Epilepsy and other comorbidities in Down syndrome

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Abstract

Down syndrome (DS) is the most common cause of intellectual disability. Due to many genetic and biochemical aberrations, people with DS suffer from several somatic disorders and concomitant diseases. Epilepsies occur more often than in the normal population, but less often than with intellectual disability of other causes. Epilepsies in DS have a trimodal distribution with age-related phenotypes, etiologies, and prognosis. The most common epilepsy syndrome in infancy is the often-self-limiting West syndrome; if Lennox–Gastaut syndrome occurs in the later course of development, the seizure prognosis worsens significantly. From the fourth decade of life, myoclonic epilepsy often occurs, which in combination with rapidly progressive Alzheimer’s dementia is life-limiting within a few years.

Keywords
Mental retardation · Seizures · Dementia · LOMEDS · Therapy

Introduction

Down syndrome (DS) is the most frequent condition leading to intellectual disability. The cause of DS is the numerical aberration of the 21st chromosome, leading to a typical and widely known phenotype and several age-related comorbidities. A relevant diagnostic and therapeutic challenge in people with DS (PWDS) is the increased prevalence of episodic disorders, including epileptic seizures of variable etiologies, falls, fainting, and other seizure mimics. Other patients with intellectual disabilities but it increases after that [15]. Since the 1940s, the average life expectancy of PWDS gradually increased from 12 years [31] to around 60 [20]. In our cohort, the median age at death of 42 PWDS was 58 years (see Fig. 1). A relevant gender difference in age-dependent mortality among PWDS is still under discussion due to inconsistent study results [12]. There is evidence of higher mortality in standard trisomy 21 or Robertsonian translocations compared with mosaic trisomy 21 [47].

General demographic data

The prevalence of DS varies considerably from country to country and continent to continent. With increasing maternal age at birth and changes in attitude to abortion and to people with disabilities, the prevalence of DS in the European Union increased from an average of 16 per 10,000 live births in 1990 to 23 in 2015 [16]. In the United States, population-based surveillance programs of birth defects showed that there were 14.85 newborns with DS per 10,000 live births between 2004 and 2010 [27]. Up to the age of 40, mortality in patients with DS hardly differs from

Pathophysiology

The cause of DS is trisomy of all or at least a critical portion of chromosome 21 (47 + 21). In about 95% of cases, DS follows a free (standard) trisomy 21, which means an additional chromosome 21 in all cells [3]. Approximately 3–5% of DS arises from a mosaic trisomy. Here two cell lineages exist, one with the diploid chromosome set and another one with an extra chromosome 21 [46]. Robertsonian translocations are found in about 4% of PWDS. Here, the long arm of chromosome 21 is attached to another chromosome (predominately...
Clinical features and comorbidities

People with trisomy 21 show a typical spectrum of physical stigmata, which enables this chromosomal disorder to be easily recognized postnatally (Table 1).

People with DS regularly suffer from many concomitant conditions and disorders in different organ systems. Due to the frequent occurrence of behavioral disorders related to cognitive deficits, the risk of undetected or misinterpreted somatic diseases is high. This diagnostic overshadowing is a lifelong challenge for PWDS, the attending doctors, and the care environment.

The most frequent associated disorders and diseases in DS and recommendations for assessment and management are listed in the Supplementary Information (Additional Table 1).

Epilepsy in Down syndrome

Compared with the overall cohort of people with ID, the prevalence of epilepsy in DS is lower. Due to differences in study populations, observation periods, and other methodological issues, data on epilepsy prevalence rates in DS vary considerably, ranging from 0 to 15.7% [33]. The prevalence of epilepsy in 256 PWDS from our group was 12.1% (n = 31/256). Since the clinical features, treatment strategies, and prognosis vary between the different age-related epilepsy syndromes, prevalence should be considered separately for each age group (see Figs. 2 and 3). The age-related epilepsy syndromes in DS are presumably based on chromosome 14), mainly following an error or misdivision after fertilization [5, 29].

Fig. 1 | Age-related mortality in 42 patients with Down syndrome (data from the Rotenburg Working Group)

| Age at death in years | No. of patients |
|-----------------------|----------------|
| 0-10                  | 15             |
| 11-20                 | 10             |
| 21-30                 | 10             |
| 31-40                 | 5              |
| 41-50                 | 2              |
| 51-60                 | 1              |
| 61-70                 | 1              |
| 71-80                 | 1              |

Fig. 2 | Prevalence of epilepsy in 256 people with Down syndrome according to various age groups: The age-independent prevalence of epilepsy was 12.1% (n = 31/256; data from the Rotenburg Working Group). LGS Lennox–Gastaut syndrome, LOMEDS late-onset epilepsy in Down syndrome

| Age in Years | No Epilepsy | LGS | Other Epilepsy Syndrome | LOMEDS |
|-------------|-------------|-----|-------------------------|--------|
| 11-15       | 40          | 10  | 5                       | 5      |
| 16-20       | 35          | 10  | 5                       | 5      |
| 21-25       | 30          | 10  | 5                       | 5      |
| 26-30       | 25          | 10  | 5                       | 5      |
| 31-35       | 20          | 10  | 5                       | 5      |
| 36-40       | 15          | 10  | 5                       | 5      |
| 41-45       | 10          | 10  | 5                       | 5      |
| 46-50       | 5           | 10  | 5                       | 5      |
| 51-55       | 5           | 10  | 5                       | 5      |
| 56-60       | 5           | 10  | 5                       | 5      |
| 61-65       | 5           | 10  | 5                       | 5      |
| 66-70       | 5           | 10  | 5                       | 5      |
| 71-75       | 5           | 10  | 5                       | 5      |
| 76-80       | 5           | 10  | 5                       | 5      |
| 81-85       | 5           | 10  | 5                       | 5      |
| 86-90       | 5           | 10  | 5                       | 5      |
different etiological processes. Late-onset epilepsy is usually linked to neurodegenerative processes of AD. By contrast, early-onset epilepsies seem to be associated with other epileptogenic abnormalities that have not been definitively elucidated causally. Histological findings revealed lower neuronal density and abnormal neuronal distribution, particularly in cortical layers 2 and 4 [13].

### Types and onset of epilepsy in DS

In general, epilepsy in DS has a trimodal distribution with age-dependent phenotypes, etiologies, and prognosis [35].

West syndrome is the most common encephalopathic epilepsy in children with DS. Infantile spasms occur in 5–15% of cases [22]. The age of onset ranges between 4 and 18 months [4]. The etiology is either non-structural and probably related to genetically caused neuronal dysfunction or attributed to secondary hypoxic encephalopathy following congenital heart disease or perinatal hypoxia–ischemia [42]. Other supposed mechanisms for epileptogenesis of early childhood epilepsies in DS are cortical dysgenesis, reduced density of cortical neurones and interneurons with a GABAergic inhibition, changes in the function of ion channels (especially the GluR5 receptor encoded on chromosome 21), and other metabolic disorders in DS [2]. The prognosis of West syndrome in DS is generally good, and long-term seizure control until early infancy is reported in up to 90% of cases [22]. Early and effective treatment of infantile spasms in children with DS may improve the neurodevelopmental outcome [14, 22, 44]. Furthermore, there is some evidence of an onset of infantile spasms after infancy being associated with a more favorable neurodevelopmental outcome [22].

### Childhood and early adulthood epilepsy

Lennox–Gastaut syndrome (LGS) is the second most common encephalopathic epilepsy in DS with a mean onset age of 9.1 years (range: 5–16) and about two-thirds of cases beginning after the age of 8 [17]. In contrast to West syndrome, seizure prognosis in LGS is much more unfavorable, and seizures often persist for life.

### Late-onset myoclonic epilepsy in Down syndrome (LOMEDS)

Since the 1970s, a growing number of publications described the phenomenon of progressive myoclonic jerks associated with dementia in late-stage DS [1, 9, 11, 13, 26]. Due to a uniform seizure semiology, course, and prognosis, the condition was finally recognized as a separate epileptic entity, abbreviated as LOMEDS [30]. Due to the life-limiting course of LOMEDS, comparability with progressive myoclonus epilepsy is postulated [11]. The mean age at LOMEDS onset is between 45.1 [1, 11, 13] and 59 years (own data; see Fig. 3). The development of dementia often precedes the seizures [2] and vice versa [26]. The average elapsed time between dementia and epilepsy was 6.9 months [11]. Based on the data from 12 patients with LOMEDS, three stages of the disease were described [11]:

- Stage 1 (mean age 51 ± 6.6 years): Dementia onset diffuse, electroencephalographic (EEG) changes during sleep, cerebral atrophy on computed tomography (CT) / magnetic resonance imaging (MRI).
- Stage 2 (mean age 51.4 ± 7.2 years): Onset of myoclonus (temporally associated with diffuse epileptiform changes in the waking phase), which antiepileptic drugs can often control.
- Stage 3 (mean age 54.8 ± 7.6 years): Epileptic myoclonus is replaced by non-epileptic myoclonus, cerebellar symptoms, severe dementia, and EEG photosensitivity.

Most patients with LOMEDS develop generalized tonic–clonic seizures as their disease progresses. The first occurrence of the latter often leads to the diagnosis. However, a careful history-taking retrospectively typically reveals pre-existing myoclonus, which is often not sufficiently recognized by the care environment by the time of the epilepsy diagnosis.

### Other epilepsy syndromes

Beyond these three main age-dependent categories, epileptic seizures for various reasons (e.g., stroke, traumatic brain injury, tumors, etc.) may occur at any age and do not differ from those in people without DS with or without intellectual disability.

Some authors reported a frequent occurrence of reflex seizures [38]. As with other encephalopathic epilepsies, long-lasting seizure clusters may evolve into refractory non-convulsive status epilepticus. Due to the frequent occurrence of concomitant cardiopulmonary diseases, the risk of sudden unexpected death in epilepsy patients with DS is assumed to be high [39].

### EEG findings

In principle, there are no pathognomonic EEG changes for DS. In the absence of seizures or severe neurodegeneration, most PWDS show regular EEG findings, especially in childhood and early adulthood [19]. Early pathological EEG changes are often minor and may present as an alteration of the EEG background activity.
In PWDS over 35 years of age, the frequency of generalized EEG slowdowns increases more strongly with progressive dementia [11, 28].

The EEG recordings of 25 adult PWDS from our group showed pathological beta activity (8%), generalized slow theta or delta activity (72%), and focal slow activity (12%). Interictal epileptiform discharges were generalized in 20% of cases and focal in 16% of cases, while ictal seizure patterns were 4.2% generalized and 8.3% focal.

With ongoing dementia in patients with LOMEDS, the physiological EEG background activity is increasingly replaced by generalized theta or delta activity [1], interictal fast spike waves, polyspikes, polyspike and waves, or photoparoxysmal response [9]. Myoclonic seizures are not necessarily associated with ictal epileptiform discharges, especially in the late course of the disease, as an indicator of non-epileptic myoclonus.

### CT and MRI findings in DS

The main finding in cerebral CTs and MRIs of PWDS with or without epilepsy is generalized atrophy, more rarely focal or infratentorial [1, 13]. In our series of 28 PWDS, generalized atrophy was found in 25% and post-ischemic lesions in 17.8%. In a series of 21 PWDS with LGS (average age at epilepsy onset: 9.1 years [17]), generalized brain atrophy was found in the majority of these patients too. Therefore, it can be assumed that generalized brain atrophy is not primarily a consequence of secondary neurodegeneration in DS with LOMEDS and AD, but probably represents a superordinate finding in DS.

Another common cerebral finding is basal ganglia calcification in 10.7–26.7% of PWDS ([23]; see Fig. 4). As one study described basal ganglia calcification more frequently in young PWDS [23], a relationship with the premature aging characteristic of DS was postulated [23]. The CT and MRI scans of our 28 adult PWDS showed mild (n = 10) or severe (n = 2) calcification in the basal ganglia. In contrast to the aforementioned study, we did not see any absolute predominance of basal ganglia calcification in younger patients. Histological and histochemical postmortem studies showed a maximum of calcification in the

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**Fig. 3** Incidence age of epilepsy in 31 people with Down syndrome (bar chart, left axis of ordinate) and age at the time of hospital admission for epileptic seizures (n = 40; red crossbars, right axis of ordinate; data from the Rotenburg Working Group). LGS Lennox–Gastaut syndrome, LOMEDS late-onset epilepsy in Down syndrome, Other epilepsy syndromes other than LGS and LOMEDS.

**Fig. 4** a Magnetic resonance imaging (MRI) of a 47-year-old patient with Down syndrome and structural focal epilepsy after ischemic infarction in the territory of the left middle cerebral artery. Note the generalized atrophy and the bilateral MRI hypointense signals in the basal ganglia (white arrows), indicating calcification. b Computed tomography scan of a 65-year-old patient with dementia and epilepsy. The generalized atrophy and hyperdense areas (dashed white arrows) indicate bilateral basal ganglia calcification.

[36]. Like people without DS, pathological EEG findings depend on epilepsy and its syndromic assignment.

People with DS with infantile spasms show typical interictal hypsarrhythmia with random high-voltage slow waves and spikes arising from multiple foci and spreading to various cortical areas. Seizure patterns present as low-amplitude fast activity [41]. While some authors used EEG-normalization after successful treatment of infantile spasms in PWDS for therapy control, there is no evidence of pretreatment EEG findings being predictive of seizure prognosis [22, 41].

The EEG in LGS typically consists of the triad of (a) slowed background activity, (b) slow spike and wave patterns (mostly bilateral synchronous, repeated at 1–2 Hz), and (c) burst of mostly bilateral low-amplitude fast activity with or without polyspike and wave complexes, the latter predominantly present in slow sleep or as seizure patterns in tonic seizures [8].
specific recommendations in LOMEDS

side effects should principally be preferred.

advantageous pharmacokinetic profile and fewer

treatment strategies, AED with a more fa-

have entirely lost their place in today’s

of a possible negative impact on cogni-

show frequent use of enzyme-inducing

antiepileptic drugs. Because of its po-

patients without DS, unclassified epilepsies

side effects should be avoided. As in pa-

tic drugs with potential arrhythmogenic

show frequent use of enzyme-inducing

The principles of antiepileptic pharma-

– Structured anamnesis with patient and caregivers

– Neurological examination

– Note possible triggers (e.g., metabolic disturbances, infections, sleep

depression, medication failure, traumatic brain injury)

– Home videos and/or video-EEG as soon as possible after episodes or

relevant changes in seizure semiology

– Limit long-term antiepileptic medication to a high risk of recurrence,

dangerous, and/or distressing seizure semiology, and realistic expecta-

tion of significant seizure reduction*, **

– Unless established focal origin of seizures, use broad-spectrum AED*

– Monitoring therapy is preferred*

– Monitoring seizure frequency, treatment compliance, tolerance, and

possible interactions (drug monitoring if reasonable)*

When choosing suitable AED, administra-

tion aspects (e.g., suspension vs. tablets,

tablet size, dosage frequency, etc.) should

be considered since a significant propor-

tion of PWDS show a general aversion to

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Zusammenfassung

Epilepsie und andere Komorbiditäten beim Down-Syndrom

Das Down-Syndrom (DS) ist die häufigste Ursache für Intelligenzminderung. Aufgrund vieler genetischer und – in der Folge – biochemischer Aberrationen findet sich beim DS eine große Anzahl somatischer Störungen und Begleiterkrankungen. Epilepsien treten häufiger auf als in der Normalbevölkerung, jedoch seltener als bei Intelligenzminderung anderer Ursache. Epilepsien bei DS haben eine trimodale Verteilung mit altersabhängigen Phänotypen, Ätiologien und Prognosen. Das häufigste Epilepsiesyndrom im Kleinkindalter ist das oft selbstlimitierende West-Syndrom. Kommt es im späteren Entwicklungsverlauf zu einem Lennox-Gastaut-Syndrom, verschlechtert sich die Anfallsprognose relevant. Ab der 4. Lebensdekade kommt es häufig zu einer Myoklonusepilepsie, welche in Verbindung mit einer rasch progredienten Alzheimer-Demenz innerhalb von wenigen Jahren lebensbegrenzend ist.

Schlüsselwörter
Geistige Behinderung · Epileptische Anfälle · Demenz · LOMEDS · Therapie

Summary

Due to different epilepsy syndromes, the high number of possible comorbidities, and an early onset of neurodegeneration, the diagnosis and treatment of epilepsy and other seizure disorders pose a challenge in the care of people with Down syndrome; this requires a transparent, coordinated, and interdisciplinary care setting. A summary of general and specific recommendations for diagnosing, treating, and following up people with Down syndrome and epilepsy is listed in Table 2.

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Declarations

Conflict of interest. F. Bösebeck declares that he has no competing interests.

Due to the nature of this research, participants of this study did not agree for their data to be shared publicly, and thus supporting data are not available.

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