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Drug induced hypertension – An unappreciated cause of secondary hypertension

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ABSTRACT

Most patients with hypertension have essential hypertension or well-known forms of secondary hypertension, such as renal disease, renal artery stenosis, or common endocrine diseases (hyperaldosteronism or pheochromocytoma). Physicians are less aware of drug induced hypertension. A variety of therapeutic agents or chemical substances may increase blood pressure. When a patient with well controlled hypertension is presented with acute blood pressure elevation, use of drug or chemical substance which increases blood pressure should be suspected. Drug-induced blood pressure increases are usually minor and short-lived, although rare hypertensive emergencies associated with use of certain drugs have been reported. Careful evaluation of prescription and non-prescription medications is crucial in the evaluation of the hypertensive individual and may obviate the need for expensive and unnecessary evaluations. Discontinuation of the offending agent will usually achieve adequate blood pressure control. When use of a chemical agent which increases blood pressure is mandatory, anti-hypertensive therapy may facilitate continued use of this agent.

We summarize the therapeutic agents or chemical substances that elevate blood pressure and their mechanisms of action.

1. Introduction

Most patients with high blood pressure have essential hypertension or well-known forms of secondary hypertension. Drug and chemical substances are frequently overlooked as a secondary cause of hypertension. Therefore, a comprehensive history including use of medications, over the counter agents and illegal substances should be elicited in every hypertensive individual. Identification of these substances may obviate the need for unnecessary, costly, and potentially dangerous evaluations, treatments, or both (Elliott, 2006; Grossman and Messerli, 1995; Rossi et al., 2011).

We recently reviewed the therapeutic agents and chemical substances that may elevate blood pressure (Grossman and Messerli, 2012). This manuscript updates our previous review on drug induced hypertension (Table 1). For some agents that are more commonly used we will review the available data.

2. Anti neoplastic agents

Several alkylating agents can increase blood pressure. In one serious 15 of 18 patients treated with multiple alkylating agents following autologous bone marrow transplantation developed hypertension (Grossman and Messerli, 2008). Hypertensive reactions associated with paclitaxel treatment have been reported (Solimando et al., 1996). Cis-diamminedichloroplatinium (CDDP) is an organic platinum compound with an antineoplastic effect. It has been demonstrated in four of five patients that intraarterial administration of CDDP produces sustained systemic hypertension. This complication has not been observed in patients receiving the drug by the intravenous route (Grossman and Messerli, 2008).

In the last years vascular endothelial growth factors (VEGF)

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inhibitors are being increasingly used for the treatment of various malignancies. This group of agents includes mainly monoclonal antibodies such as bevacizumab (Avastin), or orally-available small molecules that inhibit the tyrosine kinases stimulated by VEGF such as lapatinib (Tykerb), sunitinib (Sutent), sorafenib (Nexavar), axitinib, and pazopanib. These new drugs are most frequently involved with the development of hypertension (Milan et al., 2014). The development of hypertension is important in patients with malignancies because chemotherapy reduces cancer-related morbidity and mortality and patients are expected to live longer. Therefore, the impact of poorly controlled hypertension on cardiovascular morbidity and mortality may become a major issue. Bevacizumab (Avastin) is used to treat metastatic cancers of various origins. In clinical trials the development of moderate hypertension was more prevalent in patients treated with bevacizumab. The incidence of severe hypertension(blood pressure > 200/100 mmHg) was > 3- to 5-fold higher in the bevacizumab groups compared with placebo.(Hurwitz et al., 2004; Kozloff et al., 2009; Shih and Lindley, 2006) In the initial studies on bevacizumab, hypertension was reported in up to 32% of patients, but only 1% had life threatening hypertensive crisis (Izzedine et al., 2009). In a recent report describing the real-world association between baseline clinical characteristics, blood pressure response, and survival in patients prescribed anti-VEGF therapies., treatment-induced hypertensive response was identified in 49.7% of patients (Hamnvik et al., 2015). The absolute observed mean increase in blood pressure was 21 mmHg (systolic)/15 mmHg (diastolic), both in patients with and without preexisting hypertension. The incidence of hypertension was dose related and preexisting hypertension, old age (≥ 60 years), and overweight (≥ 25 kg/m^2) were risk factors for anti-VEGF therapy-induced blood pressure elevation (Hamnvik et al., 2015). In addition, it seems that blood pressure elevation associated with bevacizumab predicts a favorable response to treatment (Hamnvik et al., 2015).

Intravitreal bevacizumab injection is safe in terms of BP in both hypertensive and normotensive patients (Lee et al., 2009), however in one study 27 out of 768 patients (3.5%) reported a new episode of hypertension during intraocular injections of bevacizumab (Sheybani et al., 2009).

Sorafenib that is approved for advanced renal cell carcinoma and hepatocellular carcinoma can also increase blood pressure. In the Treatment Approaches in Renal Cancer Global Evaluation Trial (TARGET), treatment-related hypertension was reported in17% of the patients. Stage 2 hypertension was reported in 4% of the sorafenib treated patients compared to less than 1% in the controls (Escudier et al., 2007). Unlike the findings of the TARGET trial in the Sorafenib Hepatocellular Carcinoma Assessment Randomized Protocol (SHARP) patients with advanced hepatocellular carcinoma who were treated with sorafenib 400 mg BID did not develop more hypertension than the control group (Llovet et al., 2008). In a recent meta-analysis the relative risk for the development of hypertension was increased (relative risk [RR]: 2.93; 95% CI: 1.52–5.66) with the use of sorafenib compared with placebo and was independent on the tumor type and treatment regimen (Abdel-Rahman and Fouad, 2014a). Maitland et al. showed, by using 24 h ambulatory blood pressure monitoring, that sorafenib 400 mg BID increased systolic blood pressure by 8.2 mmHg and diastolic blood pressure by 6.5 mmHg within 24 h of treatment (Maitland et al., 2009).

Sunitinib (Sutent), an oral receptor tyrosine kinase inhibitor, has also been associated with hypertension (Zhu et al., 2009).

Hypertension should be considered as a class effect of all anti-angiogenic therapies and it seems that the risk of hypertension is similar with all agents(Abdel-Rahman and Fouad, 2014b; Wu et al., 2008). The use of VEGF signaling inhibitors is usually associated with mild blood pressure increase. However, it may be associated with severe hypertension, and Reversible Posterior Leukoencephalopathy Syndrome-a significant event likely secondary to hypertension (Levy et al., 2009). About 1% of all patients on anti-angiogenic therapy develop a hypertensive emergency.

The mechanism of elevated blood pressure in patients treated with anti-angiogenic agents is likely to be multifactorial and may include a decrease in nitric oxide (NO) production, loss of anti oxidative effect, and activation of the endothelin-1 system(Gonzalez-Pacheco et al., 2006; Hood et al., 1998; Kappers et al., 2010; Small et al., 2014). Recently, Thijs et al. showed that bevacizumab may increase peripheral resistance and blood pressure by reducing endothelium-mediated vasodilation (Thijs et al., 2013).

Several studies have suggested that hypertension may predict a beneficial response to antiangiogenics (Bono et al., 2009). Anti-VEGF therapy-associated hypertension is often transient and typically resolves with discontinuation of the provoking agent. Elevated blood pressure is usually easily controlled and the immediate risk of hypertensive target organ diseases is low in most patients. However, one should keep in mind that hypertensive crisis may occur, and therefore a close monitoring of blood pressure and early initiation of anti-hypertensive agents when necessary is recommended.

In an animal model Lankhorst et al. showed that the dual endothelin receptor antagonist-macitentan and the calcium channel blocker amlodipine can prevent sunitinib induced hypertension (Lankhorst et al., 2014).

Renin angiotensin system inhibitors, diuretics, beta blockers and calcium antagonists may be used to lower blood pressure. The nondihydropyridine calcium antagonists such as verapamil and diltiazem, are CYP3A4 inhibitors and nifedipine a dihydropyridine calcium antagonist has been shown to induce VEGF secretion, and therefore should be used with caution in combination with oral angiogenic inhibitors (Izzedine et al., 2009). Nitrates may increase the production of endogenous NO thereby facilitating blood pressure control (Dirix LYMaes and Sweldens, 2007). A favorable blood pressure response to sublingual test dose of 5 mg isosorbide dinitrate has been used to predict response to long acting nitrates (Dirix LYMaes and Sweldens, 2007).

3. Nonsteroidal anti-inflammatory drugs (NSAIDs) and analgesics

NSAIDs can produce a clinically significant increment in mean blood pressure of 5 mmHg (Johnson, 1997). While the mechanism involved in blood pressure increase remain speculative, salt and water retention coupled with increased total peripheral vascular resistance, via increased renal endothelin-1 synthesis may be the culprits (Johnson, 1997). NSAIDs diminish the effectiveness of some antihypertensive agents such as diuretics, beta-blockers and ACE inhibitors, but do not interfere with the action of calcium antagonists and central acting drugs (Krum et al., 2009; Morgan and Anderson, 2003; Singh et al., 2014).

NSAIDs vary considerably in their effect on BP. Armstrong and Malone found among the various NSAIDs, that indomethacin, naproxen, and piroxicam were associated with the greatest increase in blood pressure (Armstrong and Malone, 2003). Among the selective NSAIDs rofecoxib was more likely than celecoxib to raise systolic blood pressure (Armstrong and Malone, 2003). Two meta-analyses showed that selective COX-2 inhibitors increase blood pressure more than the nonselective agents (Aw et al., 2005; Chan et al., 2009). Unlike these findings Wang et al. (2007) showed that there were similar hazard rates of incident hypertension with celecoxib and nonselective NSAIDs users. Several studies showed that rofecoxib (which is now off the market) increased blood pressure more than celecoxib (Aw et al., 2005; Sowers et al., 2005).

Table 1
Different drug classes and their effect on blood pressure.

Drug	Clinical use	Notes
Anti cancer agents		
Anti vascular endothelial growth factor (VEGF) signaling	Anti cancer therapy	HTN should be considered as a class effect. The incidence of HTN is dose related and preexisting hypertension, old age ($\geq$ 60 years), and overweight ($\geq$ 25 kg/m^2) are risk factors for anti-VEGF therapy-induced BP elevation
Bevacizumab	Metastatic cancers of the colon, rectum, kidney, breast and glioblastoma multiforme	
Sorafenib	Approved for advanced renal cell carcinoma and hepatocellular carcinoma	
Sunitinib	Advanced gastrointestinal stromal tumor and renal cell carcinoma	
Alkylating agents	Antineoplastic agent	
Paclitaxel	Antineoplastic agent	
Cis-diamminedichloroplatinium	Antineoplastic agent	Only during intra-arterial administration
Analgesic, anti-inflammatory		
Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)	Analgesic, anti inflammatory	Mild, dose dependent increase in BP. Elderly patients, those with pre-existing hypertension, salt-sensitive patients, patients with renal failure and patients with renovascular HTN are at a higher risk to develop severe HTN. Calcium antagonists are the preferred choice of treatment
Acetaminophen	Analgesic	The effect of acetaminophen on BP is unclear
Psychiatric drugs		
Clozapine	Anti psychotic agent	
Venlafaxine	Antidepressive and anti anxiety agents	At dose above 300 mg/day
Monoamine oxidase inhibitors	antidepressive agents	Mainly with sympathomimetic amines and with certain food containing tyramine. Tranylcypromine is the most hazardous because of its stimulant action, whereas moclobemide and brofaromine are the least likely to induce hypertensive reaction
Tricyclic antidepressants	Antidepressive agent	More common in patients with panic disorders
Buspirone	Anxiolytic agent	Mild dose dependent increase in BP
Fluoxetine	Antidepressive agents	In combination with selegiline
Thioridazine hydrochloride	Psychotic and depressive disorders	Massive overdose may cause severe HTN
Carbamazepine	Bipolar depression and seizures	
Lithium	Manic depressive illness	Acute intoxication can cause severe HTN
Steroids		
Glucocorticoid	Replacement therapy, rheumatic disease collagen disease, dermatologic disease, allergic state, opthalmic disease, inflammatory bowel disease, respiratory disease, hematologic and neoplastic disease, nephropathies	HTN occurs more often in elderly patients and in patients with a positive family history of primary HTN. BP rise is dose-dependent and at low doses cortisol has less effect on BP
Mineralocorticoid		
Liquorice	A flavoring and sweetening agent	Dose dependent, sustained increase in BP characterized by hypokalemia, metabolic alkalosis and suppressed plasma renin activity and aldosterone levels
Carbenoxolone	Ulcer medication	
9-alpha fluoroprednisolone	Skin ointments, antihemorrhoid	
9-Alpha fluorocortisol	Cream	
Ketoconazole	Ophthalmic drops, and nasal sprays Anti mycotic	
Sex hormones Estrogen+Progesterone	Contraception, replacement therapy	Mild, sustained BP elevation, more common in premenopausal women. History of high BP during pregnancy, a family history of HTN, cigarette smoking, obesity, black, diabetes, and renal disease increase the risk of developing HTN. Severe HTN has been reported.
Androgens	Prostate cancer	Mild dose dependent sustained increase in systolic BP. Severe hypertension has been reported
Danazol (semisynthetic androgen)	Anabolic effect Endometriosis, hereditary angioedema	
Immunosuppressive agents		
Cyclosporine A	Immunosuppressive agent, prophylaxis of organ rejection, autoimmune disease,	Dose dependent mild to moderate increase in BP. Presence of HTN before transplantation, elevated creatinine levels and maintenance therapy with corticosteroids, increase the risk of HTN. Severe HTN has been reported

Table 1 (*continued*)

Drug	Clinical use	Notes
	dermatologic disorders	
Tacrolimus	Prophylaxis of organ rejection	Produces less HTN than cyclosporine A
Rapamycin	Prophylaxis of organ rejection	Produces little BP increase
Recombinant human erythropoietin	Anemia of renal failure and of some malignancies	Dose-related mild increasein BP. The risk to develop or worsen HTN is increased in the presence of pre-existing HTN, the presence of native kidneys, a genetic predisposition to HTN, when the initial hematocrit is low and when it increases rapidly. Hypertensive crisis with encephalopathy has been reported
Highly active antiretroviral therapy (HAART)	Anti HIV treatment	Recent studies reported that HTN was associated with traditional cardio metabolic risk factors and was unassociated with the treatment itself
Cocaine	Local anesthetics	Cocaine use is associated with acute but not chronic HTN. Transient severe increase in BP especially when used with β-blockers
Caffeine	Analgesia, vascular headache, beverages	The reaction to caffeine is more pronounced in males, in those with a positive family history of HTN and in African-American subjects. Caffeine may cause persistent BP effects in persons who are regular consumers, even when daily intake is at moderately high levels. Variability in the acute BP response may be partly explained by genetic polymorphisms of the adenosine A2A receptors and alpha(2)-adrenergic receptors.
Alcohol	Beverage	Dose dependent, sustained increase in BP. The BP effects of alcohol are independent from obesity, salt intake, cigarette smoking, and potassium intake.
Anti emetic drugs		
Metoclopramide	Anti emetic	
Alizapride	Anti emetic	
Prochlorperazine	Anti emetic	
Herbal products	Complementary and Alternative medicine	Mainly relate to dietary supplements that contain ephedra alkaloids
Miscellaneous		
Phenylephrine hydrochloride	Upper respiratory decongestant, ophthalmic drops	Dose dependent, sustained increase in BP.
Dipivalyl adrenaline hydrochloride	Opthalmic drops	Severe HTN has been reported
Epinephrine (with β blocker)	Local anesthetic, anaphylactic reaction, bronchodilatation, decongestant, anti hemorrhoidal treatment	
Phenylpropanolamine	Anoretic, upper respiratory decongestant	
Pseudoephedrine hydrochloride	Upper respiratory decongestant	
Tetrahydrozoline hydrochloride	Opthalmic vasoconstrictor drops	
Naphazoline hydrochloride	Opthalmic vasoconstrictor and nasal decdecongestant drops	
Oxymetazoline hydrochloride	Upper respiratory decongestant drops	
Ketamine hydrochloride	Anesthetic agent	Transient severe increase in BP has been reported
Fentanyl Citrate	Narcotic analgesic and anesthetic agent	
Smokeless tobacoo	Alternative to smoking	
Methylphenidate Demethylphenidate Amphetamine	Attention deficit hyperactivity disorder	
Yohimbine hydrochloride	Impotence	Acute, dose dependent increase in BP
Sibutramine	Weight loss	Mild increase in BP
Glucagon	Prevent bowel spasm	Only in patients with pheochromocytoma.
Selegiline	Used mainly for Parkinsons' disease	
Physostigmine	Reverse anticholinergic syndrome, myasthenia gravis, glaucoma, Alzheimer's disease,	
Ritodrine hydrochloride	Inhibition of pre-term labor	Hypertensive crisis has been reported
Disulfiram	Management of alcoholism	Slight increase in BP. Severe HTN may occur in alcoholic-induced liver disease
Lead	Industry	Also activates the sympathetic nervous system
Scopolamine	Pre-anaesthetic medication, Motion sickness	
Naloxone hydrochloride	Opioid overdose	Transient BP elevation
Cadmium	Industry	The association between cadmium exposure and HTN is equivocal
Arsenic	Industry	
Bromocriptine mesylate	Suppression of lactation, and prolactin inhibition in prolactinoma	Severe HTN with stroke has been reported following the use for suppression of lactation. Patients with pregnancy-induced HTN are at increased risk to develop HTN.
Amphotericin B	Fungal infections	

HTN=hypertension, BP=blood pressure.

Sowers et al. (2005) showed that at equally effective doses for osteoarthritis management, treatment with rofecoxib but not celecoxib or naproxen induced a significant increase in 24-h systolic blood pressure from 130.3 ± 1.2 to 134.5 ± 1.4 mm Hg. However, celecoxib may also increase blood pressure in a dose dependent way. In the studies that compared the efficacy and safety of celecoxib with placebo in reducing the rate of colorectal cancer, patients who received celecoxib 400 mg twice daily exhibited a 5.2 mmHg increase in systolic blood pressure after three years of treatment (Solomon et al., 2006). No change in blood pressure was observed in those who took the drug once daily or in the usual doses of 100–200 mg/d. Naproxcinod was developed as a NO-donating NSAID that upon absorption is rapidly cleaved to produce naproxen and a NO donating moiety. Preliminary studies showed that this agent has an anti-inflammatory effect and that it is superior to naproxen in terms of blood pressure elevation (Weber, 2009). The drug was not approved by the US Food and Drug Administration (FDA) and additional efficacy or safety data are required. Low dose aspirin has no effect on blood pressure control in hypertensive patients and may even lower blood pressure when taken at bedtime (Messerli, 2005). In patients who take NSAIDs, calcium antagonists would appear to be a preferred choice over other antihypertensive agents (White, 2007).

In hypertensive individuals, acetaminophen is recommended as the preferred analgesic since it does not raise blood pressure. Although several observational studies linked acetaminophen with a higher incidence of hypertension (Forman et al., 2007; Montgomery, 2008), old interventional studies failed to show the hypertensive effect of acetaminophen (Pavlicevic et al., 2008). In a more recent prospective study acetaminophen induced a significant increase in ambulatory blood pressure in patients with coronary artery disease (Sudano et al., 2010). It has been shown that acetaminophen slightly but significantly affects blood pressure response to ramipril, valsartan and aliskiren and therefore blood pressure should be followed closely in treated hypertensive patients treated with acetaminophen (Gualtierotti et al., 2013). Turtle et al. in a systematic review of the effect of acetaminophen on blood pressure concluded that the overall effect of acetaminophen on blood pressure is unclear (Turtle et al., 2013). The mechanism by which acetaminophen may increase blood pressure is not clear. It can be mediated by COX-2 inhibition, by central COX-3 inhibition (Hinz et al., 2008) or by an indirect activation of cannabinoid receptors (Bertolini et al., 2006).

4. Antidepressants

Venlafaxine hydrochloride is a serotonin/norepinephrine reuptake inhibitor that is used for the treatment of depression and anxiety. It can cause elevation of blood pressure probably through its noradrenergic mechanism. A large meta-analysis showed that blood pressure increase with venlafaxine is more pronounced in older patients and in men, and is dose dependent. The incidence of elevated diastolic blood pressure (> 90 mmHg) was statistically and clinically significant only at a dosages above 300 mg/day (Thase, 1998).

Several other antidepressant agents may also increase blood pressure by activating the sympathetic nervous system (Grossman and Messerli, 1995).

5. Glucocorticoids

Synthetic glucocorticoids can increase blood pressure in a dose-dependent fashion (Sholter and Armstrong, 2000). Glucocorticoid-induced hypertension occurs more often in elderly patients and in patients with a family history of essential hypertension (de Leeuw, 1997)

Hemodynamically, ACTH and adrenocortical steroids increase blood pressure through increasing cardiac output, with little change in peripheral resistance (Grossman and Messerli, 1995). The mechanism of glucocorticoid-induced hypertension remains uncertain and it seems to be multi-factorial. Discontinuation of steroid therapy usually leads to normalization of blood pressure. When steroid treatment cannot be interrupted, a diuretic is the drug of choice, since volume overload is the main mechanism by which steroids raise blood pressure. Addition of ACE-I or ARB may be required and careful monitoring of potassium is necessary.

6. Licorice-induced hypertension

Glycyrrhizic acid-the main active ingredient in licorice has a mineralocorticoid like activity by inhibition of the enzyme 11 β hydroxysteroid dehydrogenase 2. This enzyme inhibits the conversion of glucocorticoids to mineralocorticoids; therefore its inhibition enhances binding of steroids to mineralocorticoid receptors. Excess consumption of licorice may produce arterial hypertension characterized by increased exchangeable sodium and blood volume, hypokalemia with metabolic alkalosis, and suppressed plasma renin and aldosterone levels (Bisogni et al., 2014). Some other compounds may stimulate mineralocorticoid receptors and increase BP (Table 1).

7. Sex hormones

Hypertension develops in approximately 5% of women using compounds containing at least 50 μg of estrogen and 1–4 mg of progestin (Chasan-Taber et al., 1996). Although the increase is usually minimal, severe hypertensive episodes, including malignant hypertension, may occur. The risk of hypertension decreases quickly with cessation of oral contraceptives. No significant association between hypertension and use of progestin-only pills has been found over 2–4 years of follow-up (Hussain, 2004), but this matter has not been addressed by randomized trials. Recently it has been shown by 24H ambulatory blood pressure monitoring that combined hormonal contraceptive vaginal ring that releases 15- mcg ethinylestradiol and 120 mcg of etonogestrel each day, can also increase mean diastolic BP by 2.75 ± 5.13 mmHg (Cagnacci et al., 2013).

Postmenopausal hormonal replacement therapy has minimal if any effect on blood pressure in normotensive women (Affinito et al., 2001).

8. Immunosuppressive agents

The incidence of cyclosporine associated hypertension one year after renal transplantation ranges in different studies from 32.7% to as high as 81.6% (Ponticelli, 1993; Snanoudj et al., 2004).

In bone marrow transplants, Loughran et al. (1985) reported a 57% incidence of hypertension in cyclosporine treated patients, compared with a 4% incidence in methotrexate-treated patients. The frequency of cyclosporine associated hypertension in cardiac transplant recipients is approaching 100% and virtually all patients develop hypertension soon after transplantation (Grossman and Messerli, 1995).

Cyclosporine associated hypertension is also common in patients with autoimmune disease and in patients with psoriasis treated with cyclosporine (Grossman and Messerli, 1995). Cyclosporine associated hypertension is characterized by a disturbed

circadian rhythm with the absence or reversal of the normal nocturnal decrease in blood pressure (Cifkova and Hallen, 2001).

Several potential mechanisms may contribute to the development of cyclosporine associated hypertension. These include systemic and renal vasoconstriction, renal sodium retention, and nephrotoxic effects which has been documented in both animals and humans (Hoorn et al., 2012). The toxic effects of cyclosporine can be mediated by one or more of several mechanisms; stimulation of the renal sympathetic nervous system, imbalance in the production of renal vasoconstrictor eicosanoids and/or failure of vasodilatory prostaglandin synthesis, alteration of the renin-angiotensin-aldosterone system, and a direct vasoconstriction effect mediated by either stimulation of endothelin synthesis or interference with the production of endothelial-derived relaxation factor (Hoorn et al., 2012).

Blood pressure usually decreases after the withdrawal or substitution of cyclosporine immunosuppression but may not remit completely (Cifkova and Hallen, 2001). Calcium antagonists have been used successfully to lower blood pressure, but are known to increase cyclosporine blood levels (Rodicio, 2000). If necessary, cyclosporine can be continued and multidrug therapy should be used to control cyclosporine associated hypertension.

Tacrolimus, another immunosuppressive agent that inhibits calcineurin has also been associated with hypertension. However, it produces less blood pressure elevations than cyclosporin, and therefore conversion to tacrolimus may be considered in patients with cyclosporine associated hypertension. Rapamycin and mycophenolate mofetil are immunosuppressive agents, that do not inhibit calcineurin and produce little if any nephrotoxicity or hypertension (Manzia et al., 2005; Morales et al., 2001).

9. Recombinant human erythropoietin

Recombinant human erythropoietin (r-HuEPO) is effective in correcting the anemia of patients with end stage renal failure, and patients with malignancies. Hypertension has been reported to develop or worsen, in 20–30% of patients treated with recombinant human erythropoietin and it may appear as early as 2 weeks and as late as 4 months after the initiation of treatment (Grossman and Messerli, 1995). Erythropoiesis-stimulating agents increase blood pressure even in pre-dialysis patients(Suttorp et al., 2013). Hypertension is usually not a serious general problem in the recombinant human erythropoietin treated patient; however hypertensive crisis with encephalopathy has been reported (Novak et al., 2003).

Several potential mechanisms may explain how recombinant human erythropoietin may increase blood pressure in haemodialysis patients. They include increased blood viscosity, loss of hypoxic vasodilation, activation of neurohumoral systems (catecholamines, the renin-angiotensin system), and especially a direct vascular effect (Shimada et al., 2003).

The blood pressure can usually be controlled with a combination of fluid removal with dialysis and conventional antihypertensive therapy. If these measures are unsuccessful, the dose of recombinant human erythropoietin should be lowered or therapy should be withheld for several weeks. Phlebotomy of 500 mL of blood may rapidly lower blood pressure in refractory patients (Grossman and Messerli, 1995).

10. Anti retroviral treatment

Although Highly active antiretroviral therapy (HAART) has been reported to increase systolic blood pressure, recent studies reported that hypertension was associated with traditional cardio metabolic risk factors and was unassociated with the treatment itself (Hejazi et al., 2014; Krauskopf et al., 2013). Even when reported to be associated with hypertension, HAART does not usually increase blood pressure before 6 months of use (Grandominico and Fichtenbaum, 2008). The reaction to HAART is more pronounced in the elderly and in those with higher baseline systolic blood pressure, higher baseline cholesterol levels and low baseline CD4-cell count (Baekken et al., 2008). In a large cohort study hypertension was reported in 26.1% of the infected individuals (Glass et al., 2006).

Among 444 patients who initiated HAART, 83 exhibited an increase in systolic blood pressure of 10 mmHg or greater, 33 exhibited an increase in diastolic blood pressure of 10 mmHg or greater, and 11 patients had a new diagnosis of hypertension confirmed by antihypertensive treatment. Patients on lopinavir/ritonavir had the highest risk and patients receiving atazanavir had the lowest risk of developing elevated blood pressure. The effect of the treatment on blood pressure was mainly mediated through an increase in body mass index. The impact of antiretroviral medications on cardiovascular disease risk factors will increasingly influence treatment decisions (Crane et al., 2006).

Severe hypertension and renal atrophy as well as posterior reversible leukoencephalopathy syndrome have both been reported in association with use of the protease inhibitor indinavir (Cattelan et al., 2000). Hypertensive crisis secondary to phenylpropanolamine interacting with triple-drug therapy for human immunodeficiency virus prophylaxis has also been reported (Khurana et al., 1999). In addition potential drug interactions exist between antiretroviral medications, particularly the protease inhibitors, and calcium antagonists (Fichtenbaum and Gerber, 2002).

11. Cocaine

Cocaine intoxication and abuse is characterized by adrenergic over activity associated with increased blood pressure. Cocaine use is associated with acute but not chronic hypertension. In one small study isradipine significantly reduced cocaine-induce blood pressure elevation (Johnson et al., 2005).

12. Caffeine

Caffeine causes a pressor response due to increased sympathetic activity and antagonism of endogenous adenosine (Cohen and Townsend, 2006). Several investigators showed that caffeine may increase blood pressure levels (Savoca et al., 2005). Caffeine in 2–3 cups of coffee can acutely raise blood pressure by as much as 10 mm Hg in patients who are infrequently exposed to it, although the average response is an increase of about 4–5/3 mmHg (Cohen and Townsend, 2006). Noordzij et al. found that regular caffeine intake increases blood pressure, however when ingested through coffee, the blood pressure effect of caffeine is negligible (Noordzij et al., 2005). It has been recently suggested that variability in the acute blood pressure response to coffee may be partly explained by genetic polymorphisms of the adenosine A2A receptors and alpha(2)-adrenergic receptors (Renda et al., 2012). Of note, caffeine content of one cup of coffee can vary more than 10 fold (Bangalore et al., 2007). The conclusion from the literature is that caffeine/coffee can induce a brief acute increase in blood pressure, but there no evidence to support an association with chronic hypertension (Guessous et al., 2014).

13. Alcohol

Excessive alcohol use has clearly been shown to raise blood

pressure and can also cause resistance to antihypertensive therapy. Apart from the acute effects of alcohol, an increased prevalence of hypertension has been observed in heavy drinkers (Klatsky et al., 1977; MacMahon et al., 1984). In the Australian Risk Factor Prevalence Study (MacMahon et al., 1984) 7% of the prevalence of hypertension was attributed to alcohol consumption, whereas in the Kaiser–Permanente Study (Klatsky et al., 1977), the rate for men was 11 per cent. In a prospective cohort study of 3900 Japanese men, Yoshita et al. (2005) found that annual systolic blood pressure increase was greater in those who consumed 300 g/week or more alcohol corresponding to 13 glasses of wine (240 ml each), 13 bottles of beer (633 ml each), or 26 shots of whiskey (35 ml each) than non drinkers. A reasonable approach is to limit daily alcohol consumption to less than 30 g.

14. Salt containing medications

Exposure to sodium-containing formulations of effervescent, dispersible, and soluble medicines was found to be associated with significantly increased odds of hypertension (adjusted odds ratio of 7.18) (George et al., 2013) in a population based nested case-control study. In that study it was concluded that sodium-containing formulations should be prescribed with caution only if the perceived benefits outweigh these risks. Such formulations are frequently purchased over the counter. Whether use of such formulations directly leads to blood pressure elevation is unknown, but excessive use of these commonly used medications should be inquired for as they may influence blood pressure control.

15. Herbal products

Some herbs certainly have the potential to cause an increase in blood pressure, whereas others may aid in its control (Izzo et al., 2005). The evidence is anecdotal and therefore it is difficult to estimate the true incidence of these effects. Several reports have noted that dietary supplements that contain ephedra alkaloids can increase blood pressure (Haller and Benowitz, 2000). Some herbs can interfere with bioavailability of concurrently administered drugs (Awang and Fugh-Berman, 2002). Hypertension has also been reported after co-administration of ginkgo and a diuretic thiazide (Izzo et al., 2005).

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