Chapter 7
Epidemiology of TGA (1): Possible Predisposing Factors

Abstract This chapter examines factors identified in clinical and epidemiological studies as predisposing to episodes of TGA. None of these factors is either necessary or sufficient for the occurrence of TGA. Nevertheless, the more consistently implicated predisposing factors, such as a personal history of migraine, may give insights into disease pathogenesis. Precipitating factors for TGA are considered in the subsequent chapter.

Keywords TGA · Incidence · Predisposing factors

A number of factors have been described which though temporally remote from the onset of an attack of TGA may nevertheless predispose to it (i.e. increase the chance or risk of its occurrence). Of the reported predisposing or risk factors for TGA, some are more certain than others, based on the existing evidence.

7.1 Incidence

As a transient condition, of duration less than 24 h (if concordant with clinical diagnostic criteria [1] (see Table 2.1), no meaningful data on TGA prevalence can be collected, rather only incidence.

A limited number of studies of TGA incidence have been reported (Table 7.1) [2–10], most population-based [2–6, 8, 10] but some based on experience at a single centre [7, 9]. Annual incidence rates in these studies range between 2.9 and 12/100,000 of the population. The highest of these measures was recorded at Davos, Switzerland, located at relatively high altitude, prompting the suggestion that low temperature might contribute to TGA pathogenesis [9] (see Sect. 8.3). Govoni et al. noted a statistically significant difference in incidence rates with level of urbanisation and population density, prompting the suggestion that the stress related to urban living might contribute to pathogenesis [10] (see Sect. 7.10). Of course, one may

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Table 7.1 Incidence studies of TGA

Reference	Study location	Annual incidence	Sex-specific incidence
Miller et al. (1987) [2]	Rochester, Minnesota, USA	5.2/100,000	–
Koski and Marttila (1990) [3]	Turku, Finland	10/100,000 (32/100,000 amongst those ≥50 years)	–
Hodges (1991) ([4], p.13)	Oxford, UK	3/100,000	–
Matias-Guiu et al. (1992) [5]	Alcoi, Spain	2.9/100,000	–
Lauria et al. (1997) [6]	Belluno, Italy	10.4/100,000 (crude); 8.6/100,000 (adjusted); 5.81/100,000 (retrospective study)	9.35/100,000 for men; 11.37/100,000 for women
Berli et al. (2009) [7]	Uster Hospital, Switzerland	6.8/100,000	–
Brigo et al. (2014) [8]	Merano, province of Bolzano, Italy	9.6/100,000 (crude); 6.4/100,000 (adjusted)	10.1/100,000 for men; 8.9/100,000 for women
Erba and Czaplinski (2017) [9]	Regional Hospital, Davos, Switzerland	12/100,000	–
Govoni et al. (2020) [10]	Ferrara, Italy	10.10/100,000 (crude)	8.40/100,000 for men; 11.60/100,000 for women

posit alternative explanations for these observations, such as underascertainment of cases in rural areas with less readily available access to medical services.

Only limited data on sex-specific incidence rates are available (Table 7.1), permitting no definitive conclusion as to whether this is greater in men or women. For example, the gender difference observed by Govoni et al. was not statistically significant [10].

7.2 Chronobiology: Time of Onset by Day, Month and Season

Quinette et al. reported a peak of TGA occurrence in spring and summer in their literature review (n = 46), but in their own cohort they found TGA episodes were distributed evenly throughout the year [11].

Keret et al. [12, 13] examined the seasonal incidence of TGA cases seen in a single tertiary care centre in Israel. Initially, they reported (in abstract) a series of 86 TGA patients (F:M = 54:32, 63% female; mean age 61 ± 10.3 years) seen over the period 2005–2013, in whom they found two incidence peaks, in November–December and

in March [12]. In a later, substantive, paper, the time frame was broadened to 15 years (2000–2014), in which period 154 TGA patients were seen (F:M = 91:63, 59% female; mean age 62.8 ± 10.6 years), with incidence peaks in winter (December) and spring (March) [13]. The authors concluded that seasonal factors might contribute to TGA pathogenesis.

Govoni et al. found TGA cases to be evenly distributed by month and season in their incidence study [10].

Hoyer et al. [14] analysed data from two large TGA cohorts (n = 404 and 261, respectively) and found no variation of TGA occurrence by day of the week, month or season of the year, in contrast to a robust circadian rhythm of incidence (mid-morning, late afternoon) (Sect. 2.1.3).

In the author's series, TGA seasonal incidence has been examined in two ways: by meteorological season (for the northern hemisphere: Spring = March–May; Summer = June–August; Autumn = September–November; Winter = December–February) and by quarter of the year (Q1 = January–March; Q2 = April–June; Q3 = July–September; Q4 = October–December) approximating to the astronomical seasons, defined by the solstices and equinoxes, as in the Gregorian calendar (Fig. 7.1a, b respectively; updated from [15, 16]). The null hypothesis that cases did not differ by either season or quarter was not rejected.

Rather than season per se, ambient temperature might be a predisposing and/or precipitating (see Sect. 8.3) factor for TGA. One study suggested an association between TGA occurrence and low ambient temperature [17]. Cases of TGA related to high altitude [9] might also reflect a relationship to ambient temperature.

7.3 Place of Onset: Geographical Distribution

Cases of TGA have been reported from all the inhabited continents of the world, even remote locations such as Polynesia [18]. There do not appear to be any geographical "hot-spots" of high incidence, but to the author's knowledge, no systematic study of population-based prevalence has been undertaken.

It has been reported that TGA (and migraine) is more common in Latin American patients with the antiphospholipid syndrome (APS+) than in European APS+ patients [19].

7.4 Patient Age

Most studies find that TGA is typically a condition of mid-life, particularly affecting those in their 50s and 60s, and distinctly unusual at earlier ages (<40 years; Table 7.2).

In the survey of the author's experience (n = 50), median patient age was 64.8 years (Fig. 7.2). Those acute amnesic patients excluded for not conforming to

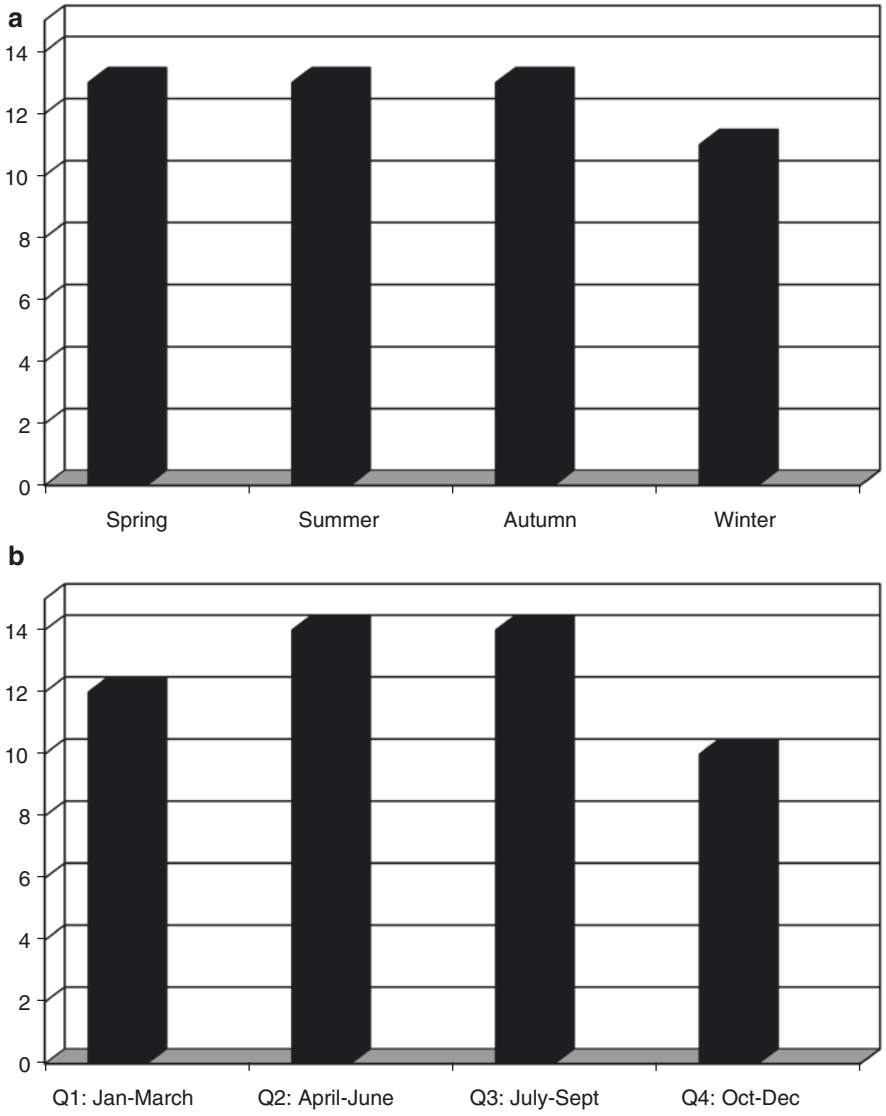

Fig. 7.1 Distribution of consecutive cases fulfilling diagnostic criteria for TGA (n = 50) seen in author's clinic over 20-year period (2002–2021). (**a**) by meteorological season (Northern Hemisphere) of presentation (Spring = March–May; Summer = June–August; Autumn = September–November; Winter = December–February). (**b**) by quarter of presentation (Q1 = January–March; Q2 = April–June; Q3 = July–September; Q4 = October–December)

Hodges and Warlow's diagnostic criteria [1], and not definitely diagnosed with TEA based on Zeman's criteria [29], were slightly younger (median 62.2 years; Fig. 7.3). In the Oxford TGA study, non-TGA cases were non-significantly younger than definite TGA cases (mean age 60.8 vs 62.3 years) ([4], p.113–4, Fig. A.2).

Table 7.2 Age and gender of TGA patients (selected reports)

Reference	N	Age (years)	Gender
Hodges and Warlow (1990) [1]	114 prospective, single clinic (UK)	62.3 ± 8.5 (range 35–85)	66% male
Hodges and Warlow (1990) [1]	752 literature review	61.2 (range 20–92)	53% male
Quinette et al. (2006) [11]	142 prospective, single clinic (France)	63.9 ± 8.3 (range 32–81)	33.1% male
Quinette et al. (2006) [11]	246 (age) 1333 (gender) literature review	60.3 ± 9.6 (range 21–85)	46.4% male
Berli et al. (2009) [7]	20 retrospective, single centre (Switzerland)	67 ± 7.3 (range 58–86)	60% female
Agosti et al. (2010) [20]	243 consecutive enrolment, 2 hospitals (Italy and Lebanon)	64.0 ± 8.3	44.9% female
Ahn et al. (2011) [21]	203 retrospective, single centre (South Korea)	60.1 ± 9.3	41.4% male
Ryoo et al. (2012) [22]	73 single centre (South Korea)	59.7 ± 9.5 (range 43–76)	72.6% female
Döhring et al. (2014) [23]	113 single centre (Germany)	65.4 ± 7.6	54.9% female
Keret et al. (2016) [13]	154 retrospective, hospital data (Israel)	62.8 ± 10.6	41% male
Arena et al. (2017) [24]	221 epidemiology database for single county (USA)	65.6 ± 12.2	50.2% female
Alessandro et al. (2019) [25]	203 single centre (Argentina)	65 (20–84)	52% female
Higashida et al. (2020) [26]	261 databases of four medical centres (Japan)	65.3 ± 8.6	61% female
Morris et al. (2020) [27]	1044 retrospective, single centre (USA)	75.0 ± 11.5	55.1% male
Szabo et al. (2020) [28]	390 prospective, single centre (Germany)	66.1 ± 7.8 (range 37–86)	39.5% male
Larner (2022)	50 prospective, single clinic (UK)	64.8 ± 6.9 (range 47–78)	58% female

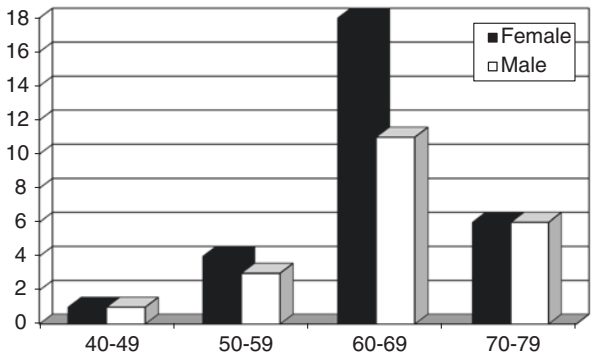

Fig. 7.2 Age and gender distribution of consecutive cases fulfilling diagnostic criteria for TGA ($n = 50$) seen in author's clinic over 20-year period (2002–2021)

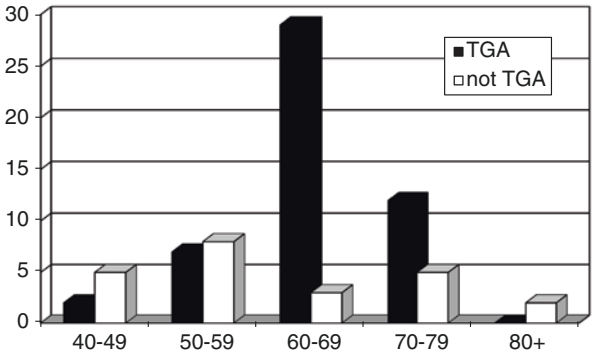

Fig. 7.3 Age distribution of consecutive cases of transient amnesia fulfilling ($n = 50$) and not fulfilling ($n = 23$) diagnostic criteria for TGA seen in author's clinic over 20-year period (2002–2021)

Although cases of TGA have been reported in young people (e.g. [30–35]), these are rare, and some predate diagnostic criteria so that caveats about the diagnosis apply (Sect. 1.3 and Sect. 2.2.2). Some have occurred in the context of exercise [33, 35], others in the context of migraine [30, 35]. An adolescent with two episodes labelled as TGA precipitated by emotion had temporal and occipital lobe embolic infarction in the context of congenital heart disease [34], raising the possibility of epileptic events. Certainly, the differential diagnosis requires careful consideration in patients under 40 years of age who are suspected of having TGA, and should include the possibility of acute confusional migraine [36] (Sect. 3.4.1; Case Study 3.3).

Cases of TGA are rarely reported in the oldest old people (>80 years). It is not clear whether this is simply underascertainment, against the background of the increasing prevalence of memory disorders in older people, or whether the elderly oldest old people are in some way protected from TGA, mechanism(s) unknown (see Sects. 9.4 and 9.7.6).

7.5 Patient Gender

The precise distribution of TGA by gender is uncertain, with different findings in different studies (Table 7.2). For example, in Hodges and Warlow's series of 114 patients, males outnumbered females [1], whereas Quinette et al. found no significant gender difference when pooling 1333 patients reported in 52 published case studies and 34 group studies (46.4% male, 53.6% women; $\chi 2 = 0.48$; df = 1; $p = 0.49$), although in their own series of 142 cases there was a 2:1 F:M preponderance (66.9% female, 33.1% male) [11].

A four-year survey (2002–2005 inclusive) of the author's practice [37] identified eight cases fulfilling diagnostic criteria for TGA, all of whom were female (age range 48–71 years). The preponderance of female cases was confirmed when the

survey was extended to 6 years (F:M = 10:1 = 91% female) [38], 9 years (F:M = 11:5 = 69%) [39], 12 years (F:M = 14:10 = 58%) [40], 15 years (F:M = 20:14 = 59%) ([16], p.99), and at 20 years, the ratio was F:M = 29:21 (= 58%; Fig. 7.2). The falling ratio may indicate that the initial female preponderance was simply a chance observation associated with the small number of cases seen.

7.6 Patient Ethnicity

There do not seem to be any studies specifically addressing the role of patient ethnicity in the pathogenesis of TGA. It has certainly been reported from around the world, including relative geographical isolates such as Polynesia [18]. A nationwide inpatient sample analysis from the USA, including nearly 50,000 TGA patients, explored race-specific variables associated with TGA and reported that the odds of being diagnosed with TGA was lower for African Americans, Hispanics and Asians/others compared to Whites [41].

7.7 Patient Social Class

Hodges found no definite evidence of differences in TGA cases according to social class, although there was a non-significant difference in the proportion of patients from social class I ([4], p.15).

7.8 Family History of TGA

TGA has generally been considered as a sporadic condition. Although there is no suggestion that it has a monogenic Mendelian pattern of inheritance, nevertheless occasional familial clusters have been reported in the literature (Table 7.3). Hodges and Warlow suggested that the overall rate of familial TGA in their series was 1.75% (95% confidence interval = 0%–4.2%) [1].

Most familial reports have involved siblings (Table 7.3; Case Study 7.1), with two sets of twins (one monozygotic [51], the other probably so [45]), with only occasional definite [43, 47, 49, 57] or possible [1, 48, 56] instances of parental involvement. Only one account of familial involvement with more distant relatives, specifically a proband whose two aunts were apparently affected [52], has been found.

Case Study 7.1: Family History of TGA

Following a previous publication on the subject of familial TGA [56], the author was contacted by a family from western Canada with a family history of TGA. A 61-year-old woman had an episode of amnesia following a bike ride and during a period of emotional stress. Features were typical for TGA. CT brain scan and CT angiogram of the circle of Willis performed on the same day were both normal. There was no recurrence over the next five years. However, at that time her 60-year-old brother had an episode of TGA. After skiing, he took a chairlift to ascend, but on getting off had no recollection of the ride up or where he was. He knew who he was but had no understanding of why he was on a mountain. He repeatedly asked where his wife was. From the time on the chair lift to when he started to retain short-term memory was approximately 1 hour. There was no personal or family history of migraine.

Table 7.3 Reports of familial cases of TGA and their history of migraine (adapted and updated from [16], p.100–1) (see Table 6.2 for reports of recurrent TGA in these cases)

Reference	TGA patient details	Migraine history
Corston and Godwin-Austen (1982) [42]	Four male siblings, 2–3 attacks each, when aged in 60s and 70s. Three-fourth had TGA attacks in context of exercise	None
Munro and Loizou (1982) [43]	Two siblings (F:M) and their father, one to three attacks in 50s to 60s	Not commented on
Stracciari and Rebucci (1986) [44]	Two siblings (F:M), attacks in 70s and 50s respectively, latter associated with exercise on a windy day	Both had prior history of migraine, F until menopause, M in adolescence
Dupuis et al. (1987) [45]	Twin sisters (probably monozygotic), attacks (2 and 1, respectively) in 60s	Both migraineurs since adolescence; both attacks in first sister followed by severe migraine
Hodges and Warlow (1990) [1]	60-year-old man; sister, mother, also affected. 66-year-old woman; brother also affected	Not specifically commented on
Agosti et al. (2007) [46]	Three female siblings in their 60s, attacks following emotional upset, cold shower and sexual intercourse, respectively	Not commented on
Vyhnalek et al. (2008) [47]	Male, 2 episodes aged 52, 54. Father 1 episode aged 50; sister 1 episode aged 52	Proband had migraine without aura from adolescence; no migraine in father or sister
Segers-van Rijn and de Bruijn (2010) [48]	Four siblings (3F:1M) and possibly their mother, attacks after exercise (3) and air travel (1), and on birthday; attacks between 50s and 70s	One of the female siblings had history of migraine with aura

Table 7.3 (continued)

Reference	TGA patient details	Migraine history
Galovic et al. (2011) (abstract only) [49]	4 siblings and their mother; AAO ca. 70 years, all single episodes, several associated with Valsalva manoeuvres	Not commented on
Goossens et al. (2011) (abstract only) [50]	Two sisters, attacks at age 61 and 57, respectively	Both had migraine with aura from adolescence; elder had headache at time of TGA
Maggioni et al. (2011) [51]	Two monozygotic twin brothers aged 50 and 49 at onset	Elder had 5–6 attacks per year during migraine without aura (MO) attacks, frequency reduced by verapamil and valproate; younger had 4 episodes all during MO
Davies and Larner (2012) [52]	Female and two maternal aunts, attacks in 50s and/or 60s, after exercise in the index case	Migraine in index case, no information on other cases
Dupuis et al. (2013) (abstract only) [53]	7 families	No other details available from published abstract
Dandapat et al. (2015) [54]	Two sisters, age 57 (precipitated by sex) and 71	No history of migraine; older sister had mild headache at time of TGA
Dupuis et al. (2017) (abstract only) [55]	10 families in cohort of 219 patients	History of migraine reported to be more frequent in familial cases
Larner (2017a) ([16], p.101) (personal communication; Case Study 7.1)	Two siblings (female aged 61, male aged 60), both associated with exercise	No history of migraine
Larner (2017b) [56]	Male, 2 episodes aged 61 and 64; father also reportedly had TGA, 2–3 episodes (uncertain)	No history of migraine
Larner (2017c) (unpublished, personal communication)	Two siblings (male aged 60, female aged 70), both associated with exercise	No history of migraine
Larner (2018) [57] (personal communication)	Two male siblings and their mother. Younger male sibling had three events, aged 60, 63, 66, first two after exercise (cycling)	No personal history of migraine in male with recurrent events
Larner (2018) [57] (personal communication)	Two female siblings. Younger female sibling had two events, aged 54 and 55, second after sexual activity. Older sibling had single event, aged 60, after sexual activity	Both siblings had history of migraine
Larner (2019a) (unpublished, personal communication)	Three siblings (1F:2M). Female 3 events aged 63, 68, 70, first after emotional upset, second after exercise. Single events in male siblings.	No personal history of migraine in female with recurrent events
Larner (2019b) (unpublished, personal communication)	Two siblings (1F:1M). Female 67, male 55, both exercise associated (gardening, running).	Female 2 or 3 migraines about 30 years earlier; male 1 migraine 25–30 years earlier
Larner (2019c) (unpublished, personal communication)	Two female siblings aged 79 and 75.	No history of migraine

Summing all these publications from which adequate information is available (hence excluding [49, 53, 55]), there were 53 patients from 21 families, with a slight female preponderance (F:M = 30:23, 57% female), with all TGA episodes occurring in the sixth to eighth decades of life (Table 7.3). Although details were incomplete, at least 16 (=30%) of these individuals had a history of migraine (11F:5M). Two (female) patients were reported to have had migraine-type headaches at the time of or immediately after TGA episodes [45, 50], and two monozygotic male twins had episodes during attacks of migraine without aura [51]. One man with a history of migraine without aura dating from adolescence had a first attack of TGA one month after withdrawal from a beta-blocker (atenolol) prescribed for hypertension for the previous 5 years [47].

Dupuis et al. [53] also examined the possibility of an hereditary aetiology for TGA. In a publication appearing in abstract only, they identified 9 families in the literature and 7 families "reported recently by one of us" (I have been unable to locate such a report, so presume it must have appeared in abstract only). Six of their personally observed families were from the same hospital and were compared to a database of 127 consecutive TGA patients. The 6 families were said to represent 4.7% of 127 TGA cases (with reported 95% CI 1.05%–8.45%), which seems to imply only 6 cases, so presumably the familial cases were by report rather than by direct observation.

References to the 9 families in the literature were not given, but I presume them to be those reported by Corston and Godwin-Austen [42], Munro and Loizou [43], Stracciari and Rebucci [44], Dupuis et al. [45], Hodges and Warlow ([1], 2 families), Agosti et al. [46], Segers-van Rijn and de Bruijn ([48]; co-authors on the abstract), and Goossens et al. ([50], co-authors on the abstract), but not those reported by Galovic et al. [49], as these were presented in abstract only, Vyhnalek et al. [47], as the title of this paper gives "familiar" rather than "familial" TGA, and perhaps Maggioni et al. [51], as too recent to be included. Summing all 16 families, Dupuis et al. reported 41 cases with mean age 61.8 years, 22 female (=53.7%), and 12 migrainous (=29.3%), with migraine and stress as "frequent risk factors". The familial cases were reported to be indistinguishable from sporadic cases [53].

Familial cases of transient epileptic amnesia (TEA; Sect. 3.2) have rarely been reported, and never, to my knowledge, in a substantive paper [58, 59].

Possible genetic contributions to the pathogenesis of TGA are discussed in Sects. 9.5 and 9.7.

7.9 Migraine

Migraine may be a symptomatic cause of amnesia, which enters the differential diagnosis of TGA (Sect. 3.4.1; Table 3.2). This may require particular consideration in young people with attacks purported to be TGA (for example, acute confusional migraine; Sect. 7.4). Migraine might also be considered as a precipitating factor for TGA (Sect. 8.6).

The possible association between TGA and migraine was recognised early in the history of TGA (Sect. 3.4.1): for example, Evans in 1966 reported two patients with attacks suggestive of TGA in the context of a history of migraine [60]. Other possible early reports include those of Frank (1976; amnesic episodes in migraine, "Migranedammerattacken", apparently identical with TGA) [61] and Caplan et al. in 1981 [62].

Migraine was more common in TGA patients than in both normal and TIA control subjects in the case–control study reported by Zorzon et al. [63]. A case–control study by Schmidtke and Ehmsen [64] showed a markedly increased prevalence of migraine in TGA patients and also of episodic tension-type headache. Quinette et al. used cluster hierarchical analysis of TGA cases to show that in younger patients a history of headache may be a risk factor for TGA [11]. Arena et al. followed up 221 TGA cases for a mean of 12 years and found that previous migraine was more common than in a matched control group [24].

A population-based cohort study from Taiwan found that migraine was associated with a higher risk of TGA. Over 150,000 migraine patients and their matched controls were followed up for a mean of 3 years, during which time the migraine cohort had a greater risk of developing TGA than the controls (7.59 vs 3.06/100,000 person-years, incidence rate ratio = 2.48). Female patients with migraine aged 40–60 years had a significantly higher risk of developing TGA (incidence rate ratio = 3.18). Incidence rates did not differ between migraine patients with or without aura [65].

In a nationwide inpatient sample analysis including nearly 50,000 TGA patients, patients with migraine were found to have a greater odds ratio (5.98, 95% CI 5.42–6.60) of having TGA [41].

A personal or family history of migraine is associated with, and may therefore be a risk factor for, recurrent TGA [25, 27, 66, 67]. A relationship to migraine might potentially explain a female preponderance of TGA cases, if such exists (Sect. 7.5), since migraine is more common in women. Many of the familial examples of TGA (Sect. 7.8) had migraine comorbidity (see Table 7.3).

The possible role of migraine in the pathogenesis of TGA is discussed in Sect. 9.4.

7.10 Patient Personality Traits and Psychological Factors

Acute TGA episodes may be associated with symptoms of anxiety and depression (Sect. 2.1.2) [68]), but whether these behavioural features are simply part of the acute phenomenology or reflect premorbid psychopathology or personality traits has been uncertain. Neri et al. found depressive symptoms, assessed by the Geriatric Depression Scale, in 8 of 20 TGA patients [69]. Inzitari et al. found that TGA patients scored higher on a scale that measured phobic attitudes than control patients with TIAs, suggesting that emotional arousal may be involved in TGA [70]. This may be consistent with the observation of emotional factors as precipitating factors of TGA (Sect. 8.1).

An increased frequency of personal and family history of psychiatric diseases was noted in TGA patients followed up for about 7 years and compared to TIA controls by Pantoni et al. [71]. Fischer et al. found indications of depressive disorders at the time of onset in 67.9% of a group of 28 TGA patients, compared to 12.5% in 25 TIA patients, prompting the authors to suggest that depressive disorders predispose to TGA, perhaps due to an imbalance in hippocampal neurotransmitters [72]. Quinette et al. found a past history of anxiety/depression in around 20% of the 129 patients in their personally observed series for whom this was investigated, and found a high frequency of psychological and emotional instability in TGA patients. They were of the view that TGA in women was associated with a history of anxiety and a pathological personality [11]. A study by Döhring et al. found a higher level of anxiety in patients who experienced a stress-related TGA precipitant compared to both those who did not and to controls, suggesting that increased susceptibility to psychological stress may be a risk factor for TGA [23].

The possibility that stress related to urban living might account for the differential incidence of TGA seen with level of urbanisation and population density [10] has been mentioned (Sect. 7.1), likewise that emotional stress might account for increased incidence of TGA following the onset of the COVID-19 pandemic [73] (Sect. 3.5.2).

7.11 Vascular Risk Factors and Stroke

Because of its sudden onset, the possibility that TGA may have a vascular aetiology has been considered from the time it was first described (Sect. 3.1). Transient ischaemic attack (TIA) and stroke enter the differential diagnosis of TGA (Sect. 3.1). Hence, the examination of vascular risk factors in TGA patients and comparison with TIA patients has been undertaken in a number of case–control studies.

The Oxford TGA study found no difference in the prevalence of vascular risk factors between prospectively identified TGA patients and matched controls, but significant differences with matched TIA controls ([4], p.125–32). In a prospective case–control study, Zorzon et al. found no evidence of increase in any vascular risk factor in 64 TGA patients compared with matched TIA patients and normal controls [63].

Retrospective studies have sometimes reached different conclusions. Hypertension was noted to be the most common vascular risk factor in one retrospective series of TGA cases (11/28) although no vascular risk factor was noted in about half of the cases [74]. Prevalence of vascular risk factors was found to be higher in TGA patients than healthy controls by Santos et al. [75]. A retrospective study of 131 TGA patients seen between 1993 and 2004 found a higher incidence of hypertension compared to 262 TIA patients, whereas diabetes mellitus, ischaemic heart disease and cerebrovascular disease were more common in the latter group [76]. In a cohort of TGA patients identified in the Framingham Heart Study, no significant differences were observed in the prevalence of vascular risk factors with

a control group [77]. A retrospective case–control study of 293 TGA patients published by Jang et al. found a significantly higher prevalence of ischaemic heart disease and hyperlipidaemia than in TIA controls, although the latter had a higher prevalence of hypertension, diabetes mellitus, ischaemic stroke and atrial fibrillation. TGA patients also had a significantly higher prevalence of hyperlipidaemia, previous ischaemic stroke and ischaemic heart disease when compared to age- and sex-matched normal controls [78]. The difference between the findings of studies with prospective or retrospective design should be noted.

Tuduri et al. found no clinical differences between TGA patients with and without vascular risk factors [79]. Toledo et al. compared "unique-TGA" cases ($n = 98$) with "recurrent-TGA" cases ($n = 26$) and found that the latter had the same vascular risk factors as a comparison group of TIA patients. Furthermore, the recurrent-TGA patients had a significantly more frequent history of stroke and a trend to suffer new ischaemic events than patients in the unique-TGA group, prompting the suggestion that recurrent TGA be considered a manifestation of ischaemic cerebrovascular disease [80], but in a later study they appeared to revise their views [81] (see Sect. 6.2.2). Agosti et al. divided TGA patients ($n = 243$) according to whether or not they had evidence for internal jugular vein valve incompetence (IJVVI), a factor which might be relevant to TGA pathogenesis (Sect. 4.3.3.2 and Sect. 9.2.2). TGA patients with IJVVI showed a higher frequency of precipitating factors but had fewer vascular comorbidities than TGA patients without IJVVI, suggesting that different mechanisms might operate in individual episodes of TGA [20].

In a systematic review, Liampas et al. retrieved 23 observational studies from which they concluded that diabetes was protective for TGA, dyslipidaemia was not related, and only severe hypertension was associated [82]. Rogalewski et al. found that acute hypertensive peaks showed a strong association with TGA [83].

In conclusion, single episode TGA does not seem to share the same vascular risk factors as TIA but this might not necessarily be the case for recurrent episodes of TGA. The possible role of vascular pathology, arterial or venous, in the pathogenesis of TGA is considered in Sect. 9.2.

7.12 Structural Brain Lesions

Occasional reports associating TGA with the presence of a structural brain lesion have appeared. These most usually concern brain tumours, but even here the cases are rare, with one systematic review finding only about 20 cases [40] (Table 7.4). No cases of brain tumour were encountered in some large series (e.g. [1].). Agosti et al. would classify such patients as "TGA-b", in distinction from primary cases (i.e. no brain lesion seen on neuroimaging) labelled "TGA-p" [100] (see Sect. 2.2.3).

Many of the reports of brain tumour associated with TGA predate widely accepted clinical diagnostic criteria for TGA, and for this reason, some cases might be excluded as not conforming to the diagnosis. For example, in one case the amnesic episode lasted more than 24 h [84] and in another progressive memory problems

Table 7.4 Reports of concurrence of TGA with brain tumour (adapted from [16], p.106–8, and [40])

Reference	Patient details	Histology	Location
Aimard et al. (1971) [84]	F65	Glioblastoma	"Trigone and diffuse"
Hartley et al. (1974) [85]	M62	Chromophobe adenoma	Pituitary
Boudin et al. (1975) [86]	F73	Glioma	Posterior limbic system, bilateral
Lisak and Zimmerman (1977) [87]	M70	Unknown	L temporo-parietal
Shuping et al. (1980) [88]	M60	Glioblastoma	L hippocampus
Findler et al. (1983) [89]	M67	Metastasis	Non-dominant hemisphere
Meador et al. (1985) [90]	F47	Meningioma	R temporal lobe
Riva et al. (1985) [91]	F64	Meningioma	Olfactory bulb
Collins and Freeman (1986) [92]	M61	Meningioma	R parietal region
Matias-Guiu et al. (1986) [93]	M-	Unknown	R temporal lobe
Araga et al. (1989) [94]	F59	Meningioma	Falco-tentorial region
Cattaino et al. (1989) [95]	F47	Meningioma	R frontal lobe, ethmoidal
Po and Hseuh (1990) [96]	F65	Meningioma	R sphenoid ridge
Sorenson et al. (1995) [97]	F58	Astrocytoma	R hypothalamus
Honma and Nagao (1996) [98]	F68	Adenoma	Pituitary, complicated by haemorrhage
Huang and Pai (2008) [99]	M67	Unknown	L medial temporal lobe
Agosti et al. (2008) $n = 2$ [100]	–	Meningioma	Falx
Dinca et al. (2011) [101]	F75	Meningioma	R transtentorial (cerebellum to temporal lobe)
Na et al. (2019) [102]	M65	Adenoma	Pituitary, extending to L medial temporal lobe
Turki et al. (2020) [103]	F55	Unknown	R frontal lobe

followed a generalised tonic–clonic seizure [88]. One patient was reported to have six episodes of TGA and on examination had bilateral papilloedema [93]; this case was criticised as unlikely to be TGA by Hodges ([4], p.30). Caplan [104] had previously criticised the case reported by Meador et al. on the grounds that the reported clinical features (two short-lasting and unobserved episodes of loss of awareness) [90] did not suggest TGA, and Daniel thought the cases of Hartley et al. [85], Shuping et al. [88] and Honma and Nagao [98], associated respectively with a pituitary tumour, left temporal glioblastoma and chronic haematoma in a parasellar tumour compressing the right medial temporal lobe, were more likely to be transient epileptic amnesia (TEA; see Sect. 3.2), the first and last based on repeated episodes of amnesia ([105], p.187,188). This may also be the case with the patient reported by Huang and Pai [99] (Sect. 3.2.2).

Not all published descriptions of TGA and tumour can be admitted as such. For example, Ross reported a female patient (Case 3) aged about 65 years as "experiencing transient global amnesia". She had papilloedema and a right superior homonymous quadrantanopia and was eventually found to have a left temporal glioblastoma. However, the attacks of "unusual behaviour" labelled as TGA occurred 2–3 times per week, lasted 12–15 hours, had been experienced for about a year and were characterised by knowing no one, including herself [106]. These clinical features fall outwith current understanding of TGA, in terms of both the frequency and duration of episodes, and the loss of knowledge of self, not to mention the absence of any report of repetitive questioning, and accordingly, this case is not included, although other authors seem to have accepted it as a case of tumour-related TGA ([107], p.184).

In many of the reviewed cases, the finding of a tumour was deemed unlikely to be anything more than chance concurrence with TGA, based on tumour locations distant from memory-eloquent structures, and hence an entirely incidental finding [40]. A similar argument may be made with respect to other structural lesions identified in TGA patients, such as hydrocephalus [108, 109] or cyst [110], subdural haematomas [111] and cerebral angioma [112]. Hence to label these cases as "symptomatic TGA" would, in this author's view, be an error. Brain haematoma may on rare occasion (e.g. [113]) be relevant to an episode of TGA (Table 3.5).

For tumour locations more obviously of possible pathophysiological relevance, such as those involving medial temporal lobe structures (e.g. a pituitary adenoma extending to left medial temporal lobe and anterior hippocampus [102]), neoplastic lesions might be anticipated to result in abnormal electrical activity within these networks. Milburn-McNulty and Larner argued that localised tumours might lower the threshold for epileptiform events, which might masquerade clinically as TGA [40] (see also Case Study 7.2). In other words, they considered that "tumour-associated TGA" was in most, if not all, instances transient epileptic amnesia and not TGA (of note, the index case which prompted their systematic review [40] subsequently underwent diagnostic revision after long-term follow-up showed neuroradiological remission of the swelling in the amygdala region which had initially been thought to be a low-grade glioma [114]). In this context, it is of note that patients harbouring medial temporal lobe tumours [99, 115, 116] and amygdala swelling [117] have been described as manifesting episodes typical of both TGA and subsequently TEA.

Case Study 7.2: Brain Tumour and TGA?

The 79-year-old man reported in Case Study 2.1, who suffered a brief (ca. 30 minutes) amnesic episode whilst hiking which was suspected to be TGA but with no reliable witness account, hence failing to fulfil diagnostic criteria, was further assessed. On the Mini-Mental State Examination, he scored 26/30 and on the Six-item Cognitive Impairment Test (negatively scored) 10/28, dropping points for delayed recall on both these screening instruments. MR brain imaging showed a left temporal lobe mass lesion with surrounding vasogenic oedema, appearances consistent with a high-grade glioma. A possible epileptic aetiology for his transient amnesic episode now seemed more likely than TGA.

An account of five cases of TGA occurring several years after temporal lobectomy for epilepsy (related to hippocampal sclerosis or dysembryoplastic neuroepithelial tumour, DNET) has appeared, the episodes conforming to TGA diagnostic criteria [118]. Though designated by the authors as a precipitating factor, the significant delay between surgery and TGA would be more in keeping with a predisposing factor. Moreover, although these patients had been seizure-free after surgery, the possibility that these episodes were epileptic in origin, despite conforming to TGA diagnostic criteria, cannot be entirely discounted.

In addition to these structural lesions evident on clinical and/or neuroradiological grounds, it is also possible that microstructural changes within brain tissue and reorganised network hubs (e.g. [119]), as detected using sophisticated neuroimaging modalities such as voxel-based morphometry and diffusion tensor imaging (Sect. 5.1.3), may also increase vulnerability to, and hence constitute predisposing factors for, TGA.

7.13 Summary and Recommendations

Many possible predisposing factors for TGA have been examined. Of these, the most consistent observation seems to be a personal history of migraine but no factor has been shown to be necessary and/or sufficient to induce TGA. A greater understanding of the neurobiology and hence the pathogenesis of TGA (see Chap. 9) might enlighten this field of research. Meantime, a number of more proximate, precipitating factors for TGA, have been described and these are examined in the next chapter.

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