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FIELD OF VISION

Sleep-disordered breathing and stroke: A relation to be considered

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Abstract

Stroke is a leading cause of death and disability. Despite expensive and elaborative research in finding out mechanisms of interrelation between sleep-disordered breathing (SDB) and stroke, there is yet much attention to be given in stroke units worldwide to the prompt diagnosis and treatment of SDB in order to improve morbidity and mortality rates related with stroke. The preventive diagnosis and treatment of SDB reduce stroke rate and improves penumbra area in case of ischemic stroke. Stroke itself predispose to SDB, making the interrelationship more complicated. The review by Parra O and Arboix A reflects the results from carefully selected reviews reported in the literature so far. This review of the literature and presentation of the original study of the Authors based on their patients' data, enhances the conviction that there exists a direct relation between SDB and stroke. Diagnosis of SDB in new stroke cases should be sought and treated carefully whenever present.

Key words: Sleep-disordered; Stroke; Hypertension; Penumbra; Breathing

Core tip: It is of paramount importance to search for a precocious diagnosis of sleep disordered breath (SDB) in newly diagnosed stroke patients in order to establish a prompt treatment. Treatment of SDB in newly diagnosed stroke patients prevent subsequent stroke episodes and lower the rate of morbidity and mortality. Penumbra zone benefits of a better recovery in case of prompt treatment of SDB in stroke. The review of the literature and presentation of the original study of Parra O and Arboix A enhances the conviction that there exist a direct relation between SDB and stroke.

Alimehmeti R, Cecia A, Seferi A, Roci E. Sleep-disordered breathing and stroke: A relation to be considered. *World J Clin Cases* 2016; 4(5): 124-126 Available from: URL: http://www.wjgnet.com/2307-8960/full/v4/i5/124.htm DOI: http://dx.doi.

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INVITED COMMENTARY

It is generally accepted that there exist an increased risk of stroke in persons that are diagnosed with sleep disordered breath (SDB). In the other hand SDB is revealed in most of patients with stroke[1,2]. Strokes can themselves generate SDB[3]. The presence of SDB is related to worst neurologic outcome after stroke. In the literature is reported that in hypertonic and diabetic patients with SDB timely recognition and treatment of SDB would prevent onset of stroke or reduce the severity of neurological deficit in case of stroke[1,2]. Sleep apnea is the most frequent SDB found in 5%-15% of the population[4]. Other authors report as higher incidence as half of the stroke patients[5]. In such case breath pattern is marked by episodes of partial or complete block of the upper airway. During the day there is sleepiness, snoring, and apneas[4].

Xie *et al*[6] conducted a thorough searched of the literature for studies on associations between SDB and the risk of stroke. They proclaimed that SDB may be a significant predictor of serious adverse outcomes following stroke and concluded that a large-scale, multicenter randomized controlled trial would confirm if better treatment of SDB would be the cause for fewer recurrent vascular events.

Despite many predisposing risk factors for SDB, such as male gender, endocrine disorders, use of muscle relaxants, smoking, fluid retention and ageing, the strongest risk factor is obesity[7]. Sleep disordered breathing is proven to be related with cardiovascular disease, cognitive impairment, and stroke.

The putative neuromechanisms behind some of the effects of SDB on the central nervous system is related with the nocturnal intermittent hypoxia and sleep fragmentation[8].

SDB increases the risk of stroke and hypertension through intermittent hypoxia with release of cytokines, angiogenic inhibitors, free radicals, and adhesion molecules. Hypertension occurs during apneas with abrupt surge in the end of apnea. Hypertension is recorded in daytime in two thirds of patients with SDB. The studies by Mohsenin *et al*[9] and Hoffmann *et al*[10] stress the important influence of SDB in hypertension and the positive effect of SDB treatment on better control of hypertension.

The sympathetic activity is revealed to be important during rapid eye movement in protecting the brain from high perfusion pressure related with hypertension. SDB is marked by prolonged sympathetic overactivity. Sympathetic system seems to be involved in pathogenesis of abnormal hemodynamics and stroke in SDB patients[11].

Another hypothesized mechanism that relates SDB with stroke is reported the potential acceleration of the process of atherosclerosis. This has to do with the influence of SDB on hypertension and contribution to insulin resistance, diabetes and dyslipidemia. Furthermore, clinical and experimental data reveal direct proatherogenic effects of SDB such as, induction of systemic inflammation and endothelial dysfunction[12]. Numerous markers of these changes have been reported in SDB patients[13].

Snoring and SDB prior to transient ischemic attacks suggest that untreated SDB may lead to stroke. Cohort studies indicate that SDB is a risk factor for stroke[14].

The acute hemodynamic and autonomic perturbations that accompany obstructive apneas during sleep, with associated repeated arousals and intermittent hypoxemia, appear to result in sustained hypertension. In addition to the metabolic and humoral effects from obesity, SDB appears to predispose individuals to autonomic imbalance characterized by sympathetic overactivity and altered baroreflex mechanisms as well as alterations to vascular function[15].

SDB is a very common condition in patients with stroke and is found in over half of stroke patients. There is a complex relationship between SDB and stroke, attributable to shared risk factors. There are numerous mechanisms by which SDB may contribute to increased stroke risk, including promotion of atherosclerosis, hypercoagulability, and adverse effects on cerebral hemodynamics. Obstructive sleep apnea is also a risk factor for hypertension, and likely for atrial fibrillation and diabetes, conditions that in turn are risk factors for stroke. SDB is also associated with poor outcomes following stroke[16]. Other authors produce evidence of SDB modifying intrathoracic pressure and heart function giving rise to intermittent hypoxemia, which may lead to vascular endothelial dysfunction and increase sympathetic drive[4]. According to Kepplinger *et al*[17] in acute cerebral ischemia, the presence of SDB is related with clinically silent microvascular cerebral lesions that may contribute to a negative functional outcome. According to the clinical experience of the authors of this commentary there is a relation between sleep-disordered breathing and multilacunar cerebral infarction probably due to chronically-altered cerebral blood perfusion during sleep. Bonnin-Vilaplana *et al*[18] first analysed SDB in acute lacunar stroke. They reported a total of 69.1% of patients with apnoe/hypopnoe index (AHI) greater than or equal to 10; 44.1% with AHI 20 or greater and 2% of the patients with AHI 30.2 or greater. Cheyne-Stokes respiration is reported in 20.6% of cases with lacunar infarction[19]. Several studies report that continuous positive airway pressure treatment can reverse pathophysiological changes in SDB, increasing insulin sensitivity and reducing blood pressure[20]. SDB is very frequent in patients with transient ischemic attacks and stroke. Treatment with continuous positive airway pressure of stroke patients with SDB is believed to prevent cardiovascular accidents and may improve neurologic deficit[21]. Neurologist should diagnose and treat properly stroke patients with SDB[5].

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CASE REPORT

Cardiac resynchronisation therapy after percutaneous mitral annuloplasty

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Author contributions: Swampillai J was involved in this case and wrote this report.

Conflict-of-interest statement: There are no conflicts of interest to report.

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Abstract

Percutaneous approaches to reduce mitral regurgitation in ischemic cardiomyopathy have stirred interest recently. Patients with ischemic cardiomyopathy and functional mitral regurgitation often meet criteria for cardiac resynchronisation therapy to improve left ventricular function as well as mitral regurgitation, and alleviate symptoms. This case shows that implantation of a pacing lead in the coronary sinus to restore synchronous left and right ventricular contraction is feasible, despite the presence of a remodeling device in the coronary sinus.

Key words: Mitral regurgitation; Cardiac implantable electronic device; Percutaneous mitral regurgitation; Ischemic cardiomyopathy; Cardiac resynchronisation therapy

Core tip: A review of cardiac resynchronisation therapy in a patient with ischemic cardiomyopathy and previous percutaneous mitral annuloplasty using a remodeling device in the coronary sinus.

Swampillai J. Cardiac resynchronisation therapy after percutaneous mitral annuloplasty. *World J Clin Cases* 2016; 4(5): 127-129 Available from: URL: http://www.wjgnet.com/2307-8960/full/v4/i5/127.htm DOI: http://dx.doi.org/10.12998/wjcc.v4.i5.127

INTRODUCTION

This case reports a successful upgrade of an implantable cardioverter defibrillator (ICD) to a biventricular ICD, despite the presence of a remodeling device in the coronary sinus.

CASE REPORT

A 76-year-old man underwent mechanical aortic valve replacement 23 years ago and developed an ischemic cardiomyopathy [left ventricular (LV) internal diastolic dimension 69 mm, ejection fraction (EF) 30%] with coronary artery disease not amenable to revascularisation. Percutaneous mitral valve annulo-

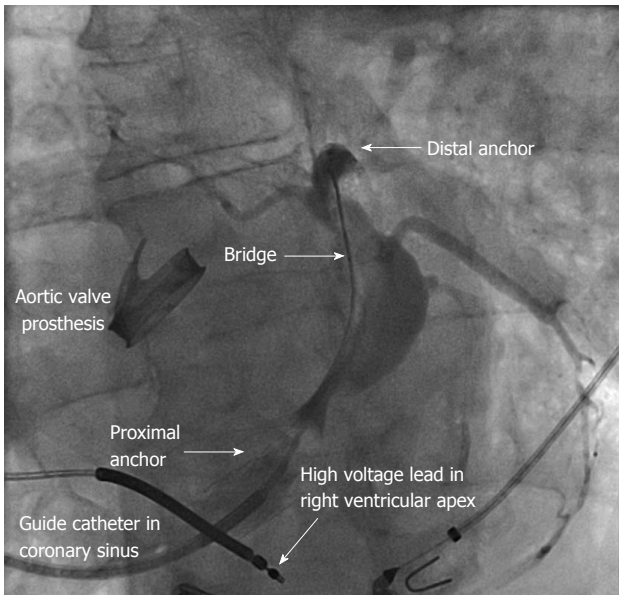

Figure 1 Balloon occlusion venogram in the coronary sinus. The proximal and distal anchors of the annuloplasty device are shown within the coronary sinus, which is filled with contrast to show target branches for the LV lead. LV: Left ventricular.

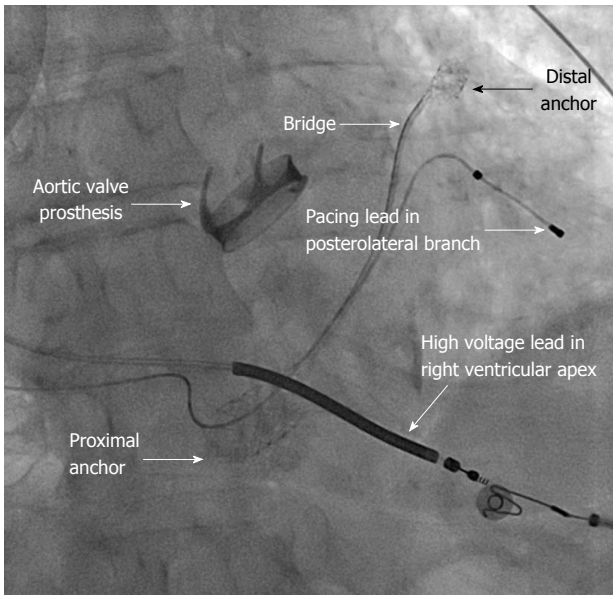

Figure 2 Pacing lead in posterolateral branch of the coronary sinus. The final position of the LV lead is shown. LV: Left ventricular.

plasty had been performed 7 years ago for severe functional mitral regurgitation, using a coronary sinus device (MONARC, Edwards Lifesciences, California). One year after mitral annuloplasty he was in persistent atrial fibrillation with a slow ventricular response rate, and as his LV function remained poor a single chamber ICD was implanted as primary prevention (Virtuoso generator with 6947 Sprint Quattro Secure lead, Medtronic, Minnesota). At generator replacement 6 years later he was in New York Heart Association (NYHA) class 3, with a paced left bundle branch block morphology, QRS duration 130 ms and his EF had deteriorated to 20%. Upgrade to a biventricular ICD was recommended in order to improve LV function, mitral regurgitation and symptoms.

After left subclavian vein access was achieved we advanced a guiding catheter over a wire and into the right atrium. We manipulated a polymer-tip wire past the proximal anchor of the annuloplasty device and into the body of the coronary sinus, and advanced the guide catheter over it. A balloon occlusion venogram was performed (Figure 1) and an 88 cm 5F Medtronic Attain Ability 4296 LV pacing lead was positioned in the posterolateral vein (Figure 2). Electrical parameters were good (threshold 1.0 V at 0.4 ms, R wave 11 mV, impedance 900 ohms) with no diaphragmatic pacing. The guiding catheter was slit and removed without dislodgement, and the lead was sutured to the pectoral muscle fascia. The existing generator was removed and a Medtronic Viva S cardiac resynchronization therapy device was connected to the leads, placed in the subcutaneous pocket, and the wound was closed. At 3 mo post-procedure the patient had symptomatically improved to NYHA class 2, with a mild improvement in LV function and mitral regurgitation on echocardiography.

DISCUSSION

Implantation of a pacing lead into the coronary sinus is potentially challenging with an annuloplasty device already in the venous system. The device consists of a distal anchor in the great cardiac vein, a flexible shortening bridge, and a proximal anchor in the proximal coronary sinus[1]. The anchors draw the proximal coronary sinus and distal great cardiac vein together, displacing the posterior annulus anteriorly, reducing mitral annulus diameter and septal-lateral distance, and thereby improving mitral insufficiency[2-4]. As left ventricular systolic dysfunction and conduction abnormalities often coexist in patients with valvular heart disease, biventricular pacing is often indicated to attempt to restore synchronous contraction and improve ventricular function. This case shows that cardiac resynchronisation therapy is possible in the presence of a mitral annular remodeling device in the coronary sinus.

COMMENTS

Treatment

Left ventricular systolic dysfunction and conduction abnormalities often coexist in patients with valvular heart disease, biventricular pacing is often indicated to attempt to restore synchronous contraction and improve ventricular function.

Experiences and lessons

This case shows that cardiac resynchronisation therapy is possible in the presence of a mitral annular remodeling device in the coronary sinus.

Peer-review

The paper is well written.

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CASE REPORT

Sepsis associated delirium mimicking postoperative delirium as the initial presenting symptom of urosepsis in a patient who underwent nephrolithotomy

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Author contributions: Nag DS, Chatterjee A and Singh H were responsible for management of the case, writing of the case report and research for the discussion; Samaddar DP was responsible for writing of the case report, final editing and research for the discussion.

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Informed consent statement: Written informed consent has been taken from the patient for the publication of the case report.

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Abstract

We report a case of 70 years old male who underwent percutaneous nephrolithotomy for renal calculi. After an uneventful recovery from anaesthesia, the patient developed delirium which manifested as restlessness, agitation, irritability and combative behavior. All other clinical parameters including arterial blood gas, chest X-ray and core temperature were normal and the patient remained haemodynamically stable. But 45 min later the patient developed florid manifestations of septic shock. He was aggressively managed in a protocolized manner as per the Surviving Sepsis Guidelines in the Critical Care Unit and recovered completely. There are no case reports showing postoperative delirium as the only initial presentation of severe sepsis, with other clinical parameters remaining normal. Both urosepsis and sepsis associated delirium have very high mortality. High index of suspicion and a protocolized approach in the management of sepsis can save lives.

Key words: Urosepsis; Nephrolithotomy; Postoperative delirium; Sepsis associated delirium; Delirium

Core tip: Postoperative sepsis can initially manifest as delirium only, with other florid manifestations developing later. When delirium is the only initial manifestation, it should be treated with haloperidol, and benzodiazepines

are best avoided. Awareness that delirium can be the only initial presentation of sepsis, having a low threshold for its diagnosis after urological surgery and aggressive early management, can save lives.

Nag DS, Chatterjee A, Samaddar DP, Singh H. Sepsis associated delirium mimicking postoperative delirium as the initial presenting symptom of urosepsis in a patient who underwent nephrolithotomy. *World J Clin Cases* 2016; 4(5): 130-134 Available from: URL: http://www.wjgnet.com/2307-8960/full/v4/i5/130.htm DOI: http://dx.doi.org/10.12998/wjcc.v4.i5.130

INTRODUCTION

Percutaneous nephrolithotomy (PCNL) is indicated for large renal stones. Although complications after PCNL have been reported to be as high as 12.5%, the incidence of serious complication like severe sepsis with multiple organ failure, indicated by the Dindo-Clavien classification of surgical complications as Ⅳ-b, has been quite low[1] (0.1%). The incidence of postoperative delirium (POD) in various studies range from 5%-15%[2] but sepsis associated delirium (SAD) has been observed in 9%-71% of patients with severe sepsis[3]. Although delirium is a major component of sepsis and "mental dysfunction may even precede the cardinal findings of sepsis"[4], there are no case reports of severe sepsis, initially presenting as SAD mimicking POD with normal hemodynamic parameters and near normal arterial blood gas (ABG). Severe sepsis as the cause of POD could be established only on development of shock and on further investigation. High index of suspicion, especially in urological surgeries, and aggressive management can save lives.

CASE REPORT

A 70 years old male presented to our emergency department with abdominal pain in the right flank. Supine computed tomography scan of the abdomen and pelvis revealed large obstructing calculi (3 cm × 3 cm) at renal pelvis. Urine analysis showed plenty of pus cells and its culture showed *Escherichia coli* sensitive to ciprofloxacin. He was discharged on a course of oral ciprofloxacin. An elective PCNL was planned for him 3 wk later.

A preoperative kidney, ureter, and bladder X-ray was done on the day prior to surgery (Figure 1).

With the patient under general anesthesia the kidney was accessed through a puncture towards the upper calyx guided by fluoroscope. While no pus was noted during the procedure, the stone of 3 cm × 3.5 cm was noted to have two layers. A white soft outer layer and a hard dark coloured inner layer. The stone was fragmented through a 28 Fr tract by pneumatic lithotripsy and was completely extracted. At the end of the procedure a Double J ureteral stent was placed antegrade and 20 Fr nephrostomy was placed. The intraoperative period was uneventful with stable hemodynamics during the entire duration of surgery lasting for 115 min. The time interval between the introduction of the nephroscope and the end of surgery was 85 min, during which about 10 L of normal saline was used as the irrigation fluid. At the end of the procedure he received 4 mg of intravenous ondansetron and 75 mg of intramuscular diclofenac sodium. Neuromuscular blockade was reversed with glycopyrrolate and neostigmine. Postoperatively he was fully awake and responding well to verbal commands. He was subsequently kept in the post-anaesthesia recovery area. His pulse rate was 76/min, blood pressure was 140/88 mmHg, and respiration was regular with no apparent discomfort. The respiratory rate remained 14-16/min and oxygen saturation (SpO_2) remained 100% with supplementary oxygen 4 L/min by face mask. The patient was comfortable for the next 30 min and had no complaints.

Over the next 50 min of his stay in the post-anaesthesia recovery area he was pain free, interacted normally with the doctors and his relatives and was doing well. Then suddenly he started getting agitated and wanted to get up from the bed and go home. He did not complain of any pain at the operated site. In the next few minutes he exhibited restlessness, agitation, irritability and combative behavior. He became disoriented about the time and place, and did not want to acknowledge that he was in a hospital or the fact that he underwent a major surgery a few hours ago. He wanted to pull out the intravenous lines, nephrostomy tube and urinary catheter. Although no formal delirium assessment tools like the confusion assessment method or delirium rating scale[5] was administered, his clinical manifestations of disturbance of consciousness (agitation), change in cognition (wanted to get up and go home, did not acknowledge that he was in a hospital), its acute onset (he was normally responsive in the immediate postoperative period) and the fact that it developed in the postoperative period met all the all four criteria (A-D) required to confirm a diagnosis of delirium as per the "Diagnostic and Statistical Manual of Mental Disorders, 4th edn, text revision (DSM-Ⅳ-TR®; American Psychiatric Publishing, Inc., Arlington, VA)"[6]. His pulse rate at this time was 91/min, blood pressure 154/86 mmHg and had regular respiration with a rate of 20-21/min with no apparent discomfort. He maintained oxygen saturation (SpO_2) of 100% with oxygen by face mask at 4 L/min. He was immediately sedated with midazolam 2 mg.

An ABG was done immediately and the report showed pH of 7.31, pCO_2 29.3 mmHg, pO_2 107.4 mmHg, lactate 0.9 mmol/L, bicarbonate 17.4 mmol/L and a base deficit of 7.0 mmol/L. The serum electrolytes were within the normal range. The Haemoglobin level was 13.1 g/dL. A chest X-ray done immediately was unremarkable and a 12-lead electrocardiogram was also normal.

However over the next 45 min his consciousness

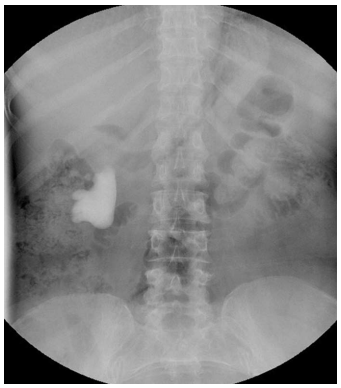

Figure 1 A kidney, ureter, and bladder X-ray demonstrating the calculi.

level started deteriorating. He became drowsy and unresponsive with a glasgow coma scale (GCS) of E1, V1, and M3: 5/15. His pulse rate was 99/min, blood pressure of 124/76 mmHg, respiration became laboured with a respiratory rate of 24-25/min and the oxygen saturation (SpO_2) dropped to 76%. Tracheal intubation was done and the patient was ventilated by manually with 100% oxygen. Over the next few minutes the patient developed overt shock with blood pressure of 93/66 mmHg and a pulse rate of 112/min with slightly warm extremities. Central venous access was secured and the central venous pressure (CVP) was observed to be low (2-3 cm of water). Since his blood pressure remained unresponsive to a 1 L of crystalloids transfused over the next 20 min, an infusion of dopamine at 6 μg/kg per minute was started. He was immediately transferred to the critical care unit (CCU).

The differential diagnosis of postoperative delirium includes arterial hypoxemia, preexisting cognitive disorder, hypoventilation with hypercapnia, metabolic derangements, drugs, alcohol withdrawal, electrolyte abnormalities, incomplete muscle relaxant reversal, seizures, acute central nervous event and infection[2,7]. All, except drugs and central nervous event, could be excluded from the available history or investigation.

In the CCU, he was mechanically ventilated and the ABG was repeated. The ABG revealed pH of 7.18, pCO_2 35.5 mmHg, pO_2 120 mmHg, lactate 8.1 mmol/L, bicarbonate 13.5 mmol/L and a base deficit of 13.9 mmol/L. Cardiac enzymes were within normal limits and an echocardiography showed good ejection fraction (69%). Although old age and many of the perioperatively used drugs including beta-blockers, narcotics, neostigmine can cause postoperative delirium, shock state, high serum lactate level along with lukewarm extremities and low CVP indicated Systemic Inflammatory Response Syndrome. The patient was empirically started on intravenous pipracillin and tazobactum 4.5 g 6 h. The core body temperature measured at nasopharynx was 34.9 ℃. The complete blood picture revealed leucocytosis, a raised total count (31000 cells/cumm) with 82% neutrophils and toxic granules on a peripheral smear.

In the CCU, severe shock necessitated intravenous infusion of crystalloids and inotropes (norepinephrine, epinephrine and dopamine). Inotropes were titrated with an aim to maintain a mean arterial pressure of > 65 mmHg. Over the next 24 h his conscious level gradually improved, core temperature normalised, inotropes were tapered and he was weaned from mechanical ventilation. Tracheal extubation was done 30 h after being shifted to the CCU. Despite negative blood and urine cultures, a diagnosis of severe postoperative urosepsis with septic shock was made and intravenous antibiotics were continued for 7 d.

He was shifted to the ward on the 4th postoperative day and discharged from the hospital on the 7th day on a 10 d course of oral levofloxacin. He had an uneventful course subsequently and the Double J ureteral stent was removed one month later. Over the next one year he was followed up in the urology clinic and remained asymptomatic.

DISCUSSION

Delirium is defined as a "transient, usually reversible cause of cerebral dysfunction and manifests clinically with a wide range of neuropsychiatric abnormalities"[8]. Postoperative delirium should be differentiated from "emergence from anaesthesia"[9], and by definition "do not have an identifiable aetiology"[2]. In our patient, the presentation of restlessness, agitation, irritability and combative behavior appeared about an hour after complete recovery from anaesthesia (50 min after being shifted to the post-anaesthesia recovery area). All the possible causes of distress experienced by the patient were excluded by the available investigations. Due to initial hemodynamic stability, normal core temperature and a serum lactate level of 0.9 mmol/L, sepsis was not suspected as the immediate cause of delirium. Normal respiratory rate, pattern, oxygen saturation, ABG and chest X-ray ruled out the possibility of hypoxemia and an ABG based electrolyte assessment also excluded dyselectrolytemia. Our patient's age and the perioperative use of narcotics, benzodiazepines, cholinesterase inhibitors and drugs with anticholinergic properties initially led us to conclude POD as the cause of the disturbing symptoms[7]. However, for treating POD, the drug of choice should have been haloperidol[7]. In fact sedatives like benzodiazepines have the potential to aggravate POD[7].

POD is a diagnosis of exclusion[10]. The multiple risk factors are illustrated in Table 1[2,7,9,10]. Although POD can manifest any time during the perioperative period, with or without any lucid interval, unlike in our patient, it most commonly develops between the 1st and 3rd postoperative day[2]. Modifying or reducing the potentially avoidable perioperative triggers consisting of pain, physical restraints, narcotics or benzodiazepines, anaemia or blood transfusion and urinary catheter can only reduce its risk[2]. Haloperidol, a D2 dopamine receptor antagonist, remains the drug of choice for treating POD[2]. It is used as an intravenous injection of

Table 1 Risk factors for postoperative delirium[2,7,9,10]

Age > 65-70 yr
Pre-existing dementia, physical or cognitive impairment
Tobacco or alcohol use
Metabolic derangements (dyselectrolytemia)
Hypoxia, hypercarbia, hypotension
Sepsis
Drug withdrawal
Nature of surgery (cardiac surgery)
Use of certain drugs (narcotics, benzodiazepines, cholinesterase inhibitors and drugs with anticholinergic properties)
Physical restraint
Sleep deprivation
Pain
Anaemia
Urinary catheter

0.5-1 mg every 15 min till the resolution of symptoms. Two to 10 mg titrated over 60-90 min is needed for most patients[2]. Careful titration is important as higher doses are associated with over-sedation for prolonged periods[2].

Based on the clinical presentation and in the absence of suggestive investigations, we initially diagnosed the case as POD, but the subsequent manifestations revealed that we were actually dealing with a patient of SAD. SAD is manifested by "acute onset of impaired cognitive function"[3] with symptoms ranging from restlessness, irritability and agitation at one end of the spectrum to sluggish mentation, inattention, stupor and coma at the other end[3]. Although the incidence of SAD has been reported to be 9%-71%[3], if electrophysiological testing is used to diagnose it, its evidence may be found in almost all cases with severe sepsis[3]. SAD manifests early in the course of sepsis, but to diagnose SAD, neurologic dysfunction must be correlated to infection or systemic inflammation[3]. SAD continues to remain as an independent predictor of death[11]. In SAD mortality increases from 16% to 63% with fall in GCS from 15 to < 8[3].

Septicemia has been infrequently observed after PCNL with an incidence of 0.9%-4.7%[12]. Life threatening complication like severs sepsis with and multiple organ dysfunction is extremely rare (0.1%)[1]. It can occur if infection is introduced during access to the kidney or if the stones are infected[13]. It is mandatory to give prophylactic antibiotics and drain a pyonephrotic kidney before doing a PCNL[13]. The volume of irrigation fluid used, duration of surgery (> 90 min), pre-existing renal insufficiency and high pressure in the renal collecting system also increases the risk of developing sepsis[13]. Although the blood and urine cultures postoperatively were negative in our patient, the clinical features (sever shock, low CVP and hypothermia) and investigations (leucocyte count, ABG) led us to a diagnosis urosepsis. The negative blood and urine cultures could have been due to use of preoperative antibiotics and administration of intravenous pipracillin and tazobactum before the cultures could be sent. In our case, white soft outer layer of the stone could have consisted of infective debris. However, culture of the stone was missed which probably could have helped in modifying the choice of antibiotics after the culture report. But our patient responded to empirical pipracillin and tazobactum and had shown remarkable clinical improvement by the 3rd day. The mortality of urosepsis is high (20%-40%)[14]. Identifying severe sepsis early is the biggest challenge and remains the "greatest barrier to implementing the guidelines"[15,16]. Early (immediate) initiation of goal directed therapy with particular focus on rapid administration of appropriate antibiotic has been shown to reduce mortality[17].

There is experimental evidence to show that in the initial phase of sepsis, endothelial nitric oxide (NO) synthase derived NO demonstrates "proinflammatory characteristics and contributes to the activation and dysfunction of cerebrovascular endothelial cells"[18]. Sepsis is also associated with "mitochondrial dysfunction" and early sepsis can cause cytokine, reactive oxygen species and NO mediated "decrease in mitochondrial ATP generation"[18]. This can result in "neural cell apoptosis and an insufficient energy supply to the neurons"[18]. The exact mechanism causing delirium is complex and involves the neurological impact arising out of the immune response causing "prolonged inflammation, brain cells activation, over expression of NO, dysfunction of intracellular metabolism and cell death"[3]. However, to the best of our knowledge, no case report has ever noted delirium presenting as the only initial manifestation of severe sepsis. None of the causes of neurological symptoms explain why it would only manifest itself without the common clinical signs of severe sepsis like temperature derangement, significant tachycardia or tachypnoea, hyperglycaemia (in absence of diabetes) or any other adverse impact on haemodynamic variables, organ dysfunction or tissue perfusion. Any laboratory investigation like the leucocyte count, plasma C-reactive protein or plasma procalcitonin would delay the diagnosis. Aggressive management based on the clinical signs and symptoms of sepsis without waiting for any laboratory evidence resulted in a positive outcome in our patient.

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COMMENTS

Case characteristics

A 70 years old male underwent percutaneous nephrolithotomy under general anaesthesia and developed delirium after complete recovery from anaesthesia.

Clinical diagnosis

Sepsis associated delirium (SAD).

Differential diagnosis
He was initially diagnosed as postoperative delirium due to absence of any other manifestation of sepsis.

Laboratory diagnosis
Subsequent development of severe metabolic acidosis with elevated serum lactate and a complete blood picture revealing leucocytosis with toxic granules on a peripheral smear indicated that the patient's symptoms of delirium was the initial manifestation of impending urosepsis.

Imaging diagnosis
A normal chest X-ray and echocardiography showing good ejection fraction (69%) excluded respiratory or cardiac cause for the developing symptoms.

Treatment
The patient managed with organ support and intravenous antibiotics in the critical care unit.

Related reports
The clinical presentation initially led the authors to a diagnosis of postoperative delirium; however the subsequent manifestations of Systemic Inflammatory Response Syndrome (SIRS) revealed that they were actually dealing with a patient of SAD. The exact mechanism causing delirium is complex and involves the neurological impact arising out of the immune response.

Term explanation
SAD has an acute onset and is a potentially reversible organic brain dysfunction in patients with SIRS or sepsis. Postoperative delirium is a diagnosis of exclusion and can be attributed to any delirium in the postoperative period which does not have an identifiable cause.

Experiences and lessons
Urosepsis can initially manifest as delirium without any other common clinical signs of severe sepsis. Haloperidol is the drug of choice in delirium and benzodiazepines are best avoided. Aggressive management of sepsis without waiting for any laboratory evidence can result in better outcomes.

Peer-review
This is a rare and very interesting case report.

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CASE REPORT

Removal of a large foreign body in the rectosigmoid colon by colonoscopy using gastrolith forceps

Xiao-Dong Lin, Guang-Yao Wu, Song-Hu Li, Zong-Quan Wen, Fu Zhang, Shao-Ping Yu

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Author contributions: Lin XD and Wu GY wrote the article; Lin XD, Li SH and Zhang F obtained the data; Wu GY analyzed the data; Lin XD and Wen ZQ performed the surgery; Yu SP guided the whole work; all authors approved the final version.

Institutional review board statement: This case report was exempt from ethical approval by the Ethics Committee of Dongguan Kanghua Hospital.

Informed consent statement: The patient involved in this study gave his written informed consent authorizing use and disclosure of his protected health information.

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Abstract

Rectal foreign bodies are man-made injury that occurs occasionally. The management depends on its depth and the consequence it caused. We here report a case of rectal foreign body (a glass bottle measuring about 38 mm × 75 mm) which was located 13-15 cm from the anus. The patient had no sign of perforation, and we managed to remove it using endoscopy with gastrolith forceps.

Key words: Foreign body; Rectosigmoid; Endoscopy; Removal; Gastrolith forceps

Core tip: Rectal foreign bodies happen occasionally. The majority of rectal foreign bodies inserted by adults are for self-gratification. As such they are likely to be smooth, rounded, cylindrical, or egg shaped to allow ease of introduction and removal. The factors that determine whether a rectal foreign body can be removed transanally are the shape, size, location of the object, and the presence or absence of perforation. We here report a case of rectal foreign body (a glass bottle measuring about 38 mm × 75 mm) which was located 13-15 cm from the anus. The patient had no sign of perforation, and we managed to remove it using endoscopy with gastrolith forceps.

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INTRODUCTION

The majority of rectal foreign bodies inserted by adults are for self-gratification. As such they are likely to be smooth, rounded, cylindrical, or egg shaped to allow ease of introduction and removal. The factors that determine whether a rectal foreign body can be removed transanally are the shape, size, location of the object, and the presence or absence of perforation. We here report a case of rectal foreign body (a glass bottle measuring about 38 mm × 75 mm) which was located 13-15 cm from the anus. The patient had no sign of perforation, and we managed to remove it using endoscopy with gastrolith forceps.

CASE REPORT

A 40-year-old male was admitted due to retention of a large foreign body in the rectal colon (Figure 1). He put a glass bottle into his anus in his self-sexual play 3 d before admittance. On physical examination, a cylindrical shape glass plat about 4 cm in diameter could be touched in the rectal touch 4 cm from the anus. Plain abdominal radiograph revealed a bottle shape foreign body (measuring about 38 mm × 75 mm) retained in hypogastric zone adjacent to pubic symphysis, without sign of perforation. The open of the bottle was in the proximal region of the colon, and the basal of the bottle was near the anus, which made it difficult to remove by anal speculum. The bottle slid into the rectosigmoid colon deeper during the procedure (Figure 2). We decided to have a colonoscopy as a last method before cutting him open. In the colonoscopy, it was found that a wide basal glass bottle filled the colon 15 cm from the anus. The use of snare and basket failed to noose the bottle (Figure 3). We decided to use gastrolith forceps to noose the bottle. After full gas filling, we were able to noose the basal part of the bottle. The patient felt painful, so lumbar anesthesia was performed to relieve the spasm of the colon and pain. The bottle was carefully and slowly dragged to the open of the anus, and it was successfully removed outside the patient by colonoscopy. There was no sign of bleeding, injury or perforation in the post-extraction sigmoidoscopy and 2 d observation following. Then the patient was discharged. The patient had no perforation, bleeding or fecal incontinence during one-month follow-up.

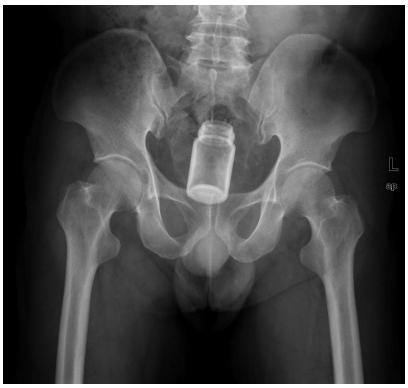

Figure 1 A 40-year-old male was admitted due to retention of a large foreign body in his rectal colon. Plain abdominal radiograph revealed a bottle shape foreign body (measuring 38 mm × 75 mm) retained in the rectal colon, with no sign of perforation.

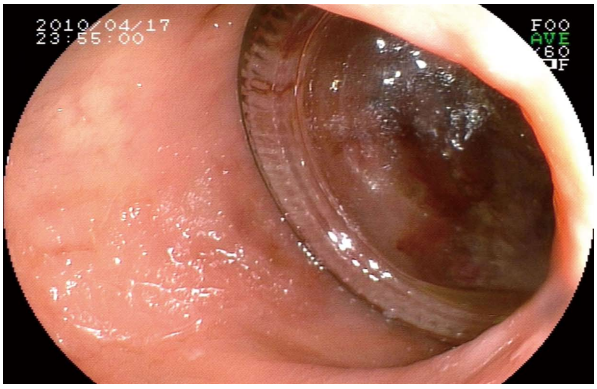

Figure 2 Bottle slid into the rectosigmoid colon deeper during examination. We decided to have a colonoscopy as a last method before cutting him open. In the colonoscopy, the glass bottle filled the colon 15 cm from the anus.

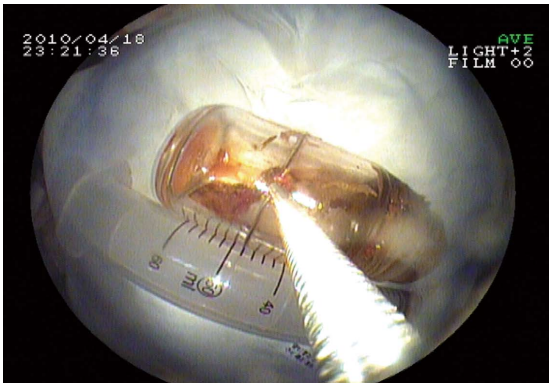

Figure 3 The use of snare and basket failed to ensnare the foreign body. We used gastrolith forceps to retrieve the bottle. The bottle was carefully and slowly manipulated to the opening of the anus and successfully removed out by colonoscopy.

DISCUSSION

The majority of rectal foreign bodies inserted by adults are for self-gratification[1]. As such they are likely to be smooth, rounded, cylindrical, or egg shaped to allow ease of introduction and removal[2]. The factors that determine whether a rectal foreign body can be removed transanally are the shape, size, location of the object, and the presence or absence of perforation[2]. In the non-perforated stable patient, the object should be removed with a local block and/or conscious sedation

via the transanal approach. If this fails, the patient should be brought to the operating room for a deeper anesthetic and attempt at transanal extraction. Surgery with a laparotomy should be reserved for patients with perforation or ischemic bowel or cases of failed transanal attempts[3]. Most of rectal foreign bodies can be removed transanally, but when they go deeper to the sigmoid colon, it is impossible to extract except *via* colonoscopy[4]. In our patient, the basal, smooth, large part of the bottle was in the distal colon, which makes it much difficult to remove out. We used gastrolith forceps to loop the bottle, and seized it tightly enough to extract.

COMMENTS

Case characteristics
A 40-year-old male presented seeking for medical help due to rectal foreign body insertion.

Clinical diagnosis
Rectal foreign body (glass bottle) insertion.

Differential diagnosis
Bowel perforation, intestinal necrosis, and foreign body rupture.

Imaging diagnosis
A bottle shape foreign body (measuring about 38 mm × 75 mm) retained in hypogastric zone adjacent to pubic symphysis, without sign of perforation.

Pathological diagnosis
Rectal foreign body (glass bottle).

Treatment
Removed using colonoscopy.

Related reports
Foreign bodies of different shapes were reported, little smooth ones can be removed *via* a transanal approach while irregular shape ones needed to be surgically removed.

Term explanation
RFB: Rectal foreign bodies.

Experiences and lessons
For removal of rectal foreign bodies *via* a transanal approach, relaxation of patients and full dilation of the anus are very important.

Peer-review
This is an interesting case and the X-ray imaging showed a typical rectal foreign body. Anesthesia before retreating the foreign body should be considered.

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