Tidsskriftet Tidsskrift for Den norske legeforening

Recurrent delirium caused by concealed Wolff-Parkinson-White syndrome in a 77 year old woman

CASE REPORTS

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Conflicts of interest: The author has received lecture fees from AstraZeneca, Nycomed, Roche and Pfizer.
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«Admission for social reasons» and «causa socialis» are terms often used to describe hospitalisations that are assumed to be unnecessary. However, a thorough assessment of so-called unnecessarily admitted patients may often reveal medical causes that are both serious and curable.

A 77-year old widow was admitted to the medical ward while waiting for a place in a nursing home, which she had been promised would be available within a few days. Her GP apologised on the telephone for having to burden the hospital with this patient during the waiting period. She had been discharged from the hospital to a nursing home about a year before, but for the last four months she had lived at home under the supervision of the home-care services. During the last week, she had experienced increasing panic attacks with psychotic traits, and had shouted, wept and hyperventilated. The admitting doctor described dyspnoea, reduced physical strength and insomnia. Each night, the home-care services had gone out on up to ten emergency calls. For four days preceding the admission she had been treated with zuclopenthixol (Cisordinol) and diazepam. Her other medications included atenolol, isosorbide mononitrate, lansoprazole, furosemide, acetylcysteine and diazepam.
On admission she was in a considerably reduced general condition, with possible dysarthria, a fluctuating level of consciousness and cognitive function, and the house officer who received her described periodic hyperventilation. Her blood pressure was 102/84, the pulse was a consistent 110 bpm, and her temperature was 36.6°C. Her tongue was dry, and intermittent rattling sounds could be heard over basal parts of the lungs. An examination of the heart and abdomen gave normal findings. Blood tests showed normal values for haemoglobin, inflammation markers, electrolytes, calcium, albumin and glucose. The creatine level was slightly elevated to 106 µmol/l and D-dimer to 1.0 mg/l. Arterial blood gas showed pCO_2 4.0 kPa and pO_2 7.4 kPa. The chest x-ray showed clear lungs and possibly small amounts of pleural effusion. The tentative diagnosis was admission for social reasons.
In our hospital, «social admission» has been used to refer in particular to patients who need a higher level of care on a more or less emergency basis, but who are hospitalised because the municipality cannot provide this quickly enough. The concept is poorly defined, and is used mainly when the admission is made for non-medical reasons. The patient was not discussed at the morning conference, because the admission was assumed to be unnecessary. The ECG recordings were not scrutinised any further.

An interview with her son and a home-care officer revealed that things had gone well at home until a couple of weeks prior to the admission in question. She suffered from right-side hemiparesis after a stroke nine years previously, but could walk using a cane while under supervision, and generally had a good memory. During the recent period she had felt very unsafe at home, and her son and the home-care officer agreed that she needed to move to an institution.

The history and clinical observations indicated that she suffered from delirium, previously often referred to as «acute confusional state» (1). The patient had a cognitive failure that had developed over a couple of weeks, had become restless, vexatious and disoriented, and had intermittent hallucinations (Box 1). During the doctor’s visit on the day following her admission, contact could periodically be

established with her, although she had to be woken up several times, which indicated a reduced level of attention. The nurse reported that she occasionally had moments of clarity and the condition was thus intermittent, as is normally seen in cases of delirium. The diagnosis of delirium should always lead to the next question: What precipitated the delirium, and what factor acts to maintain it?

Box 1

Diagnostic criteria for delirium according to ICD-10. All the criteria (A-F) must be met.

- A. Clouding of consciousness, i.e. reduced clarity of awareness of the environment, with reduced ability to focus, sustain, or shift attention
- B. Disturbance of cognition, manifest by both:
  - impairment of immediate recall and recent memory, with relatively intact remote memory, and
  - disorientation in time, place or person
- C. At least one of the following psychomotor disturbances:
  - rapid, unpredictable shifts from hypo-activity to hyper-activity
  - increased reaction time
  - increased or decreased flow of speech
  - enhanced startle reaction
- D. Disturbance of sleep or the sleep-wake cycle, manifest by at least one of the following:
  - insomnia, which in severe cases may involve total sleep loss, with or without daytime drowsiness, or reversal of the sleep-wake-cycle
  - nocturnal worsening of symptoms
  - disturbing dreams and nightmares which may continue as hallucinations or illusions after awakening
- E. Rapid onset and fluctuation of the symptoms over the course of the day
- F. Objective evidence from history, physical and neurological examination or laboratory tests of an underlying cerebral or systemic disease (other than psychoactive substance-related) that can be presumed to be responsible for the clinical manifestations in A-D

A renewed clinical examination on the day following the admission did not yield any findings other than that she still had a consistent tachycardia and Cheyne-Stokes' respiration. We could not detect any pain or other subjective symptoms, and she had no other emerging neurological findings.

Several of her medications have anticholinergic effects, and may thus produce side effects. Zuklopentixol is the most unfortunate one in this respect, but could hardly have caused the patient's delirium, since it was administered after the onset of her disorientation. However, zuklopentixol had most likely helped exacerbate and maintain the delirium. Furosemide is also reported to have anticholinergic effects (2), and could also contribute to delirium through dehydration, hypotension and electrolyte disturbances.

Echocardiography on the day after her admission showed sequelae following an infarction in the septum and the lower cardiac wall; the ejection fraction was estimated to approximately 40 %. Furthermore, a moderate to severe mitral insufficiency, a moderate aorta insufficiency and pulmonary hypertension were detected. Cerebral computer tomography showed an old infarction in the left hemisphere, unchanged. The most common causes of delirium could quickly be ruled out. She had no signs of infections, traumas, metabolic disturbances or stroke. Neither was there any evidence of a new myocardial infarction in the history, the ECG or the echocardiography, although the likely sequelae from an old myocardial infarction as well as her tachycardia and congestive heart failure could possibly explain the delirium. Pulmonary embolism was another possibility, with hypoxia, hypocapnia, pleural effusion and increased D-dimer. At the time, our hospital had not yet obtained a CT scanner good enough to undertake serial examination for pulmonary embolism, and patients with suspected pulmonary embolism had to be sent on a three-hour return trip to Ålesund. This is not very appropriate for a patient who is poorly and delirious, so initially we chose to pursue other avenues for differential diagnostics.

A review of her medical records showed that she had suffered from recurrent spells of tachycardia since the age of 35. At age 72 she had suffered a minor myocardial infarction, and in the subsequent period she had repeatedly been admitted to hospital for chest pains, perceived to be angina pectoris. A little less than a year prior to the admission in question she was re-admitted, and on this occasion the hospital had succeeded in undertaking an ECG during an ongoing spell of tachycardia. The records showed a supraventricular tachycardia with a frequency of 127 bpm and retrograde P waves approximately 100 ms after the start of the QRS complex (Figure 1), thus indicating a possible Wolff-Parkinson-White's syndrome (WPW). However, the diagnosis of Wolff-Parkinson-White's syndrome had not been included in the case history from this hospitalisation, and no explanation was provided as to why no preventive medication had been prescribed to the patient.

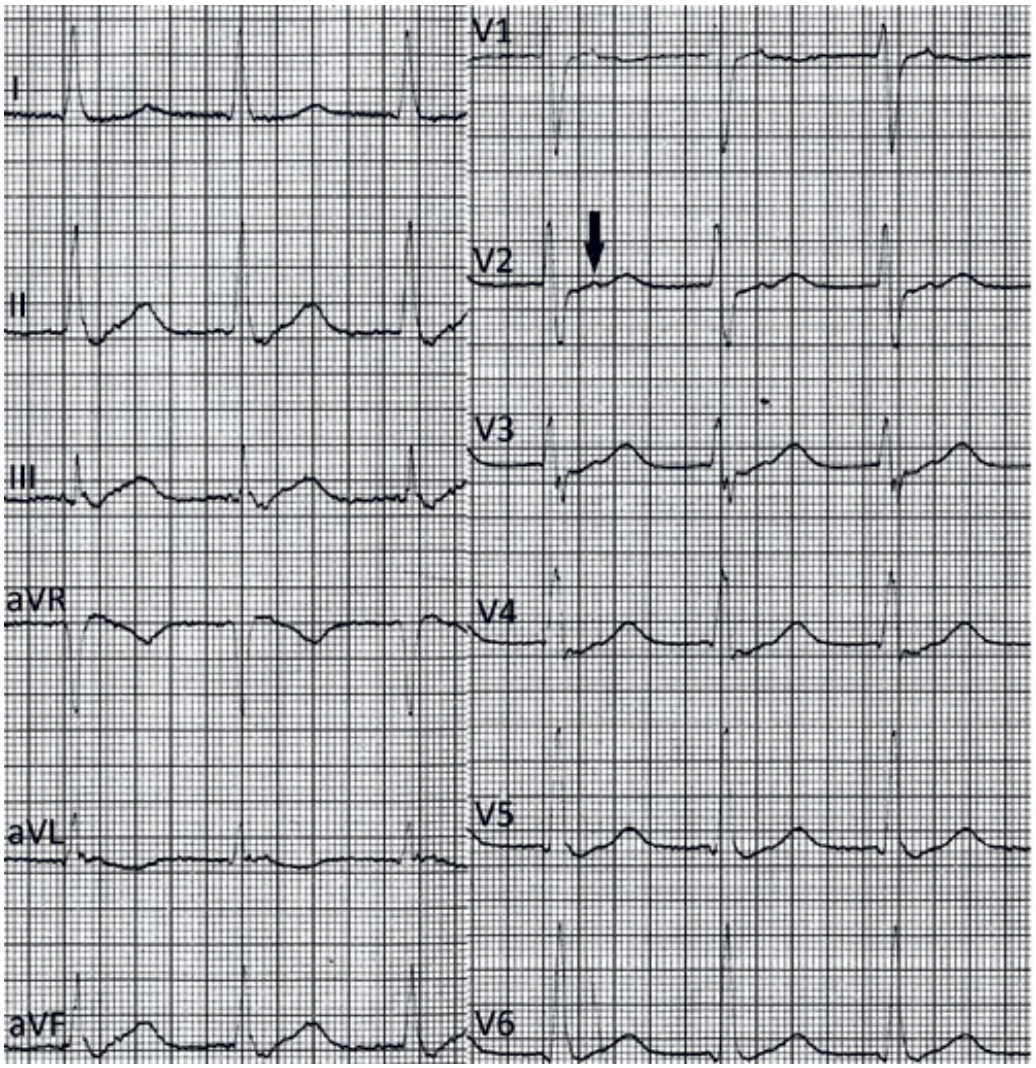

Figure 1: ECG taken approximately one year prior to the admission in question. Supraventricular tachycardia with a frequency of 127 bpm and P waves approximately 100 ms after the start of the QRS complexes (arrow) give rise to suspicion of Wolff-Parkinson-White's Syndrome (WPW).

The case history gave rise to the suspicion that the patient might be suffering from spells of supraventricular tachycardia of increasing frequency and duration, which in combination with the sequelae from the previous myocardial infarction had resulted in congestive heart failure, Cheyne-Stokes' respiration and delirium. Inappropriate treatment with psychoactive drugs worsened her respiratory problems, which in turn worsened the anxiety, and combined with the anticholinergic effects of these drugs worsened the delirium. A renewed inspection of the ECG taken during the hospitalisation in question also revealed retrograde P waves.

Supraventricular tachycardia is common in elderly people, and is divided into several sub-groups, of which atrial fibrillation and atrial flutter are the most common. In cases of atrio-ventricular reentry tachycardia (AVRT – Wolff-Parkinson-White's syndrome) and atrio-ventricular nodal reentry tachycardia (AVNRT) the patients have a congenital extra bundle between the atria and the ventricles. Because of this extra pathway, a circular electrical current can occur between the atria and the ventricles, resulting in spells of tachycardia. In classic cases of WPW syndrome, a pre-excitation of parts of the ventricular myocardium occurs through the extra bundle. This gives rise to so-

called delta waves in the ECG. In cases of concealed WPW syndrome the extra bundle conducts impulses only in one direction, from the ventricles to the atria, and thus delta waves are present only during attacks (3, 4).

Our patient had suffered from spells of tachycardia since age 35. ECGs taken when she had no spells were inconspicuous, and her type of tachycardia was therefore uncertain. ECG records taken during spells (Figure 1) indicated that she had spells with concealed WPW syndrome, even though AVNRT could not be ruled out completely. Both of these conditions can be treated by ablation with a high rate of success (> 90 %), or prophylactic anti-arrhythmic drugs can be provided. Spells can be treated with anti-arrhythmic drugs, carotid massage, other vagal manoeuvres or electroconversion. During her spells, the patient had a relatively low ventricular frequency, which could have been caused by her use of beta blockers, as well as by a slow conduction velocity in the pathway system including the AV node.

The patient was administered 450 mg of amiodarone intravenously. Sinus rhythm was established, although with a frequency of 38 – 40 bpm, her blood pressure fell and her condition deteriorated. In the following days she had short periods of sinus rhythm, and during these periods her general condition and cognitive function improved considerably. We tried to use amiodarone at a lower dosage as well as sotalol for stabilisation of the heart rhythm after having discontinued the beta blockers, but we were unable to achieve a consistent sinus rhythm. Verapamil and flecainide were out of the question because of the coronary disease and the heart failure, and ablation was not recommended because of the previous stroke. Finally, the treatment was changed back to amiodarone, combined with a permanent pacemaker to counteract the drug-induced bradycardia.
To our knowledge, she had no further spells of tachycardia, and her heart failure improved as judged by repeated chest x-rays and the degree of peripheral oedemas.. Her mind cleared and she could have sensible conversations, but her general condition was poor after her long period of immobilisation.
One month after the hospitalisation she was moved to a rehabilitation unit, and after two weeks there she was discharged to her own home under supervision by the home-care services. She was seen in the out-patient department six months later At that time she reported no further spells of anxiety, disorientation, dyspnoea or palpitation, and she had only insignificant ankle oedemas. A little more than three years later she died from pneumonia, but apart from a short stay in a nursing home immediately prior to her death, she had lived at home all the time.

Discussion

Delirium is very common. A total of 15 – 30 per cent of all patients over 75 who are admitted to medical departments as emergency cases (1) and about half of all patients who are operated for a hip fracture suffer from delirium some time during their hospitalisation (5).

Knowledge on delirium remains poor, and the condition is therefore still underdiagnosed. Patients with delirium are demanding not only in terms of care, but also in terms of diagnostics. It is difficult to obtain a reliable case history from such patients. Without a diagnosis of delirium, sufficiently thorough efforts to detect the

precipitating cause cannot be undertaken, and the chosen solution often includes applying for a place in a nursing home. A good diagnostic tool is available – the Confusion Assessment Method (CAM) (6, 7). This simple form could be used more systematically in clinical work with elderly patients. A Norwegian translation of CAM is available from the website of the Norwegian Geriatrics Society (8). Use of CAM requires permission from the copyright holder.

One often distinguishes between risk factors and precipitating factors for delirium. Among the risk factors, our patient had advanced age, organic brain disease and polypharmacy. Her arrhythmia-induced spells of heart failure were interpreted as the precipitating cause, although we cannot rule out that she may also have suffered from pulmonary embolism. At an earlier stage in life her spells of arrhythmia had been of short duration, and with a healthy myocardium she did not suffer any heart failure during the spells at that time. The spells had therefore not been diagnosed, and could have remained undiagnosed this time as well, since her ventricular frequency was as slow as 110 bpm and her clinical condition was dominated by her delirium.

Common causes of delirium in departments of internal medicine include infections, acute heart infarction, side effects of drugs, electrolyte disturbances and urinary retention. Any acute disease may in principle precipitate delirium in a frail, elderly patient, including a supraventricular arrhythmia without a particularly rapid heartbeat. However, we have not found that delirium precipitated by a concealed Wolff-Parkinson-White's syndrome has been previously described in the literature.

Delirium prolongs hospitalisation, often causes permanent institutionalisation and increases mortality. The condition may probably also precipitate or exacerbate the development of dementia (9). The pathophysiology is poorly studied, and relevant theories emphasise hypoxia, cholinergic failure, hypercortisolism and pro-inflammatory cytokines. No evidence-based medical prevention or treatment is yet available. If the patients are very restless and agitated, drugs such as haloperidol, risperidone or clomethiazole are often used to facilitate adequate examination and treatment (10). It is essential to provide the patients with a simple, quiet environment, with nightly sleep and appropriate care. Most important, however, is to diagnose and treat the precipitating factors.

Acutely ill elderly patients often present with an acute failure of the functions of daily life as the most prominent symptom. If their symptoms are met with only increased care, there is a risk that serious, although reversible, conditions may be overlooked. Acute failure to function should therefore always give rise to active diagnostic efforts, which must include a review of the case history and identification of the pre-morbid function. Our patient had alarming signs of serious disease, but the admission was initially still perceived as «unnecessary». Comprehensive geriatric assessment comprises a systematic work-up of the level of physical functioning, cognitive status, emotional status, nutritional condition, co-morbidity, medications and social networks. This is an effective approach to elderly patients with complex health problems and atypical symptomatologies (11). Due to this kind of approach, our patient was able to live in her own home for nearly three more years, almost to her death. In this case, an «unnecessary» admission ended with the implantation of a pacemaker and prescription of an anti-arrhythmic drug. If a place in a nursing home had been more available for

emergency admissions, as called for by many, the patient would have been unlikely to receive adequate treatment. Attempts to avoid «unnecessary admissions» of elderly patients may easily lead to serious loss of health and functional ability (12).

The patient's next of kin has given consent to publication of this article.

LITTERATUR

1. Inouye SK. Delirium in older persons. N Engl J Med 2006; 354: 1157 – 65. [PubMed] [CrossRef]

2. Chew ML, Mulsant BH, Pollock BG et al. Anticholinergic activity of 107 medications commonly used by older adults. J Am Geriatr Soc 2008; 56: 1333 – 41. [PubMed] [CrossRef]

3. Al-Khatib SM, Pritchett EL. Clinical features of Wolff-Parkinson-White syndrome. Am Heart J 1999; 138: 403 – 13. [PubMed] [CrossRef]

4. Tresch DD. Evaluation and management of cardiac arrhythmias in the elderly. Med Clin North Am 2001; 85: 527 – 50, xii. . xii. [PubMed] [CrossRef]

5. Juliebø V, Bjøro K, Krogseth M et al. Risk factors for preoperative and postoperative delirium in elderly patients with hip fracture. J Am Geriatr Soc 2009; 57: 1354 – 61. [PubMed] [CrossRef]

6. Inouye SK, van Dyck CH, Alessi CA et al. Clarifying confusion: the confusion assessment method. A new method for detection of delirium. Ann Intern Med 1990; 113: 941 – 8. [PubMed]

7. Wei LA, Fearing MA, Sternberg EJ et al. The Confusion Assessment Method: a systematic review of current usage. J Am Geriatr Soc 2008; 56: 823 – 30. [PubMed] [CrossRef]

8. www.legeforeningen.no/geriatri

9. Krogseth M, Wyller TB, Engedal K et al. Delirium is an important predictor of incident dementia among elderly hip fracture patients. Dement Geriatr Cogn Disord 2011; 31: 63 – 70. [PubMed] [CrossRef]

10. Ranhoff AH. Medikamentell behandling av delirium hos eldre. Tidsskr Nor Lægeforen 2004; 124: 3072-4. [PubMed]

11. Van Craen K, Braes T, Wellens N et al. The effectiveness of inpatient geriatric evaluation and management units: a systematic review and meta-analysis. J Am Geriatr Soc 2010; 58: 83 – 92. [PubMed] [CrossRef]

12. Wyller TB. For mange eller for få innleggelser? Tidsskr Nor Legeforen 2010; 130: 1702. [PubMed]

Publisert: 21. februar 2012. Tidsskr Nor Legeforen. DOI: 10.4045/tidsskr.11.0119
Received 8 March 2011, first revision submitted 22 April 2011, approved 11 August 2011. Medical editor: Trine B. Haugen.

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