REVIEW ARTICLE

Drug abuse and kidney

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Abstract

Over the past 30 years, the number of drugs' dependents has increased. Drugs cause psychosomatic changes and ultimately death. The rapid increasing of illicit drug use is an important social health problem. Their use may be therapeutic under medical supervision or illegal by users in dependency. The majority of these substances or their metabolites are excreted through the kidneys and renal complications of drug abuse are frequently encountered. They include a wide range of glomerular, interstitial and vascular diseases. The damage may be acute and reversible or chronic and may lead to end stage renal failure. The involvement of the kidney in drug use is either attributed to their elimination through it, to a direct nephrotoxic effect, or through other mechanisms. Acute renal failure (ARF) can be caused by rhabdomyolysis, hypotension and dehydration or by the direct toxic effect of heroin, cocaine abuse, MDMA or volatile solutes use. Glomerulonephritis and nephrotic syndrome can be presented as focal glomerulosclerosis in heroin nephropathy and cocaine abuse, post infectious or associated to HBV, HIV or HCV infection nephropathy. Chronic parenteral drug users may develop secondary amyloidosis. Finally, drug abuse can lead to ESRD mainly by causing deterioration of pre-existing renal disease at a higher rate. In conclusion, significant alterations have been observed in the kidneys' structure since they participate in drug metabolism. There is lack of retrospective studies and information has been given from case reports. The continuation of substance abuse after the appearance of renal damage increases the risk of permanent renal disease and consequently may lead to end stage renal failure. Hippokratia 2011; 15 (Suppl 2): 4-8

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Knowledge of drug abuse dates back to ancient times. In the 3rd century BC, Arab traders of opium as well as the Aztecs were using hallucinogenic substances, particularly mushrooms around the same time[1]. Over the past 30 years, the number of drugs' dependents appears increased[2]. By 1997, 25% of the population reported use of drugs at least once in their life time. Drug abuse appears to be more common in middle social-economic class and in young men 25 to 29 years of age[3]. There are available data on drug use in the general population in Greece from the study conducted by the University of Mental Health (UMHRI), in 2004 (European Monitoring Centre for Drugs and Addiction). It seems that drug use in Greece rose significantly from 1984 to 2004. According to this study, 8.6% of the Greek population, aged 12 to 24 years, indicate to have experienced drug use, mainly cannabis. A study in 2006 indicates a rate of 17.4% having used drugs at least once (24% men and 14% women). The ESPAD study in 2007 involved high school students aged 14 to 16 years showed that 6% had tried marijuana or hashish, and 9% of the students reported use of inhalants[4]. The efforts of researchers to highlight addicted personalities of special predisposition have not yielded positive results, but in Strang J's report, a genetic predisposition on drug abuse seems to exist[2]. Drugs cause psychosomatic changes and ultimately death. Among Europeans aged 15-39 years, drug overdoses accounted 4% of all deaths[5]. The rapid increasing of illicit drug use is clearly an important social health problem.

Characteristics of drugs

Drugs are defined as natural or synthetic substances that are used for medical or recreational purposes and the repeated use leads to transient or chronic dependency. This behaviour of mental and physical dependence is described as "toxic addiction" or the recently used term "substance addiction". Drugs have toxic effects on human central nervous system; therefore, more correct is the term "toxic substances"[6]. According to the U.S. Justice Department, 33 pharmaceutical substances are classified in the group of drugs (Table 1). Their use may be therapeutic under medical supervision or illegal by users in dependency[7].

a) Heroin (diacetylmorphine, diamorphine) is the most commonly used drug of the opioids group. The intake may be through the nasal, gastrointestinal, respiratory, subcutaneous («skin popping»), or intravenous («mainlining») route. It is often injected in combination with cocaine («speed balling»)[8]. Heroin's half life is 3 minutes and is rapidly metabolized into morphine, which is mainly responsible for the pharmacological actions of heroin. Heroin is excreted in urine as free and unconjugated morphine. There are multiple renal complications from its abuse [9].

b) Cocaine is an alkaloid derived from a shrub (Erythroxylon) that grows in the Andes. It can be absorbed through

Amphetamines	Hydromorphone	Narcotics
Barbiturates	Inhalants	Opium
Benzodiazepines	K2	Oxycodone
Cannabis	Ketamine	Painkillers
Cocaine	Khat	PCP
Depressants	LSD	Peyote and Mescaline
Dextromethorphan (DXM)	Marijuana	Psilocybin
GHB	MDMA or Ecstasy	Rohypnol
Hallucinogens	Methadone	Salvia Divinorum
Heroin	Methamphetamine	Steroids (anabolic)
Hydrocodone	Morphine	Stimulants

Table 1: Pharmaceutical substances which are classified in the group of drugs according to the U.S. Justice Department (http://www.justice.gov/dea/concern/concern.htm)

any mucous membrane, smoked or injected, intravenous or intramuscular. It is estimated that it has a half-life of 30 to 90 minutes. A rate of 80 to 90% of cocaine is metabolized and the rest is excreted unchanged in urine, where metabolites can be detected for 36 to 48 hours[10]. c) Ecstasy (MDMA: 3, 4 - methylenedioxymethamphetamine), originally patented in 1914 as appetite suppressant, is a widely used recreational drug in the nightclubs of Europe during the so-called "rave" parties. It is generally taken orally. In the U.S.A., MDMA has not been taken as a dance drug and consequentially the spectrum of side effects is different with cardiac arrhythmias being more common[11]. The MDMA is rapidly absorbed, reaching maximum plasma concentration within approximately 2 hours[12]. It is metabolized by the liver and excreted by the kidneys. d) Temazepam and diazepam abuse is usually attributed to legitimate prescriptions or theft from pharmacies. Temazepam is now a controlled drug and can be taken individually or as part of a substances "cocktail". About 70% of injecting drug users has used temazepam at some time[13]. e) The mushroom species of Panaeolus muscaria and Psilocybe (including Psilocybe Semilanceata - «liberty cap», «magic mushrooms») are hallucinogenic if eaten[14]. They are not nephrotoxic themselves, however, proper identification of the mushroom is difficult and eating poisonous species is not uncommon. The Cortinarius mushrooms which contain the nephrotoxic agents of orellanine are not easily identifiable and can lead to kidney damage[15]. f) Deliberate inhalation of volatile solvents ("glue sniffing") was first appeared as a form of substance abuse in the early 1960's by inhaling glue used in model planes. The practice is diversified and includes the use of cement glue, aerosol paints, lacquers, solvents, typewriter correction fluid and fuel[16]. These products contain some volatile substances, including toluene, n-hexane, methyl ketones, chlorohydrocarbons and benzene. The euphoria induced by inhaling solvents is similar to alcohol intoxication. In addition, solvents can cause hallucinations, of short-term duration (15 to 30 minutes)[17] and may develop serious heart, lung, liver, neurological and renal complications, as well as sudden death[18].

Renal complications

Key property of drugs is their analgesic effect via the central nervous system. Consequently, this action has an impact on other functions, such as heart rate, breathing rate and blood pressure. The majority of these substances or their metabolites are excreted through the kidneys and renal complications of drug abuse are common. They include a wide range of glomerular, interstitial and vascular diseases. The damage may be acute and reversible or chronic and can leads to end stage renal failure. The involvement of the kidney in the use of drugs is either attributed to their elimination through the kidney, or a direct nephrotoxic effect, or through other mechanisms.

Acute renal failure

Coma caused by heroin overdose leads to muscle damage and rhabdomyolysis. Hypotension, hypoxia, acidosis and dehydration cause deterioration of renal function and development of acute renal failure. Grossman RA et al. indicate rhabdomyolysis in heroin users without the presence of coma, or evidence of muscle compression. They refer that this may be due to a direct toxic effect or an allergic reaction to heroin, or heroin additives flawed[19]. Also, acute or chronic cocaine use seems to be involved in acute renal failure which may occur as a result of rhabdomyolysis[20, 21]. Approximately 24% of patients examined in the emergency department with complaints related to cocaine, showed concentrations of creatine kinase over 1000 U / l[22]. A rate of almost one third of these patients developed acute renal failure[20, 23]. Cocaine can cause rhabdomyolysis through muscle ischemia caused by prolonged vasoconstriction of intramuscular arteries, by generalized convulsions and coma which leads to secondary compression of the muscles, or by direct damage to muscle fibres. Cocaine may be contaminated with arsenic, strychnine, amphetamines and phencyclidine. These substances could be responsible for convulsions and rhabdomyolysis. Acute renal failure due to massive infarction in both kidneys and accelerated atherosclerosis in kidney has been reported in drug users[24-27]. Acute renal failure may occur also to users of MDMA or other amphetamines and the main mechanism is rhabdomyolysis.

The patients usually present muscle pain and tenderness. Laboratory tests find an increase of creatinine and urea, potassium, phosphorus and creatine kinase. Myoglobin and granular casts are also detected in urine. Because of the frequency of acute renal failure, users are aware of the risk when dehydration coexists and often consume large quantities of water, so they may present hyponatremia and / or cerebral edema[28]. Hyponatremia on dilution due to excessive fluid intake can coexist with inappropriate antidiuretic hormone[29]. Moreover, there are reported cases of MDMA users with malignant hypertension and acute renal failure which is associated with intense sympathomimetic effects of MDMA[30]. Acute renal failure has been also described after intra-arterial injection of temazepam. Ischemia of the extremities is induced as a result of embolization particles and subsequent rhabdomyolysis and myoglobinuria[31]. Severe, but temporally dialysis depended, renal failure was present in 20% of temazepam users[32]. Oliguric acute renal failure may develop after ingestion of the mushroom Cortinarius within 5 to 12 days. In some patients, renal failure is transient[33], but in others may be permanent[34]. Acute kidney failure can also occur in users of volatile solutes due to acute tubular necrosis[35] or acute interstitial nephropathy[36] possibly due to toluene. Although there is no unanimity of opinion about the risk of health effects of smoking marijuana, there have been reported cases of patients with multisystemic involvement after intravenous administration of marijuana. The severity appears to be dose dependent. It includes fulminant toxic hepatitis, gastroenteritis, hypoalbuminemia, acute renal failure, electrolyte disturbances, leukocytosis, anemia, and relative thrombocytopenia[37].

Glomerulonephritis and nephrotic syndrome

The focal glomerulosclerosis is the predominant glomerular lesion in heroin nephropathy and increased mesangial matrix is considered a precursor of glomerulosclerosis, which seems to depend on the time of exposure to morphine[38]. Heroin can cause glomerulonephritis with many indirect mechanisms, such as immune-mediated in bacterial and fungal endocarditis caused mainly in intravenous use[39, 40]. There is a high rate of viral, bacterial and fungal infections associated with intravenous drug use, including heroin[41]. Thus, the occurring glomerulonephritis (GN) can be post-infectious. Local pyogenic abscesses by Staphylococcus aureus, have been associated with GN and this is due to deposition of immune complexes. Membranous glomerulonephritis due to HBV infection and mesangiocapillary glomerulonephritis due to cryoglobulinemia accompanying the HCV infection have also been described. Secondary (AA) amyloidosis has increased in frequency as a cause of renal disease in chronic drug users by parenteral route, especially among those who inject drugs subcutaneously («skin poppers»)[42,43]. Chronic use can lead to end stage renal failure. Nephrotic syndrome has been reported due to secondary amyloidosis in chronic drug users by parenteral route. Terminating the usage is the most effective therapy[44, 45]. Unfortunately, there is no experimental model that relates heroin with renal failure, but the heterogeneity of the response indicates different pathogenetic mechanisms. Also, there are no well-designed epidemiological studies providing information about heroin nephropathy[39]. In the 1970s and 1980s, nephropathy associated with heroin (HAN) was described. It is clinically shown as nephrotic syndrome and progresses rapidly to end stage renal failure. The process can be reversed with discontinuation of use[9]. The findings of renal biopsy usually present focal segmental glomerulosclerosis[46]. The pathogenesis is unclear. Heroin or any addition to its manipulation is considered to act as an antigen, leading to renal deposition of immune complexes[9]. Studies in animals have shown that morphine may have a direct effect on the glomerulus, causing proliferation of fibroblasts and reducing the degradation of collagen type IV. In North America, a reduction in the incidence of heroin nephropathy (HAN) among intravenous users has been described[47]. This is explained by the improvement of the quality of heroin supplied to addicts, thus exposed to lower doses of potentially nephrotoxic additional substances. Nowadays, nephropathy associated with the virus HIV (HIVAN) is diagnosed more frequently in heroin addicts[48]. The HIVAN is also presented with nephrotic syndrome and rapidly progressive renal failure and in some urban communities in the U.S., can cause up to 38% of end stage renal failure[49]. Renal biopsy usually reveals characteristically focal glomerulosclerosis of a glomerular collapse type (collapsing glomerular) with protrusion of epithelial cells. A recent publication incident with clinical and histological outcome of HIVAN after treatment with triple antiretroviral treatment and reduction of viral load supports the hypothesis that the virus has a direct cytotoxic action in the kidney [50]. Purpura glomerulonephritis with Henoch-Shonlein has also been described after using acetaminophen and codeine[51]. Severe renal failure has been reported in users of oxycodone while biopsy revealed by the electron microscopy, fiber depositions between the glomeruli and between the tubular basement membrane[52]. Immunologically, cocaine has been proved to increase the mesangial through the release of interleukin-6 by macrophages and evolves focal segmental glomerulosclerosis[53]. Administration of cocaine in experimental models has both non-specific lesions in the glomerulus and the interstitial tissue[39].Cases of renal scleroderma[54] Henoch-Schoenlain purpura[55], necrotizing vasculitis with multiorgan failure[56] and Goodpasture's syndrome[57] have been reported in cocaine users. Marijuana and cannabis do not seem to be implicated in glomerular injury but a de- novo posttransplant membranous GN in a chronic marijuana user after cadaveric kidney transplantation has been described[58]. The nephrotoxic action of volatile adhesives seems to be attributed to toluene[59]. Various renal lesions have been associated with its abuse. Microhematuria, pyuria and proteinuria[60], distal renal tubular acidosis and Fanconi syndrome, urinary stones[61], glomerulonephri-

tis[62], Goodpasture syndrome[63] have been described . The use of anabolic steroids can cause focal segmental glomerulosclerosis with proteinuria, either by hyperplasia or by mesangial direct nephrotoxic effect[64].

Chronic Renal Failure and Hypertension

Increasing numbers of African-Americans in urban centers, developing hypertensive end stage renal failure has been observed in recent years[65]. Forty-four per cent of these patients have a history of substance abuse, compared to a 5% of diabetics and 11% of patients with other causes of renal disease. However, a study of 301 chronic cocaine users showed no correlation with chronic hypertension or development of microalbuminuria[66]. It also seems that cocaine may cause deterioration of preexisting renal disease at a higher rate, rather than cause a de novo disease[67].

Conclusion:

Drug abuse is a major social problem of the modern world. The impact on the psychological and the organic sphere causes severe burden on social behavior and physical health in this population. Significant alterations have been observed in the kidneys' structure since they participate in drug metabolism. Glomerulus and interstitial injury has been found in case reports. Unfortunately there is a lack of an experimental model as well as an efficiently designed research plan for the drug users. The continuation of substance abuse after the appearance of renal damage increases the risk of permanent renal disease and consequently leads to end stage renal failure. Decreasing the number of users seems to be the best way in order to avoid renal complications.

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