Spontaneous non-traumatic hip dislocation in patients with stroke

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Accepted 22 June 2022

To cite: Nguyen V, Bollens B, Moyaux Z, *et al*. *BMJ Case Rep* 2022;**15**:e249549. doi:10.1136/bcr-2022-249549

SUMMARY

This is a unique clinical case of a spontaneous non-traumatic hip dislocation in a patient with stroke with a hip and knee flexion pattern. This case highlights the role of spasticity as a cause of hip dislocation and the need to combine focal spasticity treatment and surgery to restore ambulation.

BACKGROUND

Development of poststroke spasticity is common with a prevalence of up to 20%–30%.[1] Lower limb spasticity is frequently associated with gait impairment, activity limitation, restriction of mobility and social interaction, which can impact a stroke survivor's quality of life.[2] Sudden increases in poststroke spasticity can result in joint contractures in hip flexion, knee flexion and/or equinovarus foot position, which in turn may lead to worsening gait.

Spontaneous hip dislocation is classically described in children and adolescents with cerebral palsy[3] but has never been described in patients with stroke. In the adult population, hip dislocation generally results from an inciting high-energy trauma.[4 5]

In this case report, we describe the occurrence of a spontaneous hip dislocation in a poststroke spastic hemiplegic adult patient.

CASE PRESENTATION

This is a case of a woman in her 60s with a left hemiparesis secondary to a haemorrhagic stroke. She benefited from focal spasticity management with botulinum toxin A injections (BoNT-A) followed by a selective tibial nerve neurotomy for spastic equinovarus foot allowing her to walk without an orthosis. Spasticity was graded three-fourth on the Modified Ashworth Scale in the quadriceps, hamstrings and gastrocnemius muscles and treated with oral baclofen (25 mg to 12.5–25 mg). Her functional ambulation category (FAC) was graded 5.[6]

Six years poststroke, the patient reported of difficulty walking. X-ray of the left hip showed moderate coxarthrosis and an intensive rehabilitation treatment programme was prescribed (figure 1). One year later, she was unable to walk as a result of a 'triple flexion' (hip flexion, knee flexion and plantar ankle flexion) spastic pattern of the lower limb. The clinical examination showed the left hip in a 90° fixed flexion position along with an increase in hip flexors and hamstrings muscle tone and an inability to activate the quadriceps muscle. Abdominal and skin examinations were normal. Her FAC was graded 0. A new X-ray revealed a posterior left hip dislocation with bone defect of the posterosuperior acetabulum (figure 2).

INVESTIGATIONS

Not relevant.

DIFFERENTIAL DIAGNOSIS

On the 1-year preoperative hip X-ray, acetabular dysplasia cannot be completely excluded. The hip dysplasia could be worsened by the adductor spasticity, which may in time lead to the development of hip osteoarthritis and progressive subluxation.

TREATMENT

After multidisciplinary counselling and patient consent, the patient underwent and benefited from BoNT-A injections into the medial hamstrings (100 UI), gracilis (50 UI) and biceps femoris (50 UI) muscles for a total dose of 200 UI incobotulinum toxin type A followed by a total hip replacement with dual mobility design (figure 3).

The patient was placed in a lateral position to perform a posterior hip surgical approach. Trabecular metal was used to rebuild the superior defect of the acetabulum. An acetabulum Avantage and a Wagner Cone Prosthesis Stem with a neck angle of 135° were used. Gluteus minimus was reinserted and fascia lata was closed at the end of the surgical intervention. Postsurgery, the patient was able to have immediate, complete, painless body weight loading.

OUTCOME AND FOLLOW-UP

Her postoperative status at 2-year follow-up continues to be favourable with hip mobility recovery, correction of triple flexion pattern and rapid recovery of independent gait ability and her FAC was again graded 5. The patient continues to walk with a stiff knee gait and donns an AFO (Ossur) to compensate for her left equinus ankle position in the swing phase. Her walking distance exceeds 1 km.

The patient is still on baclofen. BoNT-A injections were not continued. Her left hip mobility was complete with full extension, 130° of flexion and 50° in abduction.

DISCUSSION

Spastic muscle overactivity is a common disorder in patients with central nervous system lesion. A

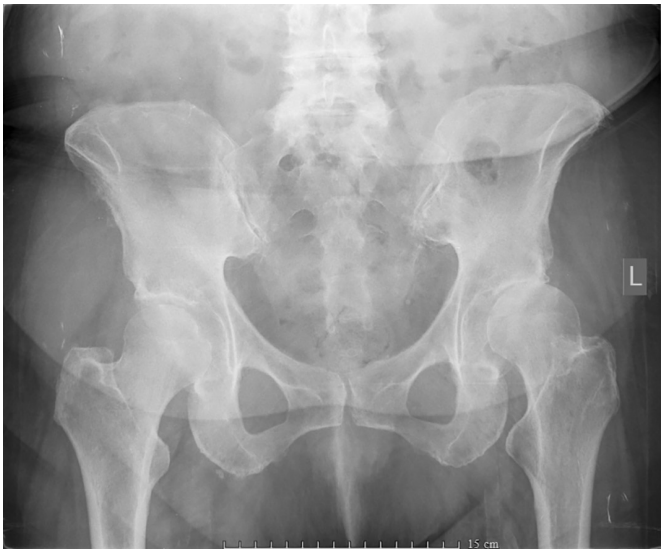

Figure 1 Left hip osteoarthritis.

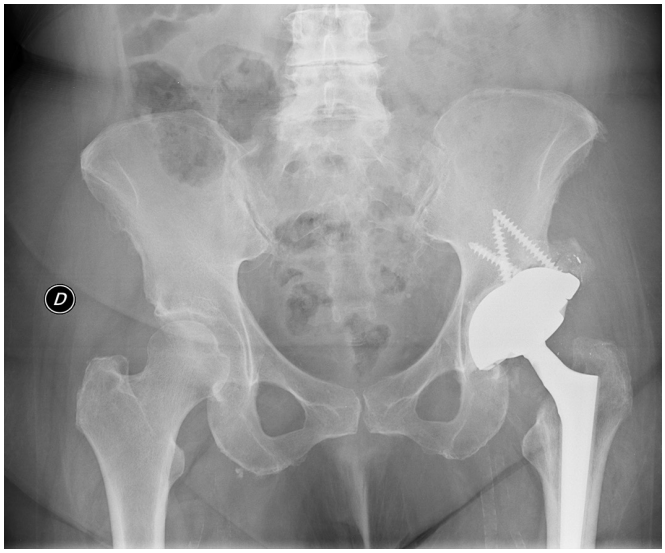

Figure 3 Total hip replacement—6 week post-operative.

sudden increase in muscle tone often results from noxious stimuli such as urinary tract infection, kidney stones, faecal impaction, bedsores, deep vein thrombosis, heterotopic ossification, fracture or a joint pathology. Adequate treatment is often sufficient to bring back spasticity to basal values.[7]

This patient was presenting with progressive worsening of functional status, resulting in quadriceps weakness, triple flexion of the lower limb and gait dysfunction. Particular attention had to be paid to exclude a new hip pathology. We hypothesise that moderate coxarthrosis progressively induced pain, which in turn led to a significant increase in hip spasticity with resultant hip dislocation and inability to walk.

Treatment of hip dislocation and coxarthrosis was necessary to restore the patient's functional ability, but preoperative treatment of the hip spasticity was essential for a successful surgical intervention.

In the literature, spontaneous hip dislocation is mostly focused in patients with cerebral palsy. Hip dysplasia and subsequent osteoarthritis are well documented in adolescent and young adults. Pain induced by coxarthrosis and abnormal constraints applied by spastic and/or shortened muscles to the growing skeleton progressively lead to hip instability, subluxation and finally hip dislocation.[8 9] Close monitoring is necessary and early surgery is described as the main treatment option.[10] In infants with spasticity, surgical treatment often consists of an open reduction associated to pelvic osteotomy and/or femoral osteotomy with a nearly 90% success rate and an acceptable rate of complications observed.[9 11 12] This surgery is sometimes associated with an additional soft tissue procedure, including tenotomy of the adductors and hip flexors or iliopsoas release, which depends on the extent of muscle contractures around the hip joint.[9 13] Intramuscular injection of BoNT-A could also be as efficient as soft-tissue surgery in prophylaxis of progressive hip subluxation or dislocation in children with cerebral palsy.[14]

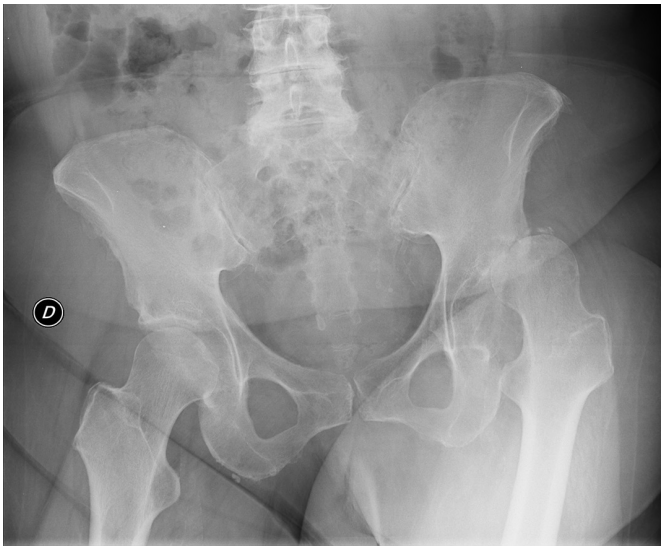

Figure 2 Posterior left hip dislocation.

In adult patients, total hip replacement is one of the recommended treatments of coxarthrosis and hip fracture dislocation.[15 16] Patients with upper motor neuron syndromes can have problematic spasticity that can lead to hip dislocation in their spastic limb. Strong guidelines or treatment recommendations are lacking regarding patients undergoing total hip replacement to their spastic limb. Total hip replacement/prosthesis could be a reasonable treatment of osteoarthritis in patients with stroke as it can reduce pain and improve functional outcomes.[17]

BoNT-A injections were proposed in this case to decrease spasticity in the preoperative period and facilitate surgery, similar to what is done to prevent hip dislocation in cerebral palsy children.[18] No additional soft tissue procedure was performed because of the lack of associated muscle contracture. However, the surgical approach is another important treatment modality to help decrease further hip instability in spastic patients. Several prosthetic options and surgical approaches have been tested to prevent hip instability, including constrained liners and dual mobility constructs. No difference was found between both techniques regarding functional outcomes, but higher survival rates, lower dislocation rates and lower acetabular loosening rates were found when using dual mobility cups designs.[19]

Patient's perspective

'Since my stroke, I was regularly followed for my spasticity. Several botulinum toxin injections and finally a neurotomy allowed me to recover a walk without orthosis.

When my spasticity suddenly increased, I did not understand what happened. I was not able to walk anymore and my independence dramatically decreased. New investigations helped to understand the problem. The multidisciplinary approach and the combined treatment of spasticity and surgery helped me recover my ability to walk, which improved my quality of life.'

Learning points

- This is the first case of a patient with poststroke spasticity and hemiparesis suffering from a spontaneous non-traumatic hip dislocation.
- A hip joint pathology has to be excluded when presented clinically with a sudden increase in spasticity, resulting in triple flexion of lower limb.
- Treatment of both spasticity and coxarthrosis was necessary to restore the ability for independent walking/mobility.

Contributors VN wrote the case report. TD was responsible of botulinum toxin injection and the follow-up of the patient. BB, ZM and TD corrected the paper.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s)

Provenance and peer review Not commissioned; externally peer reviewed.

Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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