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SHORT COMMUNICATION

The importance of brain banking for dementia practice: the first experience of Turkey

Ahmet Turan Isik · Ayca Ersen Danyeli · Derya Kaya · Pinar Soysal · Nuri Karabay · Murat Gokden

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Abstract This study reports the results of the first brain tissue banking experience of Turkey in the Unit for Aging Brain and Dementia at Dokuz Eylul University, Department of Geriatric Medicine, Izmir. Here, we have briefly described our efforts on brain banking in our country, which consist of six brains from autopsies that had at least two years of clinical follow-up in the 2015–2017 period. The evaluation led to the diagnosis of two Alzheimer's disease (AD) with cerebral amyloid angiopathy, one AD with dementia with Lewy bodies, one corticobasal degeneration, one multiple system atrophy, one vascular dementia. We believe that the study is of a special importance because of its potential of becoming a brain banking center in the region and because of its contributing to the international knowledge of the neuropathological features of dementia, while characterizing the epidemiology of these diseases in the region.

Keywords Brain banking · Neurodegenerative diseases · Dementia · Brain donation · Brain autopsy

Introduction

Diagnosis of neurodegenerative diseases such as Alzheimer's disease (AD), Lewy bodies and dementia (DLB), vascular dementia (VaD), Parkinson's disease (PD), frontotemporal lobar degeneration (FTLD), and

A. T. Isik (✉) · D. Kaya
Unit for Aging Brain and Dementia, Department of Geriatric Medicine, Faculty of Medicine, Dokuz Eylul University, 35340 Balcova, Izmir, Turkey
e-mail: atisik@yahoo.com; ahmet.isik@deu.edu.tr

D. Kaya
e-mail: derya.kaya@deu.edu.tr

A. E. Danyeli
Department of Pathology, Acibadem Mehmet Ali Aydinlar University, Istanbul, Turkey
e-mail: ayca.danyeli@acibadem.com

P. Soysal
Department of Geriatric Medicine, Faculty of Medicine, Bezmialem Vakif University, Istanbul, Turkey
e-mail: psoysal@bezmialem.edu.tr

N. Karabay
Department of Radiology, Faculty of Medicine, Dokuz Eylul University, Izmir, Turkey
e-mail: nuri.karabay@deu.edu.tr

M. Gokden
Division of Neuropathology, Department of Pathology, University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: godenmurat@uams.edu

others are currently based on established clinical criteria, which are far from making a definitive diagnosis, because they are characterized by a complex set of neuropathological features, often with marked overlapping pathologies (Murphy and Ravina 2003). Also, it is well known that there is only a moderate relationship between some clinical diagnoses and subsequent neuropathological diagnoses (Nelson et al. 2010). It can be said that the first brain banking efforts started with anatomist collections, such as William Hunter, who archived almost fifty brain samples in the eighteenth century (Teacher 1900). Today, in many countries brain banks have been established to distinguish between healthy brain tissue and that showing disease or normal ageing and disease; however, with the exception of Europe and the US, in the eastern hemisphere China, India, Japan and South Korea each have only one brain tissue bank (Lee et al. 2017).

In this paper, we reported the results of Turkey's first brain tissue banking experience. Then, approximately 32 patients enrolled to donate brain tissue. So far, the brain tissues of six of these donors have been examined.

Materials and methods

This study, which consists of six brains of autopsies performed in 2015–2017 period, is based on cases of Brain Aging and Dementia Unit of Department of Geriatric Medicine of Dokuz Eylül University. All patients admitted to an inpatient and outpatient setting were evaluated by our team of geriatrician, neurologist, neuroradiologist, psychologist and neuropathologist. They all had at least two years of clinical follow-up in our Unit, in which time they underwent comprehensive geriatric assessment, including brain imaging, neuropsychological evaluation and laboratory evaluation.

Collection of postmortem brains

Informed consent was obtained from the patients and/or the legal representatives or the relatives (NOK) during their visit to our clinic. Four of our cases died in our clinic, one died in another hospital and the other at home. The NOK of the two cases who died in out of our clinic contacted our service immediately after death and then the body was transported to our unit within 12 h. Our contact service is open 24 h including weekends and holidays. One neuropathologist, one experienced technician and at least three clinicians were always ready before the body was transported. All the brains were removed in less than 12 h after death. The legal representatives/NOK received an autopsy report with a conclusive diagnosis of their family member's neurodegenerative disease.

As a matter of fact, all these stages were carried out in accordance with the framework and reports of the BrainNet Europe consortium, which consists of 19 European brain banks (Alafuzoff et al. 2012; Bell et al. 2008; Schmitt et al. 2007).

Ethical issues

All participants and/or their NOK, signed an informed consent form approved by the Institutional Approval Board prior to death, allowing for clinical and laboratory and radiological assessments before death, and for brain donation after death. (252-SBKAEK, 2015/13-18). Our research was conducted in accordance with the criteria set by the declaration of Helsinki.

The approval is required for autopsy in our country unless the legal authorities have already ordered to perform an autopsy. However, as an essential principle, we ask for written approval containing information about the organization of our brain bank and our research objectives. Our brain bank works by getting permission from both the patient before death and from their NOK (If the patient is not capable, the written consent is requested from the NOK. Both patients and the relatives are informed that they can withdraw consent at any time. For example, NOK of five other patients have withdrawn their consent so far, after their patients died.

Clinical assessment

Clinical diagnosis of AD was made according to the criteria of the National Institute on Aging-Alzheimer's Association workgroups (McKhann et al. 2011). DLB was diagnosed according to the third report of the DLB Consortium (McKeith et al. 2005). Multiple system atrophy (MSA) was diagnosed based on the current diagnostic consensus criteria (Gilman et al. 2008). The clinical diagnosis of corticobasal degeneration (CBD)

was made according to Cambridge criteria (Bak and Hodges 2008). Idiopathic normal-pressure hydrocephalus (iNPH) was diagnosed based on the international iNPH guidelines (Relkin et al. 2005). Cerebral amyloid angiopathy (CAA) was diagnosed according to the Boston criteria (Knudsen et al. 2001). Clinical diagnosis of VaD was made according to the National Institute of Neurological Disorders-Canadian Stroke Network (Hachinski et al. 2006).

Radiological assessment

All cases had neuroimaging including brain magnetic resonance imaging (MRI) or computerized tomography. All brain MRI was performed with at least 1.5 T MRI machine.

Neuropathological evaluation

All brains underwent a standardized neuropathological assessment by one neuropathologist (AED). Cerebrospinal fluid (CSF) was collected from the lateral ventricles by transcallosal puncture. After removing the brain, its weight and volume were measured before fixation. Each step was digitally photographed from opening the skull to fixation. The Circle of Willis, the pituitary gland and the olfactory bulbs were dissected and fixated. Immediately after brain removal, tissue fragments 1 cm in size from the neocortex and hypothalamus were dispersed in 6 tubes, frozen and stored at – 80 °C for future genomic studies. The time difference between death time and brain removal was recorded for each case. The neuropathologist followed the consensus rules for AD, DLB, MSA and CBD when applying staining, sampling and microscopic evaluation. According to these guidelines, a total of 16 sections from frontal, temporal, parietal, occipital lobes, hippocampus, amygdala, cingulate, basal ganglia, thalamus, white matter of watershed areas, medulla, pons, midbrain and cerebellum were microscopically evaluated. Immunohistochemical stains were performed using antibodies against α-synuclein (LB 509; 1:400; Abcam, Cambridge, United Kingdom), phospho-PHF-tau (AT8; 1:200; ThermoFisher Scientific, Reinach, Switzerland), TDP43 (2E2-D3; 1:500; Abcam, Cambridge, United Kingdom) and β-amyloid (1:100; Abcam, Cambridge, United Kingdom) following the instructions of manufacturers.

This assessment led to the diagnosis of two ADs with Cerebral Amyloid Angiopathy (CAA), one DLB, one CBD, one MSA and one VaD. All clinical, laboratory, neuroimaging and histopathologic findings were shown in Table and figure.

Results

Case 1

Her complaints started as restlessness in 2008 at the age of 70 and she frequently asked the same questions. When she was admitted to the Unit in 2013, he had complaints of forgetfulness and poor attention. According to her clinical and radiologic (Table 1; Fig. 1a–c) findings, she was diagnosed with Probable AD. She died from urosepsis in August 2015. The neuropathologic findings were compatible with AD (A1, B2, C2) and CAA (Table 1; Fig. 2a–d).

Case 2

In 2014, at the age of 84, she was admitted to our Unit because of her forgetfulness, inability to walk, frequent falls, slow motion and speech, difficulty in swallowing, decreased interest in activities she had previously enjoyed, and feeling sad. According to her clinical and radiological findings (Table 1; Fig. 1d–g), MSA-cerebellar type was diagnosed in our department. She died in July 2016 from cardiopulmonary arrest. The neuropathologic findings were compatible with MSA (Table 1; Fig. 2e–h).

Case 3

For the past 2 years, he had a forgetfulness in which he lost his personal belongings and wandered aimlessly, and his symptoms worsened in the last 6 months, before he was admitted to our department in 2013 at the age of 68. He was diagnosed with possible AD and VaD with clinical and radiologic findings (Table 1; Fig. 1h–j). He died in May 2016 from aspiration pneumonia. The neuropathologic findings were concluded to be a VaD (Table 1; Fig. 2i–j).

Table 1 Characteristics of the cases

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Gender	Female	Female	Male	Female	Male	Male
Age at onset/diagnosis/death (year)	70/74/77	83/86/88	66/66/71	63/65/68	80/85/88	80/88/91
Education (year)	0	0	8 (Middle school)	5 (Elementary School)	15 (University)	15 (University)
Occupation	housewife	housewife	crane operator	housewife	Air force pilot	Musician
Behavioral symptoms	+	−	+	−	−	−
Dementia at diagnosis	+	−	+	−	+	+
Comorbidities	HT, DM,	HT, DM, gastroesophageal reflux, cholecystectomy	HT, DM, MNG	−	HT, Multiple Myeloma*	HT, BPH, PAD, CKD
Cause of death	Urosepsis	Cardiopulmonary arrest	Sepsis	Aspiration pneumonia	Aspiration pneumonia	Multilobar pneumonia
Physical examination	Bilateral positive grasp reflex and postural instability	OH, slow horizontal saccades, bilateral palmomental reflexes, dysarthria, brisk tendon reflexes, cerebellar ataxia, mild dysphagia, bradykinesia, mild rigidity in the upper extremities and postural instability	Positive snout reflex, brisk deep tendon reflexes on the left side, mild spasticity in lower left limb and extensor plantar response	apraxia in her left (nondominant) extremity, slowness in speech, bradykinesia, bilateral rigidity which is dominant in the left upper extremity and brisk deep tendon reflexes on both sides, bilateral grasp reflexes and postural instability	Bilateral palmomental reflex	OH, bradykinesia rigidity on both side
	CDR 2	UMPARS Part I:32. Part II: 19. Part IV: 4 Part III: SBP supine/standing: 145/114 mmHg DBP supine/standing: 81/60 mmHg HR/min supine/standing: 89/95 blurred vision +	CDR 1	UDRS: 26 UPDRS-III: 25	CDR 1	UPDRS-III: 19

Table 1 continued

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Markedly ımpaired functions	episodic memory, problem solving, semantic categorization, visuospatial abilities and attention	Attention, visuospatial abilities, executive functions	Memory and executive functions	Attention, visuospatial abilities, linguistic	Memory, attention and executive functions	Attention, visuospatial abilities
MMSE	17	12	25	26	24	NA
CDT	0	NA	4	NA	4	NA
CSF findings		Aβ42:169.29 pg/ml; Aβ40:63 pg/ml				Aβ42:39.29 pg/ml; Aβ40:63 pg/ml
		Tau_t: 4563 pg/ml; Tau_p: 935.83 pg/ml				Tau_t: 2760 pg/ml; Tau_p: 194.17 pg/ml
Treatment	Rivastigmine 15 cm^2/day	Compression stockings		L-dopa 300 mg/day	Rivastigmine 15cm^2/day	L-dopa 400 mg/day
	Memantine 20 mg/day	L-dopa 400 mg/day		Amantadine 100 mg/day	Memantine 20 mg/day	Rivastigmine 15 cm^2/day
	Risperidone					
Clinical diagnosis	Probable AD	Multiple system atrophy	Mix dementia (Probable AD + VaD)	Corticobasal syndrome + NPH	Probable AD	LBD + NPH
Neuropathologic assessment						
Definitive diagnosis	AD (A1, B2, C2) + CAA	MSA	VaD	CBD	AD (A2, B2, C2) + CAA	Alfa synucleopathy + AD (A1, B2, C2)
	Intermediate**				Intermediate**	Intermediate**
Weight (Gr); measured/expected	1042.2/ 1190 ± 10	978.6/1140 ± 10	1070/1350 ± 20	996/1240 ± 20	1243/ 1290 ± 130	1032/1290 ± 130

AD Alzheimer's disease, *BPH* benign prostatic hypertrophy, *CAA* cerebral amyloid angiopathy, *CDT* clock drawing test (0 [worst]-4 [best]), *CKD* chronic kidney disease, *DM* diabetes mellitus, *HT* hypertension, *DLB* dementia with lewy bodies, *MMSE* mini-mental state examination (0 [worst]-30 [best]), *MNA* mini nutritional assessment (0 [worst]-14 [best], *MNG* multinodular goiter, MSA multiple system atrophy, *NA* not applicable, *NPH* normal pressure hydrocephalus, *OH* orthostatic hypotension, *PAD* peripheral artery disease, *RBD* REM behaviour disorder, *UMSARS* unified multiple system atrophy rating scale, *UPDRS* Unified Parkinson's disease rating scale, *VaD* vascular dementia

*Multiple myeloma was diagnosed within the last year of his life

**Level of Alzheimer's Disease Neuropathologic Changes

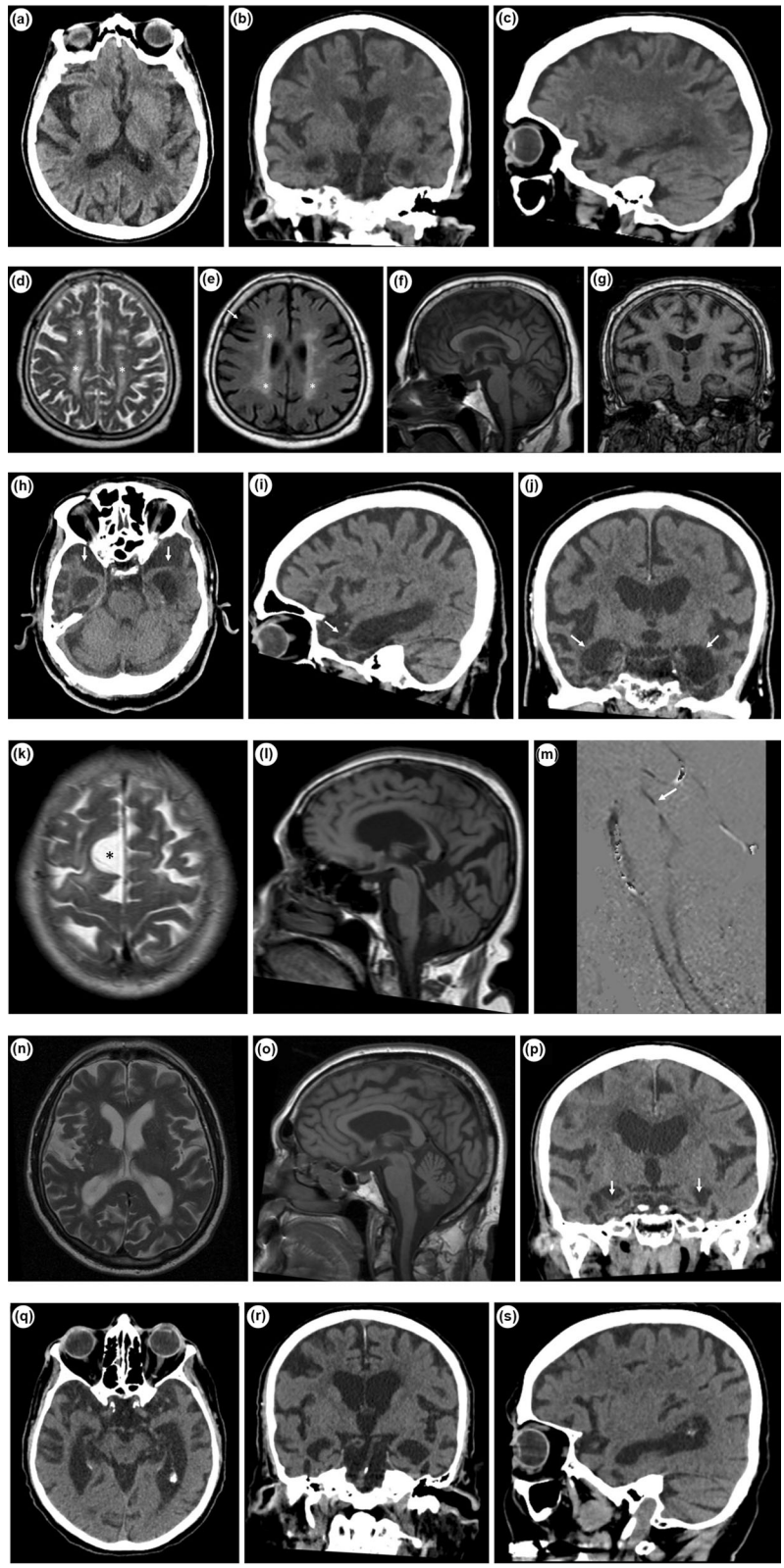
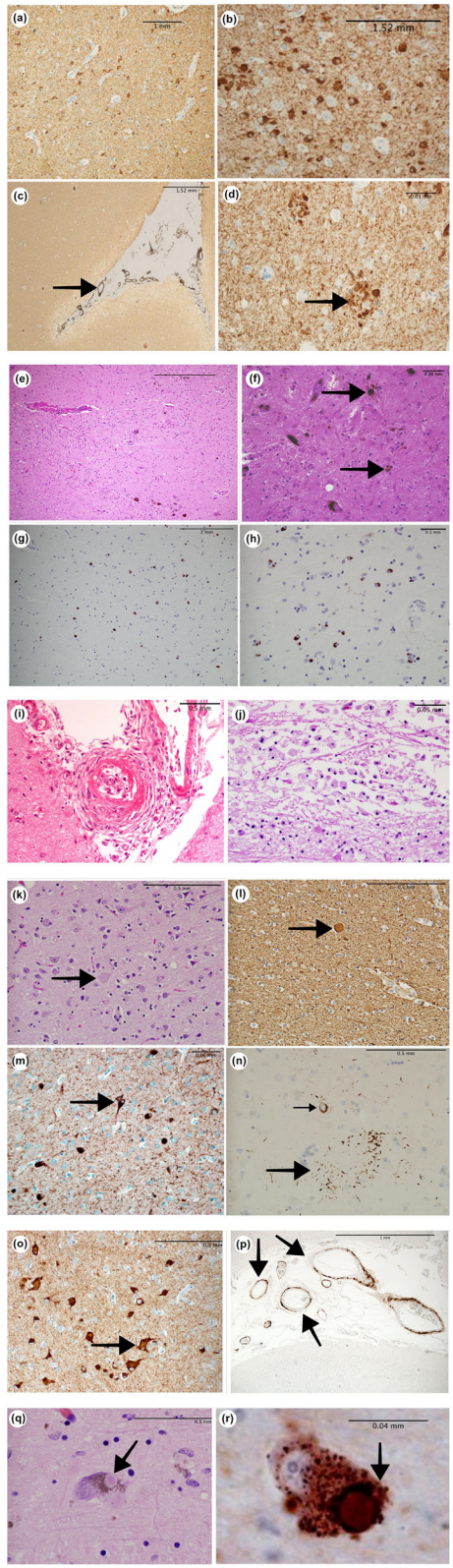


Fig. 1 Neuroimaging Findings of the Cases**. a** Axial, **b** coronal and **c** sagittal CT images show mild enlargement of the subarachnoid spaces and mild dilatation of the ventricular system; **d** Axial T2-weighted and **e** FLAIR MRI images show enlargement of the subarachnoid spaces (arrow) and hyperintensities (asterisks) in the subcortical white matter, **f** sagittal and **g** coronal T1-weighted MRI images reveal normal size of midbrain and both hippocampal bodies; **h** axial, **i** sagittal and **j** coronal CT images show enlargement of the subarachnoid spaces and dilatation of the ventricular system predominantly affecting the temporal region (arrows) and reduction in size of both hippocampal bodies; **k** axial T2-weighted, **l** sagittal T1-weighted MRI images show enlargement of the subarachnoid spaces (asterisk) and disproportionate dilatation of the ventricular system and **m** phase-Contrast MRI CSF flow measurements reveals increased velocity consistent with normal-pressure hydrocephalus (arrow); **n** axial T2-weighted sagittal T1-weighted and **o** MRI images show enlargement of the subarachnoid spaces and mild dilatation of the ventricular system, **p** coronal CT image reveals mild reduction in size of both hippocampal bodies (arrows); **q** axial, **r** coronal and **s** sagittal CT images show enlargement of the subarachnoid spaces, and dilatation of the ventricular system and reduction in size of both hippocampal bodies



◀**Fig. 2** Neuropathological Evaluations of the Cases. **a** Neurofibrillary tangles in the hippocampus and **b** neocortex, AT8 stain; **c** β-amyloid deposits in the walls of leptomeningeal vascular structures, B-amyloid stain; **d** dense-core plaques surrounded by dystrophic neurites (neuritic plaques), AT8 stain. **e** Decreased number of neurons in substantia nigra, H&E stain; **f** free neuromelanin is noted in the neuropil, H&E stain; **g** and **h** α-synuclein positivity in the cytoplasm of oligodendrocytes, α-synuclein stain. **i** Hyaline arteriolosclerosis in the walls of small vessels, H&E stain; **j** a remote infarct with reactive gliosis and macrophage infiltration, H&E stain. **k** Ballooning neurons in the cerebral cortex, H&E stain; **l** ballooning neurons shown by Neurofilament stain; **m** neuronal Tau-positivity, AT8 stain; **n** tau in the cytoplasm of oligodendrocytes (narrow arrow); named as "coiled body" and astrocytic plaques (wide arrow) surrounded by Tau-positive neurites, AT8 stain. **o** Neurofibrillary tangles in the neocortex, AT8 stain; **p** β-amyloid deposition in the leptomeningeal vessel walls, β-amyloid stain. **q** Typical view of a pigmented neuron with classical Lewy body; H&E stain; **r** α-synuclein positivity of the Lewy body; α-synuclein stain

Case 4

She presented to our Unit in December 2013 with speech alterations, clumsiness, rigidity in left upper limb, incontinence, parkinsonism, and falls with gait and balance disturbance with falls. Based on her clinical and radiologic findings, she was diagnosed with CBD and iNPH (Table 1; Fig. 1k–m). She died in September 2016 from aspiration pneumonia. The neuropathologic findings were compatible with CBD (Table 1; Fig. 2k–n).

Case 5

In 2013, he was admitted to our Unit with complaints of nervousness, gradual cognitive decline, and common memory disorders that began in 2003 at the age of 75 years. Based on these clinical and radiologic findings (Table 1; Fig. 1n–p), he was diagnosed with Probable AD. He died in September 2016 from septicemia. The neuropathologic findings were consistent with AD (A2, B2, C2) and CAA (Table 1; Fig. 2o, p).

Case 6

In 2014, he was admitted to our clinic with neuroleptic sensitivity, organized visual hallucination, variable cognitive status and postural hypotension. The brain

imaging findings are presented in Fig. 1q–s. The diagnosis of probable DLB was considered. He died in April 2017 from multilobar pneumonia. The neuropathologic findings were compatible with AD (A1, B2, C2) and DLB (Table 1; Fig. 2q, r).

Discussion

Brain bank and subsequent histopathological evaluation of the human brain in a systematic manner in large groups has had an important role in advancing our understanding of the pathogenesis of neurodegenerative diseases and age-related changes in the brain, while providing additional opportunities to other researches who can use these material and data (Vonsattel et al. 2008). The postmortem brain examination performed in the brain banks, established with these purposes, provides the accurate diagnosis of the participant whom the clinicians followed longitudinally with all available clinical, neuropsychological, radiological and biomarker data, giving the chance to correlate all these data with the neuropathological findings (Franklin et al. 2015), since no fluid and imaging biomarker is currently able to provide a definitive diagnosis. For instance, case 3 was diagnosed and followed as mixed dementia with AD and VAD, and the neuropathological evaluation of the brain identified VAD alone. Likewise, case 6, who underwent serial cerebrospinal fluid removal procedure (Isik et al. 2019), is also remarkable because he was followed with the clinical diagnosis of probable DLB and iNPH and was subsequently definitively diagnosed with AD as well as α-synucleinopathy based on histopathological evaluation. In fact, these findings are also very crucial for us to provide more accurate information for families. Therefore, brain banks are important for various reasons. For example, even in the most common dementia type AD, distinguishing variations is not always possible and the only way to achieve a definitive diagnosis is by histopathological evaluation of the brain. Nowadays, providing a single umbrella diagnostic terminology for these diseases is not satisfactory; determining the subgroups of these diseases is also essential, for instance, as in the cases of FTLD.

Some of our cases were of particular interest. Two of them were clinically thought to have NPH; however, no pathologic findings described in association with NPH in the literature were identified in either case (Leinonen et al. 2012), whereas CAA was demonstrated together with AD pathology in two cases with AD, compatible with the literature (Smith and Greenberg 2009).

We observed a rich spectrum of neurodegenerative diseases even in merely six autopsy cases that were clinically followed for at least 2 years. Thus, longitudinal assessment of the cases was an important component of this project. No doubt that these results are encouraging, but they also confirm the necessity of autopsy examination and brain banking in our country. Postmortem interval (PMI), i.e., the time between death and brain processing, was less than 12 h in our series. This is similar to a very experienced and funded donation program in which a median postmortem interval of 3.0-h was reported (Beach et al. 2015). Meanwhile, a recent study demonstrated that high-quality RNA could be obtained from tissue with a PMI of up to 36 h, and that, indicating stored tissue for more than two decades at − 80 °C retained its high quality of RNA and suitability for future researches (White et al. 2018). Most of our cases developed some type of terminal infection, giving them a chance to be hospitalized, and their death was not unexpected, providing a reasonable PMI; however, transfer of bodies from the homes of the patients or from nursing homes can sometimes be problematic.

We are aware that it is a small-scale study, and our clinical diagnosis was mostly confirmed by neuropathological diagnosis with unexpected findings. However, this is the first document of our ongoing effort in brain banking, which is one of the few sources in Asia. Recognizing the necessity of making a large number of donations, our immediate goal is to increase the number of patients enrolled in our program, as well as focus on research and work for our targets to join a brain network. Hopefully, some of the non-demented individuals whom we follow up in our department volunteer for donations and over time, they will provide control brain tissue for future studies.

Here, we briefly document our pilot efforts. Brain banking is a multifaceted joint effort that involves multiple subspecialties, with not only scientific, but also financial, social and educational aspects and ultimately depends on the participation of the patients and families. We hope that sharing our experiences and challenges will provide encouragement and a model to build on, both for ourselves and others

involved in the diagnosis of and research on the diseases of the brain.

Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

Ethical approval The study was approved by the Institutional Review Board of Dokuz Eylul University, Faculty of Medicine in Izmir (252-SBKAEK, 2015/13-18). All study procedures were performed in accordance with the principles laid down in the Declaration of Helsinki.

Informed consent Informed consent was obtained from all donors or their legal representatives.

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