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Objective: To evaluate the relation between lesions of various brain structures and the development of eating disorders and thus inform the neurobiological research on the aetiology of these mental illnesses.
Method: We systematically reviewed 54 previously published case reports of eating disorders with brain damage. Lesion location, presence of typical psychopathology, and evidence suggestive of causal association were recorded.
Results: Although simple changes in appetite and eating behaviour occur with hypothalamic and brain stem lesions, more complex syndromes, including characteristic psychopathology of eating disorders, are associated with right frontal and temporal lobe damage.
Conclusions: These findings challenge the traditional view that eating disorders are linked to hypothalamic disturbance and suggest a major role of frontotemporal circuits with right hemispheric predominance in the pathogenesis.

Eating disorders, including anorexia and bulimia nervosa, are characterised by abnormal eating behaviour and typical psychopathological features, including fear of fatness, drive for thinness, and body image disturbance. In most patients, there is no detectable focal brain abnormality. Nonetheless, associations of anorexia and bulimia nervosa with history of perinatal complications[1 2] and head injuries[3] suggest a role of cerebral pathology in some cases. A number of case studies describe eating disorders with intracranial tumours, injuries, or epileptogenic foci. However, many clinical descriptions are limited to changes in appetite and lack psychopathological features characteristic of eating disorders. A previous review of 21 anorexia cases associated with brain tumours found that only three of them fulfilled formal diagnostic criteria.[4] In the present paper, we provide a systematic review of published case reports and highlight those relatively rare cases where typical eating disorders appear to be causally associated with localised brain damage.

METHODS

The Web of Knowledge and Medline were searched for articles published up to April 2004 with any combination of keywords: ''eating disorders'', ''anorexia nervosa'', ''bulimia'', ''binge eating'', and ''compulsive eating'' with ''brain damage'', ''brain lesion'', ''tumor'', ''injury'', and ''epilepsy''. We perused reference lists and performed citation searches for the identified articles. Cases in children under 7 years were excluded. We found 54 case reports of eating disorders related to brain damage. Cases of obesity with brain lesions have been reviewed elsewhere[5] and are not included unless there is prominent behavioural disturbance or psychopathology.

As most papers did not use formal diagnostic criteria, we extracted the following symptoms from the case descriptions: underweight (less than 75% of body weight expected for the height and age), vomiting (not self induced), binge eating (eating unusually large amounts of food in a discrete time period), purging (self induced vomiting, laxative, or diuretic abuse for the purpose of weight control), preoccupations and rituals concerning food (calorie counting, specific eating rules, and unusual eating habits), preoccupations and rituals concerning body weight and shape (intention to lose weight, undue fear of fatness, body checking), hyperactivity (over exercising). Based on these symptoms, cases were classified into four categories: ''anorexia nervosa'' (underweight and either food or body related preoccupations or rituals, purging, or hyperactivity), ''atypical anorexia'' (underweight without food or body weight related preoccupations, rituals, or purging), ''bulimia nervosa'' (binge eating and/or purging and either food or body weight related preoccupations or rituals without underweight), and ''atypical bulimia'' (hyperphagia or binge eating but no food or body weight related preoccupations or rituals). In general, we refer as ''typical'' to cases for which there is a record of food or body related preoccupations or rituals or when purging behaviour, food restriction, or hyperactivity are intended to cause weight loss or prevent weight gain in the absence of objective overweight.

Evidence suggestive of causal association was noted if one of the following criteria were fulfilled: 1) the onset of eating disorder coincided with the occurrence of brain damage; 2) treatment directed at the lesion (surgical, antiepileptic) resulted in remission of the eating disorder; 3) brain injury coincided with remission of an eating disorder in a patient with previously established brain damage.

RESULTS

Hypothalamic lesions

There were 23 cases with brain lesions localised in the area of the hypothalamus and the third ventricle (Table 1: cases 1–23).[6–24] With one exception these were primary tumours. In addition to abnormal eating behaviour, symptoms commonly included diabetes insipidus, visual impairment, and unprovoked vomiting.

Three female cases were of apparently typical anorexia nervosa of the purging type (Table 1: cases 1–3). Case 1 appeared as typical in the initial phases with fear of fatness and self induced vomiting; later it progressed into an atypical scenario with spontaneous vomiting and good insight; remission occurred after surgical removal of the tumour. Case 2 seems fairly characteristic with onset in adolescence, concern over becoming fat and cheating about eating; however, there was excessive sleepiness and it is unclear whether vomiting was self induced or spontaneous. In case 3, anorexia developed on the background of severe personality disorder with borderline, compulsive, and perfectionist features; after tumour removal, eating urges subsided but the personality pathology persisted. It is notable that the onset of eating disturbance preceded neurological symptoms

Table 1 Case reports of eating disorders associated with brain lesions

Case number	Author	Year	Syndrome	Sex	Age	Onset	Causal	Lesion type	Lesion location	Method of lesion localisation	Weight	Vomiting	Psychopathology	Neurological symptoms
1	Wright[23]	1990	AN	F	12	10	Y	Germinoma	Hypothalamus, third ventricle	CT, surgery	Loss	Y	Body image, purging	Headaches, hypothyroidism
2	Hollatz[15]	1976	AN	F	19	14	N	Pinealoma	Hypothalamus, third ventricle	Post mortem	Loss	Y	Body image, food preoccupations, purging	Hypersomnia
3	Climo[9]	1982	AN	F	23		N	Craniopharyngioma	Hypothalamus, third ventricle	Surgery	Loss	N	Body image, binging, purging, obsessions	Diabetes insipidus
4	DeVile[12]	1995	A	M	8	8	Y	Craniopharyngioma	Hypothalamus, third ventricle	MRI, surgery	Loss	N		Headache, growth hormone deficiency
5	DeVile[12]	1995	A	M	13	13	Y	Pinealoma	Hypothalamus, third ventricle	MRI, biopsy	Loss	Y		Visual impairment, diabetes insipidus
6	White[21]	1977	A	M	15	15	Y	Glioma	Hypothalamus, third ventricle	Surgery	Loss	Y	Lack of concern	Diabetes insipidus
7	Nicholson[18]	1957	A	F	9	7	N	Pinealoma	Hypothalamus, third ventricle	Post mortem	Loss	N		Visual impairment, diabetes insipidus, hyperthermia
8	Chipkevitch[8]	1993	A	F	10	10	N	Teratoma	Hypothalamus, third ventricle	Post mortem	Loss	Y	Lack of concern, compulsive counting, hyperactivity	Somnolence, diabetes insipidus
9	White[22]	1959	A	F	62	59	N	Postencephalopathic atrophy	Hypothalamus	Post mortem	Loss	Y	Alcoholism	Headaches
10	Lewis[16]	1963	A	F	16	15	N	Pinealoma	Hypothalamus, third ventricle, midbrain	Post mortem	Loss	N		Diabetes insipidus, visual impairment
11	Lewin[24]	1972	A	F	25	25	N	Glioma	Hypothalamus	Post mortem	Loss	N		Hirsutism
12	Daly[10]	1973	A	F	22	21	N	Pinealoma	Hypothalamus	Post mortem	Loss	Y		Epilepsy, diabetes insipidus
13	Heron[14]	1976	A	M	25	24	N	Pinealoma	Hypothalamus, third ventricle	PEG, biopsy	Loss	N		Diabetes insipidus, almost blind
14	Biebl[7]	1984	A	M	15	13	N	Pinealoma	Hypothalamus, third abd lateral ventricles	CT	Loss	Y		Diabetes insipidus, visual impairment, incontinence
15	Lin[17]	2003	A	M	19	19	N	Germinoma	Hypothalamus, ventricles	MRI	Loss	Y	Lack of concern	Diabetes insipidus, hipopituitarism
16	Haugh[13]	1983	B	F	26	24	Y	Astroglioma	Hypothalamus	Post mortem	Gain	N	Aggressive behaviour, hallucinations	Sleep disturbance
17	Skorzewska[20]	1989	B	M	11	11	Y	Craniopharyngioma	Hypothalamus, third ventricle	Surgery	Gain	N	Binge eating, aggressive behaviour, emotional lability	Visual impairment, headaches, hipopituitarism, diabetes insipidus, hyperinsulinaemia
18	Skorzewska[20]	1989	B	F	12	12	Y	Craniopharyngioma	Hypothalamus, third ventricle	Surgery	Gain	N	Eating rituals, binge eating, aggressive behaviour, emotional lability	Visual impairment, headaches, hipopituitarism, hyperinsulinaemia
19	Skorzewska[20]	1989	B	F	8	8	Y	Craniopharyngioma	Hypothalamus, third ventricle	Surgery	Gain, Loss	Y	Binge eating, aggressive behaviour, stealing	Headaches, diabetes insipidus, hyperinsulinaemia
20	DePedro[11]	2001	B	F	18	15	Y	Craniopharyngioma	Hypothalamus, third ventricle	Surgery	Gain	N	Binge eating, emotional lability	Diabetes insipidus, visual impairment, hipopituitarism

Table 1 Continued

Case number	Author	Year	Syndrome	Sex	Age	Onset	Causal	Lesion type	Lesion location	Method of lesion localisation	Weight	Vomiting	Psychopathology	Neurological symptoms
21	DePedro[11]	2001	B	F	47	43	Y	Craniopharyngioma	Hypothalamus, third ventricle, posterior fossa	Surgery	Gain	N	Binge eating	
22	Reeves[19]	1969	B	F	20	19	N	Teratoma	Ventromedial hypothalamus	Post mortem	Gain	N	Hallucinations	Diabetes insipidus, dementia, confusion
23	Beal[6]	1981	B	M	57	55	N	Gangliocytoma	Hypothalamus, ventricles	Post mortem	Gain	N		Dementia, hypersomnia
24	Ahsanuddin[25]	1983	AN	F	22	15	Y	Choroid plexus papilloma	Brain stem, fourth ventricle	CT, surgery	Loss	N	Body image, lack of concern	Impaired motor coordination, tremor
25	Lehrnbecher[27]	2001	AN	F	13	12	Y	Glioma	Brain stem, medula, posterior fossa, foramen magnum	MRI	Loss	N	Body image	Hiccup, difficulty swallowing
26	Maroon[29]	1977	A	M	14	9	Y	Astrocytoma	Brain stem, fourth ventricle	Surgery	Loss	Y		Tightness in the neck, dysdiadochokinesis
27	Liebner[28]	1957	A	F	28	22	N	Hemangioblastoma	Brain stem, fourth ventricle, cerebellum	Post mortem	Loss	Y		
28	DeVile[12]	1995	A	M	7	5	N	Astrocytoma	Brain stem	MRI	Loss	Y		Nystagmus, coughing, discomfort swallowing
29	Rohmer[30]	1975	A	F	15	14	N	Medulloblastoma	Brain stem, fourth ventricle	Surgery	Loss	Y		Headaches, visual impairment
30	Grossmann[26]	2002	A	F	11	11	N	Cavernoma	Medulla oblongata	MRI, surgery	Loss	Y		
31	Ward[38]	2000	AN	M	23	16	Y	Injury	Bilateral inferior frontal lobe	MRI	Loss	Y	Body image, food preoccupations, binge eating, purging, exercise, anxiety, lack of concern, substance abuse	Epilepsy
32	Levine[31]	2003	AN	F	36		Y	Injury	Right frontal and temporal lobe	MRI	Loss	N	Body image, depression	Epilepsy
33	Shedlack[34]	1992	AN	M	23	21	Y	Injury	Right posterior temporal lobe	EEG, SPECT	Loss	N	Body image, eating rituals, mania, obsessions, depression, hyper-religiousness	Epilepsy
34	Signer[35]	1990	AN	F	36		N	Injury	Right frontal and temporal lobe	CT, EEG	Loss	N	Body image, purging, aggression, hyper-religiousness, cognitive impairment	Epilepsy
35	Signer[35]	1990	AN	F	25	17	N	Epileptogenic focus	Left frontal and temporal lobe	EEG	Loss	N	Body image, purging, exercise, hallucinations, ideas of reference, depression	Epilepsy
36	Trabert[36]	1990	AN	F	32	16	N	Angioma	Left inferior temporal	CT, surgery	Loss	N	Body image, purging, alcohol abuse	Epilepsy
37	Trummer[37]	2002	AN	F	23	19	N	Venous malformation	Right frontal lobe	CT, surgery, histology	Loss	N	Food preoccupations, anxiety, compulsive studying	Headache, loss of consciousness
38	Trummer[37]	2002	A	M	24	21	Y	Oligoastrocytoma	Right frontal lobe	MRI	Loss	N	Hyperactivity, obsessions, compulsions	Epilepsy
39	Trummer[37]	2002	A	M	36	35	Y	AV malformation	Right frontal lobe	MRI	Loss	Y	Anxiety, obsessions	Epilepsy

Table 1 Continued

Case number	Author	Year	Syndrome	Sex	Age	Onset	Causal	Lesion type	Lesion location	Method of lesion localisation	Weight	Vomiting	Psychopathology	Neurological symptoms
40	Ward[38]	2000	A	F	42	25	N	Abscess	Right frontal lobe	Surgery	Loss	N	Personality change: dissocial, aggressive	Epilepsy
41	Levine[31]	2003	BN	F	29	20	Y	Epileptogenic focus	Right temporal and occipital lobe	MRI, surgery		N	Body image, food preoccupations, eating rituals, purging, depression	Epilepsy
42	Ott[33]	1991	BN	F	33	28	Y	MRI detected lesion	Left medial temporal lobe	MRI		N	Binge eating, purging, substance abuse	Epilepsy
43	Angelini[32]	1980	B	M	10	10	Y	Astrocytoma	Right anterior cingulate	CT, surgery		N	Binge eating, aggressive behaviour, disinhibition	Epilepsy
44	Hebebrand[39]	1993	A	M	19	16	N	MRI detected lesion	Right putamen	MRI	Loss	N	Eating rituals and preoccupation, compulsive exercising	
45	Ward[38]	2000	B	F	55	50	N	Adenoma	Pituitary	Surgery, histology		N	Binge eating	
46	Wolanczyk[40]	1977	AN	M	14	12	N	Arachnoidal cyst	Frontal and parietal lobes	MRI	Loss	N	Eating rituals, body image, exercising	
47	Weller[43]	1982	AN	F	13	8	N	Hemangiopericytoma meta	Frontal lobes, hypothalamic, posterior fossa	CT	Loss	N	Eating rituals, body image, exercising	
48	Ward[38]	2000	AN	F	24	23	N	Glioma	Ventricles, basal ganglia, frontal lobes	Post mortem	Loss	N	Food preoccupations and body image	
49	McClean[42]	1988	A	M	14	13	Y	Ependymoma	Ventricles, basal ganglia, hypothalamus, frontal lobe	CT	Loss	Y		Epilepsy, diabetes insipidus
50	Goldney[41]	1978	A	F	28	24	N	Craniopharyngioma	Ventricles, midbrain, diencephalon	Post mortem	Loss	N		Visual impairment
51	Signer[35]	1990	BN	F	28	11	Y	EEG abnormality	Diffuse	EEG		N	Binge eating, body image	
52	Pauls[45]	1991	A	M	28	26	N	Hydrocephalus	–	CT	Loss	Y	Binge eating	Sleep disturbance
53	Damluji[44]	1987	A	M	15	11	Y	Hydrocephalus	–	CT	Loss	Y		Growth arrest, nausea, transitory loss of vision, fatigue, hipopituitarism
54	Krahn[46]	1984	BN	F	29	25	Y	Hydrocephalus	–	CT	Gain	Y	Binge eating, purging, food preoccupation, body image	Headaches and scotomata

Causal – evidence suggestive of causal association. AN, anorexia nervosa; A, atypical anorexia; BN, bulimia nervosa; B, atypical bulimia; CT, computed tomography; EEG, electroencephalogram; F, female; M, male; MRI, magnetic resonance imaging; N, no; PEG, pneumoencephalography; SPECT, single photon emission computed tomography; Y, yes.

by a longer interval (2 and 5 years in the two cases where age of onset is reported) than in the atypical anorexia cases.

Twelve cases (4–15) were classified as atypical anorexia with unintentional weight loss and, in seven, unprovoked vomiting. Although typical psychopathology was not reported, lack of concern over emaciation was noted in three, depressed mood in five, and obsessive compulsive symptomatology in one case (8). In three (4, 5, and 6) there was suggestive evidence of causal association in that anorexia remitted after surgical or radiation treatment. None of these cases would fulfil formal diagnostic criteria for anorexia nervosa; their presentation was atypical in terms of sex distribution, age of onset, and associated neurological symptoms.

The eight cases of atypical bulimia (16–23) presented with voracious appetite and emotional lability. Aggressive and antisocial behaviours were manifested mainly when access to food was denied.

In conclusion, lesions in the hypothalamic area can lead to eating disturbance with either loss or increase of appetite. There is little evidence of hypothalamic tumours causing typical eating disorders. Of the three reported cases with eating disorder specific psychopathology, one can be attributed to premorbid personality disorder and the other two developed relatively long time before the neurological symptoms.

Brain stem lesions

Of the seven anorexia cases associated with primary tumours in the area of brain stem and the fourth ventricle (Table 1: cases 24–30),[12 25–30] two (24 and 25) presented as typical restrictive anorexia nervosa with fear of fatness; surgical removal of the tumours led to remission and sustained weight gain in both cases.

The other five cases were clearly atypical with weight loss in the absence of weight concerns or body image disturbance. In case 26, atypical anorexia fully remitted after radiation treatment. Other symptoms included unprovoked vomiting, difficulty swallowing, hiccup, coughing, and nystagmus.

In summary, brain stem lesions are associated with loss of appetite and effortless vomiting. The suggestive association with typical cases of restrictive anorexia nervosa relies on two case reports and needs to be substantiated by further evidence.

Hemispheric lesions

Thirteen cases of eating disorders associated with lesions in the cerebral hemispheres were identified (Table 1: 31–43).[31–38] The damage was predominantly localised in the frontal and temporal lobes (six frontal, four temporal, three frontotemporal) of the right hemisphere (nine right, three left, one bilateral). In eight cases, there was evidence suggestive of causal association between the lesion and eating disorder. Epilepsy was present in all but one. Additional symptoms included depression, mania, psychotic features, substance abuse, obsessions, compulsions, and hyper-religiousness.

Seven cases presented as ''typical'' anorexia nervosa with weight and shape preoccupations. In three of them there was a suggestive causal association between brain damage and anorexia nervosa. In case 31, the symptoms started after frontal lobe injury in a previously healthy male adolescent. In case 32, a frontotemporal injury led to remission of restrictive anorexia in an adult woman with a history of epilepsy. In case 33, restrictive anorexia nervosa remitted with a successful treatment of epilepsy by anticonvulsive medication in a young man.

There are three cases of atypical anorexia with right frontal epileptic focus. In cases 38 and 39, obsessive compulsive symptomatology was prominent and both anorexia and epilepsy remitted after embolisation of a vascular malformation or surgical tumour removal.

Two cases of typical bulimia nervosa with binging and purging symptomatology resolved with successful management of epilepsy by temporal lobotomy (41) or antiepileptic medication (42). In case 43, atypical bulimia with severe behavioural disturbance resolved with surgical removal of an astrocytoma in the right anterior cingulate cortex.

In summary, there is compelling evidence of hemispheric damage being causally associated with typical eating disorders. This is supported by a large proportion of cases with typical psychopathology, remission of eating disorders after brain lesion removal, and consistent localisation of lesions in the right frontal and temporal lobes.

Other lesions

In case 44, lesion in the right putamen was associated with obsessive compulsive disorder with food-related preoccupations and compulsive exercising.[39] Case 45 is of atypical bulimia with growth hormone producing adenoma of the pituitary.[38] In case 46, a boy developed typical anorexia nervosa at age 12 followed by a psychotic episode 2 years later; magnetic resonance imaging revealed parietal arachnoidal cyst and frontal lobe atrophy.[40]

Cases 47–50 had disseminated tumours affecting more than one brain structure.[38 41–43] Two of these were classified as atypical anorexia and two as typical anorexia nervosa. In case 49, surgery combined with radiotherapy for a disseminated ependimoma lead to remission of atypical anorexia.

Case 51 is of typical bulimia nervosa with diffuse paroxysmal abnormality on electroencephalogram, which improved with a combination of carbamazepine and lithium.[35] In cases 52–54, disordered eating was associated with hydrocephalus.[44–46] In two, anorexia (53) or bulimia (54) remitted upon placement of a ventriculo-peritoneal shunt.

These cases with diffuse or disseminated brain damage further support the association between eating disorders and brain pathology; however, they are less informative as to the location of dysfunctional circuits underlying eating disorders.

DISCUSSION

This review of published case reports challenges the traditional view that hypothalamic disturbance underlies eating disorders. Although hypothalamic lesions are the most commonly reported neural causes of anorexia-like syndrome, most of them lack the typical psychopathology. Of the eight cases with characteristic psychopathological presentation and suggestive evidence for a causal association, four had frontal and temporal cortical lesions, two brain stem tumours, one hypothalamic tumour, and one hydrocephalus. Implication of frontotemporal circuits is consistent with functional neuroimaging research in eating disorders[47 48] and with benign changes in eating, such as the gourmand syndrome.[49] Therefore, we conclude that evidence favours cortical mechanisms in the genesis of eating disorders over hypothalamic ones.

An association of disordered eating with epilepsy was reported in 12 cases. In six of these, remission after a surgical removal of an epileptogenic focus or anticonvulsant treatment suggests that eating disorder may be actively maintained by an epileptogenic focus rather than being a deficit syndrome due to missing normal brain tissue.[31 33–35 37]

In five of the reviewed cases, disturbed eating occurred alongside obsessive compulsive psychopathology.[8 9 34 37 39] This finding parallels the comorbidity and familial co-occurrence of eating disorders and obsessive compulsive disorder[50 51] and suggests a common or overlapping neural substrate of the two.

Limitations

This review relies on case reports, which represent a highly selected material prone to reporting and publication bias. Notably, sixteen of the 23 hypothalamic lesions associated with disordered eating were published before 1990 compared with only one cortical lesion. This publication trend reflects a shift of emphasis in the eating disorders research from the study of endocrine and autonomic correlates to psychologically informed explanations.

Although resolution of a disorder after removal of a cerebral lesion is highly suggestive of a causal relation, this is not a proof of causality. For example, in a case of anorexia nervosa remitting after surgical removal of a spinal meningioma, it is uncertain whether the mechanism of apparent causality relates to the removal of a tumor mass, decompression of the cerebrospinal fluid spaces, or other unspecific factors.[52]

The information provided in the case reports is of variable extent and quality. This may have affected the classification of individual cases as typical or atypical. If information on psychopathology was missing, the case was by default regarded as atypical.

Clinical implications

Neurological symptoms of hypothalamic (changes in appetite, excessive thirst and drinking) or brain stem (effortless vomiting, difficulty swallowing) lesions resemble symptoms of eating disorders but may be distinguished on clinical grounds because specific psychopathology is usually not present. Onset of disturbed eating in an unusual age or gender, history of head injury, or epilepsy should prompt neurological examination, including a magnetic resonance imaging of the brain. Finally, in a patient with epilepsy or suspected brain damage, the development of disordered eating behaviour is a localising symptom suggestive of a right anterior focus.

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