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Short reports

Enterococcal endocarditis after extracorporeal shock wave lithotripsy for nephrolithiasis

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Summary

We report a case of enterococcal endocarditis following extracorporeal shock wave lithotripsy (ESWL) for ureteral stone. Although endocarditis following ESWL is very rare, transient bacteraemia occurs during ESWL. This case is a reminder that enterococcal endocarditis may follow innovative genitourinary procedures without appropriate prophylaxis.

Keywords: endocarditis, lithotripsy, nephrolithiasis

Enterococcal endocarditis has a genitourinary or gastrointestinal source in 40% to 50% of cases.[1] Extracorporeal shock wave lithotripsy (ESWL) is currently the major mode of treatment for nephrolithiasis. The rate of bacteriuria after ESWL is 5% while the rate of urosepsis is less than 1%.[2] Endocarditis after lithotripsy has previously only been reported once.[3]

Case report

A 61-year-old man was referred to the Urologic Clinic because of attacks of left renal colic. Intravenous pyelography revealed a 9-mm calculus in the left mid-ureter. The patient was hospitalised for ESWL. On physical examination his temperature was 36.7°C; a systolic murmur was heard over the left sternal border and rectal examination revealed an enlarged prostate gland. Urinalysis was normal and urine culture was sterile. ESWL was performed successfully aided by stent insertion to the left ureter (at the same sitting). A total dose of 2700 shock waves was delivered in 50 minutes with incomplete disintegration of the stone and particles sized 2–4 mm remaining. The patient was discharged the following day. No antibiotic treatment was given prior to or following the ESWL treatment. On follow-up three weeks later urinalysis showed five red blood cells per high power field; urine culture was negative, there was no evidence on intravenous pyelography of a stone or hydronephrosis. A month later the patient complained of fever and chills. His family physican treated him with amoxicillin, 500 mg tid, but no improvement was noted and he was admitted to hospital.

On examination his temperature was 39°C, pulse 100 beats/min, blood pressure 160/90 mmHg. A harsh mid-systolic murmur grade 3/6 was heard along the left sternal border. There was no hepato-splenomegaly. Inspection of skin, nails and eyes showed no evidence of embolic phenomena. Chest X-ray showed no infiltrate, an enlarged left ventricle, and the electrocardiogram revealed sinus rhythm with signs of left ventricle hypertrophy.

Laboratory tests revealed a sedimentation rate of 105/h, haemoglobin 11.8 g/dl, leukocytes 15.6×10^9/l, (88% neutrophils), creatinine, urea, electrolytes and liver enzymes were in the normal range. The urine showed numerous red blood cells and few leukocytes. Ultrasound examination of the kidney disclosed normal-sized kidneys with no evidence of nephrolithiasis or hydronephresis. Transthoracic echocardiography revealed a hypertrophic left ventricle, prominence of the septum, a gradient of 42 mmHg over the left ventricular outlet, and a systolic anterior movement of the anterior mitral cusp consistent with hypertrophic obstructive cardiomyopathy; no vegetations were observed. Amoxicillin treatment was stopped and urine and four blood cultures were obtained during the following 24 hours. Subsequently ceftriaxone 1 g daily intravenously was commenced for a presumed urinary tract infection. During the following days the temperature did not fall below 38°C. Urine culture grew *Streptococcus* and *Staphylococcus* 1000 colonies/ml (final identification was not done) but blood cultures were sterile. Vancomycin 1 g bid was started. After four days of a gradual decrease in temperature there was an episode of rigors and the temperature rose to 39°C. Antibiotic treatment was stopped; four more blood cultures were obtained, an abdominal computed tomography (CT) scan revealed a 3 × 4 cm lesion on the spleen, compatible with an infarct.

Enterococcus faecalis sensitive to ampicillin and vancomycin grew in the blood cultures. Rheumatoid factor was 45 IU/ml (normal <30), C-reactive protein was 201 mg/dl (normal <10). Transoesophageal echocardiography revealed thrombus in the left auricle but no vegatations. Ampicillin 3 g qid and gentamicin 80 mg tid, both intravenously, were started and continued for four weeks; the patient recovered completely.

Discussion

We believe that this patient suffered from *Enterococcus faecalis* endocarditis with a splenic embolus following ESWL. The diagnosis of

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infective endocarditis is probable according to the von Reyn criteria and definite by current criteria for endocarditis (box 1).[4] The initial blood cultures were negative, probably due to prior amoxicillin treatment, the recurrence of fever and rigors following four days of vancomycin therapy might have been related to the embolic phenomenon. Up to 50% of patients with enterococcal endocarditis have had recent surgery or intervention on the genitourinary tract.[1] The major evidence for a relation between certain procedures and infective endocarditis is circumstantial; there is no documentation of bacteraemia during or after the specific procedure claimed to be the source.[1,5] The temporal relation between ESWL and endocarditis in our case is in the range described following certain procedures.[5]

The rate of bacteriuria after ESWL is 5% while the rate of urosepsis is less than 1%.[2] The risk of transient bacteraemia during invasive urologic procedures has been established previously. Bacteraemia of urologic origin may occur even when the urine is sterile but is much more common (approximately five times) in the presence of infected urine.[5]

The incidence of bacteraemia during ESWL varies widely between studies. Two series reported low rates of 2.9% and 4%, most positive cultures were due to contamination in these two series.[6] Other series documented 14.3%, 10% and 7.7% rates of bacteraemia during ESWL monotherapy.[7-9] It should be noted that in two of these series enterococci were major isolates.[7,8] It is likely that ESWL carried out with manipulation of the urinary tract poses a greater risk for bacteraemia than ESWL alone. In one series when ESWL was performed with various manipulations of the urinary tract, the rates of bacteraemia were 5% to 40%.[8]

It is likely that transient bacteraemia during ureteral stent and ESWL was the cause of the infective endocarditis in our case. In our literature search we found only one case report of infective endocarditis after lithotripsy. In that case a patient with known valvular heart disease received trimethoprim sulfamethoxazole before ESWL; when the subsequent urine culture yielded group D enterococci, treatment was changed to nitrofuradantin.[3] Four months later the patient presented with infective endocarditis.

Endocarditis: criteria

Major
- positive blood cultures

Minor
- fever
- predisposing heart disease
- vascular phenomena
- immunologic phenomena

Box 1

Learning points

- ESWL, a non-invasive procedure, may cause bacteraemia
- prophylaxis for endocarditis should be considered before ESWL for high-risk patients

Box 2

Irrespective of risk for infective endocarditis, all patients with bacteriuria before ESWL is carried out should be treated with an appropriate antibiotic.[2] It should be stressed that the cephalosporins widely used for prevention of post-ESWL urosepsis[2] are not considered an effective treatment for enterococcal bacteria.[10]

Patients known to be at risk for infective endocarditis undergoing ESWL and invasive procedures of the urinary tract should receive prophylaxis based upon the American Heart Association guidelines,[5] although currently, most authorities do not include ESWL in their recommendations for infective endocarditis prophylaxis.[5] A recent review from France suggested that prophylaxis for infective endocarditis should be given before ESWL to cardiac patients at high risk (ie, prosthetic heart valves, previous history of endocarditis, or surgically constructed systemic pulmonary shunts or conduits).[11]

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