at discharge ($p=0.29$). Cardiac problems are reported to be potentially dangerous during refeeding, however only one of our patients experienced cardiac problems. As most of the strikers included glucose and salt in their diet, hypoglycemia and hyponatremia were no major problems, whereas hypokalemia was quite common. It is very well known that hepatosteatosis is seen in WK patients and is usually associated with chronic alcoholism. However some of the hunger strikers, although they

were non alcoholics, experienced hepatic problems. Patients with abdominal discomfort, hepatomegaly or increase in liver enzymes underwent ultrasonography, however no pathological finding was detected and the abnormalities were reversible.

Although the full classical triad of WE was not seen in our group, components were seen at a milder degree. There was no meaningful statistical difference between age, sex, duration of hungerstrike, loss of BMI, vitamin B intake and components of WKS, mainly concerning ophtalmoparesis, ataxia, and cognitive impairment.

At 1996 hungerstrike ophtalmoplegia was reported to be persistant at 28% of the patients[2], unlike the alcoholic series where very well recoveries were reported[11]. Basoglu et al reported sequele oftalmoplegia in 27% and nystagmus and ataxia in 46% of the patients[6]. In our series ophtalmoplegia was seen in about one third of the patients at admission and persisted in 16% of the patients at discharge. In 1996 series the recovery percentage of nystagmus was similar however it had a higher tendency to remain as a residual sign. Similarly in our series nystagmus was seen in one third of the strikes at admission and detected in 36% at discharge. It is known that ataxia and/or cerebellar fingings seem to recover more slowly compared to ophtalmic findings. In 1996 hungerstrike it was reported that the cerebellar signs even got worse from the fifth month on mainly progression of truncal ataxia, and development of extremity ataxia and disarthria were reported. Only at the end of the first year, some improvement in ataxia was noted[2]. In Basoglu's series all patients were ataxic at admission and ataxia remained in 46% of the patients at 1 year follow up[6]. Although nearly all of our patients had ataxia of stance at admission, only 5 patients had extremity ataxia at admission, and 3 at discharge. Three patients were noted to have truncal ataxia at discharge. However, only four patients (16%) could walk freely at discharge. About half of the patients had difficulty in tandem walk, 4 needed assistance even for a short distance and 4 could not walk at all. Similarly with Basoglu's series Romberg sign at discharge, was positive in 12 patients, which could have contributed to this outcome. 3 patients had dysarthria along with other cerebellar findings and two had isolated dysarthria at discharge.

In 1996 hungerstrikers, although WE patients did quite well on neurocognitive tests, WKS patients showed characteristic impairment in memory processes (learning and recall) along with decrease in attention and verbal fluency ataxia in 46% of the patients[11]. Moreover, they showed no significant improvement over time. Similarly in Basoglu's series memory deficits were prominent and they showed correlation with duration of hunger but not with duration of vitamin B intake. They showed improvement in confabulation and anterograd amnesia with treatment. Frontal lobe dysfunction were also noted[2]. Our patients showed mild impairment in MMTS. Only 5 patients (20%) got scores below 24. None of them had severe cognitive impairment but congruous with the previous series, most of them had attention deficit and frontal type memory (recall) impairment.

Psychosis, including hallucinations, delusions, is a well defined component of WKS. Besides agitation, irritability and impulsivity is also reported in Korsakoff's psychosis[11]. In our group only 2 patients had hallucinations and 1 experienced deja vu and one of the patients was also agitated.

The MRI of WE/WKS reveal medial thalamic peri-third ventricular hyperintensities at proton and T2 weighed images during subacute period. D'Aprile et al[12] and Shogry et al[13] reported enhancement of the mamillary bodies on MRI in acute WE. Mamillary body atrophy has been reported as the sign of chronic WE[14]. Mamillary body atrophy was also seen in some cases in 1996 hunger strikers[2]. Most of the cranial MRI's were normal and only a few demonstrated mild brain atrophy in Basoglu's series[6]. Similarly, none of our patients had any related abnormalities on MRI. It might be that, as all of our imaging was done within the first days, it was too early to see the MRI changes mostly reported during the subacute period. On the other hand this may be due to efficient thiamine intake or proper re-feeding as it is shown that early thiamine replacement may result in complete resolution of thalamic and midbrain MR signal abnormalities[15, 16]. Galluci et al[16] have reported that the high signals on T2-weighted images represent pathologically non-specific findings such as edema, demyelination and gliosis.

Furthermore, Antunez et al have stated that although MRI abnormalities are useful in confirming the diagnosis of acute WE, their absence does not exclude the diagnosis as sensitivity of MRI for WKS was 53% and the specificity, 93%[17].

Mild to moderate diffuse slow EEG activity was reported in about half of the alcohol addicts with WKS[5]. Also diffuse slow wave activity or frontal/frontotemporal slow waves in EEGs were reported in WE/WKS cases (2, 6, 18). Similar EEG abnormalities were seen in about one third of our patients.

Neuromuscular effects of long-standing hunger have been rarely reported[6, 7, 9]. Oge et al[9] reported

that the most prominent finding in hunger strikers group was the low amplitude of compound muscle action potentials elicited in motor NCV studies which was concluded to be resulted from the reversible muscular changes. The other electrophysiological findings suggested that peripheral nerves and long central nervous system pathways were also mildly involved[6, 9].

Similarly in our group the most prominent findings were the decrease in median and sural nerve cnap, median and fibulary cmap, and fibulary ncv values.

Ambliopia was a severe symptom which recovered quickly and througly at 1996 series[2]. Deficiencies in B vitamins were held responsible for its etiology. Although none of our patients had ambliopia, about half of them complained about photophobia. Contradicting the high percentage of VEP abnormalitity reported before[6], there was no abnormalities in our VEP studies.

It is well known that WE may be precipitated acutely in at-risk patients by intravenous glucose administration or carbohydrate loading and to avoid this parenteral thiamine should be given before the administration of glucose. The relatively good prognosis in our series is probably due to the careful replacements of the thiamine and other vitamins.

As a result, we can conclude that vitamin B intake, independent of the quantity and timing, lowers the morbidity and mortality in the hunger strikers.

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