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ABSTRACT
Acquired brain pathology can be associated with diverse psychiatric manifestations. Three major types of psychiatric disorders potentially found in cases of acquired brain pathology are examined: (1) psychosis, (2) mood disorders, and (3) personality disorders with special emphasis in so-called “acquired psychopathy.” Two types of psychotic manifestations are reviewed: (a) Schizophrenia-like psychosis; (b) Other delusional disorder, specifically, somato-paraphrenia and delusional misidentification syndromes, which include reduplicative paramnesias, Capgras syndrome, Frégoli syndrome, and “doubles of the self-syndrome.” Schizophrenia-like psychosis has been reported as sequelae of traumatic brain injury with a prevalence of around 1 to 9%. On the other hand, the other delusional disorders are usually associated with right hemisphere or bilateral lesions. The significance of mood disorders particularly in cases of frontal lobe pathology has been reported, including: depression, bipolar disorder, alcohol abuse, panic disorder, and increased risk of suicide are frequently observed. Personality disorders are frequent in cases of brain pathology, particularly frontal lesions. It is concluded that the analysis of the psychiatric changes associated with acquired brain pathology has not only a clinical importance but also a fundamental interest, advancing the understanding of the neurological bases of major psychiatric conditions.

KEYWORDS
Acquired psychopathy; delusional disorders; mood disorders; neuropsychiatry; psychosis

Since several decades ago, the analysis of the brain abnormalities associated with psychiatric disorders has represented a major neuroscience research area, frequently known as neuropsychiatry (Cummings, 1985; Harris, 2017; Tekin & Cummings, 2002). “Psychiatric disorders,” however, is a broad and even confusing term; psychiatry as a matter of fact is a quite extensive clinical area, usually defined as “the branch of medicine focused on the diagnosis, treatment and prevention of mental, emotional and behavioral disorders” (American Psychiatric Association; www.psychiatry.org/patients-families/what-is-psychiatry). Regardless of such an extensive definition, psychiatry usually concentrates in the analysis of psychosis, mood disorders, and personality disorders; those are indeed the major psychiatric disorders. Acquired brain pathology—strokes, traumatic brain injury, tumors, among others—can also be associated with diverse psychiatric manifestations, including but not limited to psychosis, mood disorders, and personality disorders.

Consequences of acquired brain damage in children are partially different (Fornito, Zalesky, & Breakspear, 2015). Early damage to the brain may interfere with subsequent maturational processes and, hence, not in the immediate loss of an ability, but in the failure to develop that ability. For example, a frontal damage in children may have minimal behavioral and cognitive manifestations in a short term, but results in an inappropriate development of executive functions, observable only during adolescence.

Three studies can illustrate the diversity of psychiatric manifestations potentially associated with acquired brain damage.

Deb, Lyons, Koutzoukis, Ali, and McCarthy (1999) evaluated the type and extent of psychiatric syndromes in hospitalized patients one year after a traumatic brain injury. Psychiatric diagnoses were made according to ICD-10 criteria. Out of 164 patients interviewed, 30 (18.3%) had an ICD-10 diagnosis of a psychiatric illness. Among the 120 patients who were in the age range 18–64 years, 21.7% had a psychiatric disorder, compared with 16.4% in a study of the general population. A depressive illness was found in 13.9% of the patients, compared with 2.1% of the general population, and panic disorder was present in 9.0%, compared with 0.8% of the general population.
population. The authors concluded that in comparison with the general population, a higher proportion of patients developed psychiatric illnesses one year after a traumatic brain injury.

Orlovska et al. (2014) used the Danish nationwide population-based registers to investigate the incidence of schizophrenia spectrum disorders, unipolar depression, bipolar disorder, and organic mental disorders in 113,906 persons who had suffered head injuries. It was found that a head injury, especially a severe head injury or one occurring between the ages of 11 and 15, increased the risk for subsequent schizophrenia by 65%, the risk of depression by 39%, the risk of bipolar disorder by 28%, and the risk of organic mental disorders by more than 400%. The added risk of mental illness following head injury did not differ between individuals with and without a psychiatric family history.

In a more recent study Perry et al. (2016) developed a meta-analysis with the objective to determine the association of prior mild traumatic brain injury with the subsequent diagnosis of neurological or psychiatric disorder. Fifty-seven studies were included. A significant association of prior traumatic brain injury with subsequent neurological and psychiatric diagnoses was found. Prior brain injury was independently associated with both neurological and psychiatric outcomes. Analyses of individual diagnoses revealed higher odds of Alzheimer’s disease, Parkinson’s disease, mild cognitive impairment, depression, mixed affective disorders, and bipolar disorder in individuals with previous traumatic brain injury as compared to those without it. It was concluded that mild traumatic brain injury is associated with the development of neurological and psychiatric illness.

These three solid studies clearly illustrate that there is a frequent association between acquired brain pathology and major psychiatric disorders.

In this article, the three major types of psychiatric disorders potentially found in cases of acquired brain pathology are examined: (1) psychosis, (2) mood disorders, and (3) personality disorders with special emphasis in so-called “acquired psychopathy.”

**Psychosis**

**Schizophrenia-like psychosis**

Schizophrenia-like psychosis has been reported as sequelae of traumatic brain injury (Fujii & Ahmed, 2014). Its prevalence is around 1 to 9% (Chen, Chiu, Chu, & Lin, 2011; Fann et al., 2002; Harrison et al., 2006; Koponen et al., 2002; Nielsen, Mortensen, O’Callaghan, Mors, & Ewald, 2002). Schizophrenia-like psychoses usually appears about one to four years after the injury and is associated with focal lesions in the frontal and temporal lobes, slowing in the electroencephalogram (EEG), and general cognitive impairments, including memory impairments and disturbances in executive functions (Fujii & Ahmed, 2014). The majority of these patients are males after moderate to severe head injury. The general presentation included delusions and hallucinations without co-occurring negative symptoms (Fujii & Ahmed, 2002). The clinical presentation in general has considerable overlap with primary schizophrenic disorder, with a prominence of persecutory and other delusions and auditory hallucinations. The onset is often gradual, with a subacute or chronic course (Zhang & Sachdev, 2003).

To approach the association between traumatic head injury and schizophrenia, Molloy, Conroy, Cotter, and Cannon (2011) developed a meta-analysis including nine studies. The pooled analysis revealed a significant association between traumatic brain injury and schizophrenia (OR = 1.65; 95% CI = 1.17–2.32), with significant heterogeneity between the studies. This meta-analysis supported an increased risk of schizophrenia following traumatic brain injury, with a larger effect in those with a genetic predisposition to psychosis. In a literature review, Kim (2008) concluded that evidence supports a risk-modifying effect of traumatic brain injury in individuals who are genetically at risk for schizophrenia, but is less supportive of traumatic brain injury as an independent risk factor for schizophrenia in individuals without such risk.

Some authors have emphasized the involvement of the right hemisphere in delusional phenomena and, in particular, in schizophrenic-like post-traumatic psychosis (Kumral, & Öztürk, 2004; Levine & Grek, 1984; Rabins, Starkstein, & Robinson, 1991). Devine et al. (2014) analyzed three cases of poststroke psychosis. These cases were compared with a control group of patients with similar anatomical damage. The right inferior frontal gyrus and underlying white matter, including the superior longitudinal fasciculus and anterior corona radiata, were involved in all three cases. All three had a preexisting untreated psychiatric disorder. The authors suggest that pre-existing psychiatric disease provided a behavioral susceptibility to develop delusions in these individuals.

**Other delusional disorders**

**Somatoparaphrenia**

Somatoparaphrenia is a delusional belief in which a patient states that the limb, contralateral to a brain
pathology, usually the left upper one, does not belong to him/her (Invernizzi et al., 2013); somatoparaphrenia is typically associated with anosognosia, somatosensory disturbances, and unilateral spatial neglect. This syndrome is not a fully comprehended disorder, categorizing as somewhere between neurology and psychiatry.

In 2009, Vallar and Ronchi carried out an extensive literature review of somatoparaphrenia. They found 56 cases with unilateral, or mainly unilateral, hemispheric lesions and somatoparaphrenic symptoms. Fifty-one out of the patients (91%) presented somatoparaphrenia in the left side of the body, associated with a right-sided lesion. Some patients reported a sense of strangeness toward contralateral to the lesion body parts and they may wonder whether the affected limb belong to another person; in other patients, body parts feel separate from the patient’s body. However, the more frequent manifestations of somatoparaphrenia, observed in over two-thirds of the patients is a sense of disownment: the delusional belief that body parts contralateral to the brain pathology do not belong to them, but they belong to another person.

In 2012, Gandola et al. published an analysis of the anatomical correlates of somatoparaphrenia. Some proposed specific lesion correlations with the right posterior insula (Baier & Karnath, 2008), the supramarginal gyrus and the posterior corona radiate (Feinberg et al., 1990), or the right medial or orbito-frontal regions (Feinberg et al., 2010).

Somatoparaphrenia can be regarded as a syndrome in between neurology and psychiatry (Feinberg & Venneri, 2014). Different authors have even proposed that dysfunction of the right hemisphere can be associated with delusional ideas, including somatoparaphrenia, suggesting that some right hemisphere brain abnormalities should exist in psychosis (Feinberg & Roane, 2005). In this regard, the analysis of somatoparaphrenia may contribute to further our understanding regarding the neurological bases of psychosis.

**Reduplicative paramnesia**

Reduplicative paramnesia is characterized by the belief that a familiar place, person, object, or body part has been duplicated (Hakim, Verma, & Greiffenstein, 1988). Most reported cases refer to duplication of places; that is, the hospital, the city, the patient’s house, or other geographical points are duplicated.

Murai, Toichi, Sengoku, Miyoshi, and Morimune (1997) selected 77 patients with focal brain damage (47 with left hemispheric, 21 with right hemispheric, and 9 with bilateral damage) and were assessed for the presence of reduplicative paramnesia using a questionnaire. Two patients showed typical reduplicative paramnesia for place, and four patients showed atypical reduplicative paramnesia (three for place and one for person). In three patients, the lesions were situated in the right hemisphere; in two, the lesions were bilateral (right dominant); and in one, the lesions were in the left hemisphere. Other reports clearly support the finding that reduplicative paramnesia is very specially associated with right hemisphere or bilateral lesions.

It is important to bear in mind that there is a whole group of disorders known as “delusional misidentification syndromes,” where patients think that a familiar person is someone else or a certain familiar place is a duplicate (Atta, Forlenza, Gujski, Hashmi, & Isaac, 2006). Delusional misidentification syndromes are found in various psychotic and organic brain diseases. In addition to the reduplicative paramnesia, they include the Capgras syndrome consisting of the belief that one or more people (usually close relatives) have been substituted by imposters or doubles; the Frégoli syndrome (or “the delusion of doubles”) characterized the delusional belief that one or more familiar persons, usually persecutors following the patient, repeatedly change their appearance; that is, different people are in fact a single person who changes appearance. Moreover, “intermetamorphosis” is characterized by the belief that the patient can perceive that an individual has been transformed both psychologically and physically into another person. Therefore, there exists the “doubles of the self” or “subjective doubles” syndrome consisting of the belief that the patient has a double with the same appearance, but usually with different character traits, who is leading a life of its own. Important to note, Capgras syndrome can be interpreted as a reduplicative paramnesia.

**Mood disorders**

Mood disorders are frequently observed after acquired brain pathology (Jorge & Arciniegas, 2014). This observation has been reported at least since the Phineas Gage classical case (Harlow, 1868). In a well-known study, Robinson, Kubos, Starr, Rao, and Price (1984) selected a group of right-handed patients with single stroke lesions of either the right ($n = 14$) or left ($n = 22$) hemisphere. The authors observed that the severity of depression was significantly increased in patients with left anterior lesions as opposed to any other lesion location. In addition, the severity of depression correlated significantly with proximity of the lesion on CT scan to the frontal pole in the left hemisphere.
anterior group. The right hemisphere lesion group showed the reverse trend: patients with right posterior lesions were more depressed than patients with right anterior lesions, who were unduly cheerful and apathetic. The authors proposed that intrahemispheric lesion location is in some way related to mood disorder in stroke patients; furthermore, there is a graded effect of lesion location on severity of mood change. Further studies, however, have challenged these conclusions. Using a stepwise linear regression analysis including 81 poststroke patients, it was found that inferior frontal lesion location, irrespective of side, appeared to play a role as a risk factor for depression (Singh et al., 2000). Other studies have suggested that right frontal pathology is frequently associated with depression (e.g., Almeida, Burton, Ferrier, McKeith, & O’Brien, 2003).

Fazel, Wolf, Pillas, Lichtenstein, and Långström (2014) developed a large study. They studied all persons born in 1954 or later in Sweden who received the diagnoses of traumatic brain injury from 1969 to 2009 (n = 218,300). The authors compared mortality rates six months or more after traumatic brain injury to general population controls matched on age and sex. Among those who survived six months after traumatic brain injury, they found a three-fold increased odds of mortality compared with general population controls. Risks of mortality from external causes were elevated, including for suicide, injuries, and assault. The authors concluded that traumatic brain injury is associated with substantially elevated risks of premature mortality, particularly for suicide.

Ciurlu, Formisano, Bivona, Cantagallo, and Angelelli (2011) found in a sample of 120 persons with severe TBI a wide range of neuropsychiatric symptoms, including: apathy (42%), irritability (37%), dysphoria/depressed mood (29%), disinhibition (28%), and agitation (24%). Bombardier et al. (2010) studied 559 consecutively hospitalized adults with complicated mild to severe traumatic brain injury. They were followed up by structured telephone interviews at months 1 through 6, 8, 10, and 12. Two hundred ninety-seven patients (53.1%) met criteria for major depressive disorder at least once in the follow-up period. In a multivariate model, risk of major depressive disorder after traumatic brain injury was associated with major depressive disorder at the time of injury, history of major depressive disorder prior to injury (but not at the time of injury), age, and lifetime alcohol dependence. Those with major depressive disorder were more likely to report comorbid anxiety disorders than those without it.

Frequency of depression probably ranges from 17 to 61%, and risk factors include past psychiatric history, frontal lesions and atrophy, and family dysfunction (Rapoport, 2012). In a recent review of 34 studies described in 68 publications, Scholten et al. (2016) found that the prevalence rate of psychiatric disorders varied widely. Pooled prevalence estimates of anxiety and depressive disorders were 19% and 13% before traumatic brain injury and 21% and 17% in the first year after TBI. Pooled prevalence estimates increased over time and indicated high long-term prevalence of Axis I disorders (54%), including anxiety disorders (36%) or depressive disorders (43%). Anxiety disorders have also been reported in children and adolescents with traumatic head injury (Max et al., 2011).

The significance of mood changes, particularly in cases of frontal lobe pathology, has been reported by diverse authors, as described in the three major studies described at the beginning of this paper (Koponen et al., 2002; Orlovska et al., 2014; Perry et al., 2016).

**Personality disorders**

Personality disorders are not infrequent in cases of brain pathology, particularly frontal anterior lesions (Petrie, 2013). This condition is often found in cases of traumatic brain injury. For instance, there is a high frequency of emotional and behavioral dyscontrol after traumatic brain injury (Arciniegas & Wortzel, 2014). Conversely, there is a high prevalence of brain pathology in violent individuals and people with a borderline personality disorders (e.g., Witzel, Bogerts, & Schiltz, 2016).

Koponen et al. (2002) evaluated 60 patients on average 30 years after suffering a traumatic brain injury. Fourteen patients (23.3%) had at least one personality disorder. The most prevalent individual disorders were avoidant, paranoid, and schizoid personality disorders. It was concluded that traumatic brain injury may cause decades-lasting vulnerability to psychiatric conditions in some patients. Traumatic brain injury seems to make patients particularly susceptible to different disturbances, including personality disorders. Personality changes have been also reported in children and adolescents. Max et al. (2011) observed personality changes in 26 (18%) of 141 five-14 years old participants assessed at six months postinjury.

Barrash, Tranel, and Anderson (2000) reported the personality changes in seven participants with bilateral ventromedial prefrontal lesions (PF-BVM), 14 participants with prefrontal lesions but not bilateral ventromedial involvement (PF-NBVM), and 36 with
nonprefrontal lesions (NPF). Bilateral ventromedial prefrontal lesions participants showed a higher rate of acquired disturbances than nonprefrontal lesions participants in blunted emotional experience, apathy, low emotional expressiveness, inappropriate affect, poor frustration tolerance, irritability, lability, indecisiveness, poor judgment, social inappropriateness, lack of planning, lack of initiation and persistence, and lack of insight. All 7 bilateral ventromedial prefrontal lesions participants developed a syndrome, including general dampening of emotional experience; poorly modulated emotional reactions; defective decision making, especially in the social realm; impaired goal-directed behavior; and striking lack of insight. The authors suggest that similarities between this syndrome of “acquired sociopathy” and developmental psychopathy in characteristic personality disturbances and psychophysiological abnormalities indicates that diminished emotionality, impaired decision making, and psychosocial dysfunction may be related to ventromedial prefrontal dysfunction in both groups.

Psychopathy has been associated with some brain abnormalities, including a reduction in prefrontal gray matter volume, gray matter loss in the right superior temporal gyrus, amygdala volume loss, a decrease in posterior hippocampal volume, an exaggerated structural hippocampal asymmetry, and an increase in callosal white matter volume in psychopathic individuals. In general, Key regions commonly found to be impaired in antisocial populations include the prefrontal cortex (particularly orbitofrontal and dorsolateral prefrontal cortex), superior temporal gyrus, amygdala–hippocampal complex, and anterior cingulate cortex (de Oliveira-Souza et al., 2008; Weber, Habel, Amunts, & Schneider, 2008; Yang, Glenn, & Raine, 2008).

Diverse studies have suggested that prefrontal damage is associated with disturbances in moral and social behavior, a type of acquired psychopathy. Traumatic head injury can also increase sexual offending behavior. Anderson, Damasio, Tranel, and Damasio (2000) analyzed the long-term behavioral and cognitive sequelae of damage to prefrontal cortex in two young adult patients who had sustained their brain damage prior to 16 months of age. They had normal neurological examinations, but remarkable histories of impaired decision making, behavioral dyscontrol, social defects, and abnormal emotion. Performances were close to normal on diverse neuropsychological domains (memory, language, visual perception, and visuoconstruction), but selective impairments in executive functions. Other studies have supported these conclusions; acquired psychopathy is associated with frontal damage and disorders in executive functions (e.g., Blair & Cipolotti, 2000; Broomhall, 2005; Mitchell, Avny, & Blair, 2006).

Conclusions

The analysis of the psychiatric changes associated with acquired brain pathology has a double interest: clinical and fundamental. From the clinical point of view, they represent frequent clinical manifestations of neurological pathologies; a major psychiatric disorder can be the consequence of some genetic and/or maturational conditions, but it can also be the result of acquired brain pathology. From the fundamental point of view, they further the understanding of the neurological bases of major psychiatric conditions, such as psychosis, mood disturbances, and personality disorders. However, larger multicenter studies using reliable and standardized diagnostic instruments are required to further clarify the nosology, risk factors, and clinical manifestation of these disorders (Ahmed et al., 2017; Kim et al., 2007).

References


