Toxic shock syndrome: A rare complication to enhanced external counterpulsation

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Enhanced external counterpulsation (EECP) is known to reduce angina pectoris in patients in whom revascularization is not possible. The therapy is associated with few adverse effects. A case with a previously unknown complication – toxic shock syndrome – that occurred twice in an EECP-treated patient is described. Toxic shock syndrome initially resembles the state of septic shock. Early recognition of the syndrome and initiation of therapy is of vital importance to prevent rapid progression and a possibly fatal outcome. Awareness of this condition among cardiologists offering EECP is essential.

Key Words: EEC; Enhanced external counterpulsation; Toxic shock syndrome; TSS

Enhanced external counterpulsation (EECP) is a noninvasive therapy for angina pectoris offered to patients with coronary artery disease who are not candidates for revascularization. EECP has been shown to reduce chest pain in patients with refractory, stable angina pectoris (1). During treatment, cuffs are fastened around the lower extremities. During diastole, the cuffs are inflated to a pressure of 260 mmHg to 300 mmHg. The treatment is usually given for 1 h five days a week for seven weeks.

Toxic shock syndrome (TSS) is caused by staphylococcal or streptococcal toxins. The syndrome is clinically characterized by its rapid progression starting with fever, confusion, myalgia and gastrointestinal symptoms, and it is associated with a rash. After one to two weeks, the rash undergoes desquamation, especially in the palms and soles. The condition, which may resemble septic shock, has a mortality rate exceeding that of meningococcal septicemia. Early awareness of TSS is important for the right diagnosis to be made and the right treatment to be started. The antibiotic clindamycin has been shown to be an effective therapy for TSS in animal studies (2).

CASE PRESENTATION

A 64-year-old man with a long history of ischemic heart disease was admitted with a rapid onset of fever, confusion and hypotension. On admission, his blood pressure was 100/65 mmHg, but soon dropped to 75/40 mmHg in spite of fluid replacement. On his right lower extremity, an area of sharply demarcated erythema appeared with no underlying bruising or skin defects. Broad-spectrum antibiotics and vasopressor therapy were started, and the blood pressure normalized. The blood tests showed an activation of the coagulation cascade initially with no clinical signs of bleeding abnormalities. Leukocytes and differential count were normal, but C-reactive protein was 213 mg/L (normal upper limit is 5 mg/L). Creatine phosphokinase was 730 U/L (normal upper limit is 240 U/L). Septic shock was suspected and the patient was transferred to the intensive care unit.

Two days later, the patient developed dyspnea and hemoptysis. A chest x-ray showed diffuse infiltrations in both lung fields, which was interpreted as acute respiratory distress syndrome. On the fourth day of

Le syndrome du choc toxique : Une complication rare de la contre pulsation externe à haut rendement

Il est connu que la contre pulsation externe à haut rendement (CEHR) atténue l'angine de poitrine chez les patients qui ne peuvent pas subir de revascularisation. Peu d'effets secondaires s'associent à cette thérapie. Les chercheurs décrivent un cas s'accompagnant d'une complication auparavant inédite, le syndrome du choc toxique, observé deux fois chez un patient traité par CEHR. Au départ, le syndrome du choc toxique évoquait un choc septique. Il est essentiel de dépister le syndrome et d'amorcer la thérapie le plus vite possible pour prévenir une évolution rapide et une issue au potentiel fatal. Les cardiologues qui effectuent la CEHR devraient être au courant de cette complication.

admission, TSS was diagnosed and clindamycin was added to the antibiotic regimen. At this point, desquamation in the area of erythema on the right lower extremity had started.

After a few days, the vasopressor therapy was discontinued, the patient improved and the laboratory results were normal. On discharge five days later, the patient had completely recovered.

The symptoms prompting hospitalization started 12 h after the first session in a planned EECP course. During EECP therapy, the patient was dressed in specially manufactured skin tights (Vasomedical Inc, USA) and the treatment was commenced with a diastolic augmentation pressure of 80 mmHg, which was slowly increased to 260 mmHg over 20 min. No defects or exanthema were present on the patient's lower extremities before the EECP treatment.

After two months, the patient started another course of EECP. Once again, a few hours after the first session, he developed fever, confusion, shock and demarcated erythema on his right lower extremity. Treatment with broad-spectrum antibiotics, including clindamycin as well as vasopressor therapy, was started and the condition improved after a few hours of treatment. This time, there was no desquamation.

No microbiological agents were isolated from the blood or the area of erythema during either of the two admissions, but a group B streptococcus was isolated from the nasal cavity.

Six months after discharge, the patient still had severe angina and was eager to resume EECP. Although a toxin-producing microorganism could not be isolated, except for the group B streptococcus, it was decided that the patient would be given prophylactic antibiotics with clindamycin during the entire EECP course. No further complications were observed.

DISCUSSION

According to the International EECP Patient Registry, EECP is a safe procedure. In a series of 978 EECP courses (3), only 11 of these high-risk patients were withdrawn due to serious cardiac events; no incidence of infection was observed. Occasionally, patients have mild skin irritation, but bruising and pressure sores due to the mechanical pressure from the blood pressure cuffs are rare. Local soft tissue infections

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and systemic spread have not previously been described in the literature. In general, EECP should not be initiated in case of symptomatic peripheral vascular disease, history of varicosities, deep vein thrombosis, bleeding diathesis, phlebitis or stasis ulcers (1), or other signs of infection.

The symptoms of TSS observed 12 h after an EECP session could be a coincidence. However, the existence of two episodes developing a few hours after initiation of EECP suggests that pathogens colonizing the skin were transmitted into the soft tissue by the repeated mechanical pressure of the cuffs.

Toxins are known to be able to penetrate mucous membranes. Thus, TSS has been demonstrated in women using vaginal tampons colonized by pathogen bacteria. According to our knowledge, TSS following mechanical action on normal skin has not previously been described.

Microorganisms cannot always be isolated in patients suffering from TSS (2). In the present case, no definite causative organism was isolated. The group B streptococci isolated from the nasal cavity could, nevertheless, have been the responsible agent because these bacteria have the potential to produce toxins capable of inducing TSS (4).

CONCLUSION

The present case indicates that EECP may trigger TSS and that clindamycin taken during an EECP course may prevent the development of TSS. Cardiologists involved in EECP should be aware of the signs of TSS. Early contact with specialists in infectious diseases is important to prevent a possibly fatal outcome.

REFERENCES

1. Arora RR, Chou TM, Jain D, et al. The Multicenter study of enhanced external counterpulsation (MUST-EECP): Effect of EECP on exercise-induced myocardial ischemia and anginal episodes. *J Am Coll Cardiol* 1999;33:1833-40.
2. Lappin E, Ferguson A. Gram-positive toxic shock syndromes. *Lancet Infect Dis* 2009;9:281-90.
3. Barsness G, Feldman AM, Holmes DR, Holubkov R, Kelsey SF, Kennard ED. The International EECP Patient Registry (IEPR): Design, methods, baseline characteristics and acute results. *Clin Cardiol* 2001;24:435-42.
4. Schlievert PM, Gocke JE, Deringer JR. Group B streptococcal toxic shock-like syndrome: Report of a case and purification of an associated pyrogenic toxin. *Clin Infect Dis* 1993;17:26-31.