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Social cognition impairments after aneurysmal subarachnoid haemorrhage: Associations with deficits in interpersonal behaviour, apathy, and impaired self-awareness

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ABSTRACT

Behavioural disturbances are frequently found after aneurysmal subarachnoid haemorrhage (aSAH). Social cognition impairments have been suggested as a possible underlying mechanism for behavioural problems. Also, aSAH is likely to result in damage affecting frontal-subcortical circuits underlying social cognition. Therefore, we aimed to investigate social cognition after aSAH and its associations with behavioural problems (deficits in interpersonal behaviour, apathy, and impaired self-awareness) and focal as well as diffuse brain damage. 88 aSAH patients (in the subacute phase post-aSAH) and 60 age-, sex- and education-matched healthy controls participated. Tasks for emotion recognition, Theory of Mind (ToM), and empathy as well as questionnaires were used. Cortical infarctions in frontal and non-frontal areas on MRI, aneurysm circulation and aSAH-related events were taken into account. Compared to healthy controls, aSAH patients performed significantly worse on tasks for emotion recognition, ToM and empathy. Poor performance on ToM and emotion recognition was associated with proxy-ratings indicating impaired interpersonal behaviour and apathy and with indications of impaired self-awareness. No associations were found between deficits in social cognition and frontal or non-frontal cortical lesions on MRI. Also, aneurysm circulation and aSAH-related events such as hydrocephalus, vasospasm, and treatment method did not explain why and how social cognitive deficits did occur after aSAH. In conclusion, emotion recognition, ToM and empathy were clearly impaired in aSAH patients and these deficits were related to apathy and deficits in interpersonal behaviour as reported by proxies and to impaired self-awareness. This association strengthens the assumption of impaired social cognition as an underlying construct of behavioural problems after aSAH. Consequently, social cognition tests and proxy-ratings should be used in clinical practice, irrespective of lesion location on MRI or aneurysm circulation, to improve the detection and treatment of apathy and deficits in interpersonal behaviour after aSAH.

1. Introduction

Aneurysmal subarachnoid haemorrhage (aSAH) is an acute bleeding in the subarachnoid space, the area between the pia mater and the arachnoid membrane, and is caused by the rupture of a cerebrovascular aneurysm. It is a serious neurological condition associated with high mortality and morbidity (Nieuwkamp et al., 2009) with negative consequences for everyday life functioning (return to work, leisure activities) of survivors (Buunk et al., 2015; Passier et al., 2011). Common sequelae of aSAH are anxiety, depression, fatigue, and cognitive deficits in memory, executive functions, and language (Al-Khindi et al., 2010). Furthermore, behavioural disturbances are frequently found after aSAH (Al-Khindi et al., 2010), involving inadequate interpersonal behaviour (Ogden et al., 1997; Storey, 1970), apathy (Marin et al., 1991), and impaired self-awareness (Buchanan et al., 2000; Hutter and Gilsbach, 1995; Hutter and Kreitschmann-Andermahr,

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2014).

In recent years, impairments in social cognition have increasingly been put forward as a possible explanation for these behavioural problems. Social cognition refers to the mental capacities needed to recognize and understand the behaviour of others and to react appropriately in social situations (Adolphs, 2001; Amodio and Frith, 2006; Beer et al., 2006). A distinction can be made between 'hot' and 'cold' social cognition, which are related to two different systems (Frith and Frith, 2010): the mirror system and the mentalizing system. 'Hot' social cognition allows people to understand others' feelings and show empathy (Blair, 2003), which intrinsically guides their behavioural responses to others and motivates their actions. Important components are emotion recognition and affective empathy, that is the ability to share the emotions of others, while recognizing that they are distinct from one's own (Baron-Cohen and Wheelwright, 2004). Such capacities are related to the mirror system, which facilitates understanding of others' emotions by a mechanism of motor resonance, with mirror neurons being the possible substrate. Consequently, the perception of others' emotions is converted into one's own experiences (Gallese et al., 2004; Keysers and Gazzola, 2006). 'Cold' social cognition, which corresponds with the mentalizing system, entails thinking about something from another's perspective. It is the ability to understand others' behaviour based on intentions, thoughts, and beliefs, i.e. to have a Theory of Mind (ToM) (Castelli et al., 2002; Leslie, 1987). More specifically, 'cognitive ToM' is similar to knowledge about others' intentions and beliefs, and 'affective ToM' to the understanding of others' emotional states. Affective ToM is often used interchangeably with 'cognitive empathy' (Shamay-Tsoory, 2011). The aforementioned aspects of social cognition are crucial for effective social communication and therefore for appropriate social behavioural functioning (Henry et al., 2016; Kennedy and Adolphs, 2012). The various aspects of social cognition, i.e. emotion recognition, ToM and empathy, are underpinned by several interconnected brain regions and circuits. Generally, it is assumed that the neural substrates of social cognition involve a frontal-subcortical circuit, comprising the orbitofrontal cortex and ventromedial prefrontal cortex as important regions (Adolphs, 2009; Channon et al., 2007; Lieberman, 2007; Tekin and Cummings, 2002). Evidence has been found for a mediating role of the frontal cortex together with temporoparietal areas and the hippocampus in regulation of social responses and generating a context for social information (Lieberman, 2007; Phillips et al., 2003). The amygdala circuitry, with its interconnections with prefrontal and temporal brain areas, was also found to be involved in social cognition (Siegal and Varley, 2002).

Traumatic brain injury (TBI) and stroke often result in damage to these prefrontal-subcortical brain circuits underlying social cognition. In line with this, several studies on TBI (de Sousa et al., 2012; Henry et al., 2016; McDonald, 2013; Ryan et al., 2016; Spikman et al., 2013) and stroke (Blonder et al., 2012; Happe et al., 1999; Martory et al., 2015; Wilkos et al., 2015) found impairments in social cognition, as well as associations between these impairments and behavioural disturbances. As early as in 1978, Lezak related impairments in social perceptiveness to personality changes after brain injury. More recently, deficits in interpersonal behaviour have been related to impaired emotion recognition (May et al., 2017; Radice-Neumann et al., 2007; Spikman et al., 2013) and ToM (McDonald, 2013; Milders et al., 2003) after TBI. Research on stroke also revealed an association between impaired emotion recognition and interpersonal behavioural impairments (Yuvaraj et al., 2013). In addition, the relationship between apathy and social cognition has been investigated in patients with acquired brain injury (ABI) and neurodegenerative diseases. In these studies, associations between apathy and impaired social judgments (Njomboro et al., 2014), impaired ToM (van der Hulst et al., 2015), and emotion recognition deficits (Robert et al., 2014; Rosenberg et al., 2016) have been found. Furthermore, deficits in self-awareness have been associated with impairments in ToM after TBI (Bivona et al., 2014), amyotrophic lateral sclerosis (van der Hulst et al., 2015), and schizophrenia (Pijnenborg et al., 2013). To conclude, studies in various, neurological, patient groups strongly suggest that impairments in social cognition underlie behavioural disturbances.

However, in aSAH, only few studies investigated deficits in social cognition. Brand et al. (2014) investigated empathy in a small sample of aSAH patients, finding impairments when compared to healthy controls. Furthermore, in a previous study we reported impaired emotion recognition after aSAH (Buunk et al., 2016). To date, there are no studies investigating to which extent there are impairments in a broad range of aspects of social cognition after aSAH, nor whether such impairments are related to behavioural disturbances, in particular deficits in interpersonal behaviour, apathy, and impaired self-awareness.

Because aSAH is likely to result in damage affecting frontal-subcortical circuits underlying social cognition, depending not only on aneurysm location, but also due to treatment modalities and additional aSAH-related events such as vasospasm (with possible delayed cerebral ischemia) and hydrocephalus, impairments in social cognition are likely to be found. The anterior communicating artery (ACoA) aneurysm accounts for almost 40% of all aneurysms, and a ruptured ACoA aneurysm can lead to ventromedial or unilateral frontal lobe lesions. Impairments in aspects of social cognition have been described in a few patients with an ACoA aneurysm (Heberlein et al., 2008). An early study showed that behavioural problems and personality changes were more likely to be found in aSAH patients with ACoA aneurysms (Storey, 1970). However, more recent studies found no relationship between aneurysm location and behavioural problems (Bottger et al., 1998; Hutter et al., 1995). Likely, not only focal (frontal) damage, but also diffuse neural injury post-aSAH disturbs important connections in the circuits supporting social cognition, but the relation between brain damage after aSAH and social cognition has not been investigated before.

In the present study, our aim was to investigate whether and to which extent a broad range of aspects of social cognition was impaired in the subacute stage post-aSAH. This period, 3–6 months after aSAH, is generally regarded as a clinically relevant moment to evaluate SAH patients (Zweifel-Zehnder et al., 2015). To comprise different aspects of social cognition, we investigated both 'hot' social cognition, i.e. emotion recognition, and 'cold' social cognition, that is: ToM and cognitive empathy. Also, we investigated the relationship of deficits in these aspects of social cognition with behavioural problems, more specifically deficits in interpersonal behaviour, apathy, and impaired self-awareness. It is hypothesized that, in line with prior research in other groups and given the likelihood of damage to neural circuits that underlie social cognition post-aSAH, social cognition would be impaired and related to behavioural problems. Additionally, we aimed to clarify the relationship between deficits in social cognition and focal as well as diffuse brain damage, as this has not been done before. Therefore, both frontal and non-frontal cortical lesions on Magnetic Resonance Imaging (MRI), aneurysm circulation, treatment method (clipping or coiling) and additional aSAH-related events, like hydrocephalus (acute and/or chronic) and vasospasm, were taken into account. We expect that our findings will contribute to a better understanding of the nature of behavioural disturbances after aSAH.

2. Methods

2.1. Patients and procedure

All aSAH patients that were admitted to an University Medical Centre in the Netherlands between 2010 and 2012 were eligible for inclusion. Aneurysmal SAH diagnosis was determined on computed tomography (CT) on admission, in combination with CT angiography and/or digital subtraction angiography to confirm the presence of a symptomatic intracranial aneurysm. Patients were excluded in case of current or previous neurological conditions, psychiatric disorders, or substance abuse, age under 18 years, and insufficient proficiency of the Dutch language. Neuropsychological and MRI assessment were