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Mn(II) Complex of 1-(5-hydroxy-3-methyl-1-phenyl-1H-pyrazol-4-yl)octadecan-1-one: Synthesis and Anti-prostatic Hyperplasia Activity

C. E. Sokwaibe, I. E. Otuokere*

Department of Chemistry, Michael Okpara University of Agriculture, Umudike, Nigeria.

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ABSTRACT

Mn(II) complex of 1-(5-hydroxy-3-methyl-1-phenyl-1H-pyrazol-4-yl)octadecan-1-one (HMPPO) was synthesized by the reaction of HMPPO with $MnCl_2.4H_2O$. The color, yield, melting point, and conductance of the complex were determined. The complex was characterized based on elemental analysis, infrared spectrum, ^1H nuclear magnetic resonance (NMR), and ^{13}C NMR. The coordination sites of the HMPPO ligand were through C=O, C=N, OH, and H_2O. The molecular formula Mn(HMPPO).H_2O was proposed for the complex. A tetrahedral geometry was suggested for the complex. Furthermore, the biological activities of the Mn(HMPPO).H_2O against benign prostatic hyperplasia (BPH) cells were studied. Mn(HMPPO).H_2O reduced prostate-specific antigen (PSA) significantly ($p<0.05$) relative to the hormone control (HC) group. Mn(HMPPO).H_2O arrested the growth of prostatic cells in the test groups leading to a decreased secretion of prostatic acid phosphatase in the prostatic cells. There was a significant increase ($p<0.05$) in the relative prostate weight of "HC" group compared to Mn(HMPPO).H_2O. The result of this study showed that Mn(HMPPO).H_2O has an inhibitory effect on the development of BPH.

Key words: *Manganese, 1-(5-hydroxy-3-methyl-1-phenyl-1H-pyrazol-4-yl)octadecan-1-one, Prostatic hyperplasia, Synthesis.*

1. INTRODUCTION

In nature, many biological systems make an extensive use of metal ions, such as manganese, zinc, and copper, which play critical roles in the normal functioning of organisms [1]. Transition metals such as copper, iron, and manganese, among others, are involved in multiple biological processes, from electron transfer to catalysis to structural roles, and are frequently associated with the active sites of proteins and enzymes [1]. However, deregulation of some of these essential metals during normal biochemical processing has been implicated in the development of various pathological disorders, such as cancer [2]. These cellular functions only require the "trace metals" in minute but tightly regulated amounts. By comparison, other metals such as arsenic, cadmium, chromium, and nickel are less beneficial since they produce a wide range of toxic side effects, including carcinogenesis [1,3]. The field of medicinal inorganic chemistry encompasses, but is not limited to, the administration (or removal) of a metal ion into (or from) a biological system for either therapeutic or diagnostic purposes [4]. An important property of metals is that they form positively charged ions in aqueous solution that can bind to negatively charged biological molecules. Thus, the charge can be fine-tuned depending on the coordination environment involved, leading to the generation of a species that can be cationic, anionic, or neutral [1,3]. In addition, metal ions with high electron affinity can significantly polarize groups that are coordinated to them, fostering the generation of hydrolysis reactions [3]. Because of the shortcomings of the platinum-based drugs, a plethora of potential drugs containing manganese, copper, gold, iron, rhenium, rhodium, ruthenium, silver, and titanium have been explored [5]. All these metals are active against various types of tumors, some *in vitro* and others *in vivo*. In fact, complexes of titanium and ruthenium have made it into clinical trials and have showed some promising results for future use [5]. According to a research thesis [5], other metals such as molybdenum, osmium, iridium, and gallium have also exhibited toxicity toward cancer cells. These metal-based compounds have a range of activities from DNA interaction to "Trojan horse" clusters that house toxic compounds. In Lane's report [5], the metal compounds that have shown biological activity are metallocenes, which are thought to be redox active toward biomolecules [2].

*Corresponding Author:
E-mail: ifeanyiotuokere@gmail.com
Phone: +2347065297631

The redox potentials inside the cell are not known, and the mechanisms of these compounds are still under investigation. It is interesting to note that a derivative of ferrocene, ferroquine, is one of the metal complexes in the late stages of FDA testing (Phase III); it has been shown to be active against Plasmodium. Some of these ferrocene analogs have also been shown to cross the blood–brain barrier; something that most drugs cannot do [2]. Ruthenium compounds have also shown a promising activity that is dissimilar to cisplatin.

Benign prostatic hyperplasia (BPH) results from the enlargement of the prostate gland due to the proliferation of prostate stoma and epithelium in the transitional zone [6]. This proliferation causes lower urinary tract symptoms such as difficulty in urination or decrease in maximum flow rate, increase in residual urine volume in the bladder, and increase in prostate size [7]. A number of biologically active molecules, including steroid hormones, have been implicated in the reactions that lead to prostatic enlargement. In addition, Wang *et al*. [8] in their study, "A cell-based screen for anticancer activity of 13 pyrazolone derivatives," evaluated the cytotoxic activity of a series of pyrazolone derivatives against four human tumor cell lines including HepG2 (liver heptacellular cells), ovarian carcinoma cell line), KB, and multidrug resistance KBv200 cell line *in vitro* and *in vivo*. Of the 13 compounds screened, 3 compounds presented remarkable anticancer effects. Furthermore, these three compounds effectively inhibited the tumor growth of KBv200 cell xenografts *in vivo*. We, at this moment, present, Mn(II) complex of 1-(5-hydroxy-3-methyl-1-phenyl-1H-pyrazol-4-yl)octadecan-1-one (HMPPO): Synthesis and anti-prostatic hyperplasia activity.

2. EXPERIMENTAL

2.1. Chemical and Solvents

The chemicals and solvents used in this study were of analytical grade obtained from Sigma-Aldrich Chemical Company without further purification. The chemicals are β-estradiol-17-valerate and 5α-androstan-17β-ol-3-one (hormone), finasteride (anti-prostatic drug), manganese (II) chloride tetrahydrate, 3-methyl-1-phenylpyrazol-5-one, octadecanoyl chloride, and calcium hydroxide. The solvents are ethanol, methanol, toluene, benzene, acetonitrile, tetrahydrofuran (THF), dimethysulfoxide (DMSO), N,N'-dimethylformamide (DMF), acetone, diethylether, and petroleum ether.

2.2. Physical Measurements

The melting point of the complex was determined using Gallenkamp melting point apparatus. The molar conductance of the complex was determined in either DMF or DMSO using Jenway peMJ conductivity meter, with a cell constant of 1.05. Atomic absorption spectroscopy and elemental analysis were carried out on Varian AA spectrometer (AA240FS). About 0.2 g of complex was digested with 2.0 cm^3 of 50% of water/nitric acid. The digest was rinsed quantitatively into a 100 ml standard flask and made up to the mark with deionized water. The elemental analysis for C, H, and N was obtained using a Perkin-Elmer 240B elemental analysis instrument. Infrared (IR) spectrum was collected on Perkin Elmer Paragon 1000 Fourier transform IR spectrophotometer equipped with caesium iodide windo~ (4000-250 cm^{-1}) as KBr pellets. The proton and carbon-13 nuclear magnetic resonance (NMR) spectra were obtained on a Bruker Avance III HD spectrometer operated at a frequency of 600 MHz in Laboratory F22 of the Department of Chemistry, Rhodes University, South Africa. DMSO was used as the solvent.

2.3. Synthesis of the Manganese Complex of HMPPO

HMPPO ligand was synthesized as described in a previous publication [9]. The complexes were prepared following the reported procedure [10,11]. Mn(II) solutions were prepared by dissolving 5 g (0.025 mole) of $MnCl_2.4H_2O$ in 100 ml of deionized water with warming at 90°C. The solution was added to 22 g (0.025 mole) of HMPPO ligand (complexing agent) in 100 ml ethanol solution at 70°C. The complex was allowed to cool and crystals that appeared were washed, recrystallized from aqueous ethanol (1:1), filtered, and dried in a desiccator.

2.4. Mn(II) HMPPO

MP: 191-94°C. IR (KBr): 3483.56, 3551.07, 3068.55, 2854.74, 1606.76, 680.89, 611.45, 1705.13, 530.44, 572.88 cm^{-1}. ^1H NMR (400 MHz, D_2O): 7.33-7.72 (3H, t, Ar), 0.82-1.17 (m). ^{13}C NMR (100 MHz DMSO-d_6): 119.02-148.98 (Ph C), 14.01 (CH3), 17.92 (CH2), 194.02 (C=O). Analytical calculated for $MnC_{28}H_{45}N_2O_3$: C, 65.63; H, 8.79; N, 5.47; Mn, 10.74. Found C, 65.20; H, 8.63, N, 5.30; Mn, 10.72 products yield: 63%.

2.5. Biological activity

2.5.1. Animal housing

A total of 15 male Albino rats having an average weight of about 104.12 g each were purchased from an animal breeding unit in the department of Veterinary Pharmacology, University of Nigeria, Nsukka. The rats were housed in the standard steel cages with a plastic base and acclimatized for 7 days under humid tropical condition in the animal house of the College of Natural and Applied Science, Department of Biochemistry, Michael Okpara University of Agriculture, Umudike, Abia State. The rats were exposed to 12 h light/dark cycle and were given free access to clean tap water and commercial rat chow purchased from Vital Feeds Ltd., Nigeria.

2.5.2. Preparation of the hormone

This was prepared following the outlined literature [12]. About 250 mg dihydrotestosterone

and 25 mg estradiol valerate were dissolved in 25 ml olive oil and administered intravenously. The dose of the complex [Mn.HMPPO.H_2O] was formulated as follows; 0.125 g of the complex was dissolved in 25 ml of olive oil to yield a stock of 5 mg/ml.

2.6. Experimental Design

After 7 days of acclimatization, the experimental animals were randomly assigned to five experimental groups of three rats each. The groups were labeled as follows:

Group 1 – Standard test control group; received no hormone treatment, no HMPPO, and no [Mn.HMPPO.H_2O]n administration, but was fed normal diet for 28 days. This group served as the standard control group.

Group 2 – HMPPO group; received subcutaneous injection of the hormone and 5 mg ml^{-1} oral administration of the ligand HMPPO every day for 28 days.

Group 3 – Finasteride group; received subcutaneous injection of the hormone and oral administration of finasteride every day for 28 days. Finasteride was used in this analysis because it is a known drug for the treatment of BPH.

Group 4 – Mn.HMPPO group; received subcutaneous injection of the hormone every day and 5 mg/ml oral administration of [Mn(HMPPO).H_2O]n every day for 28 days. This group served as test group 1.

Group 5 – Hormone control group (HC group); received subcutaneous injection of the hormone every day with no oral administration of the ligand and the metal complex for 28 days. This group served as HC group.

2.7. Sample Collection

After 28 days, all the rats were weighed before they were sacrificed by cardiac puncture after dazing with a cervical blow. They were bled exhaustively. Serum collection was done according to the method reported in literature [13]. Whole blood was collected in a vacutainer. The blood was allowed to clot by leaving it undisturbed at room temperature for 20 min. The clot was removed by centrifuging at 2000 rpm for 5 min in a refrigerated centrifuge. The liquid component (serum) was transferred into a clean polypropylene tube using a Pasteur pipette. Vital organs for the study were excised and weighed.

Prostate-specific antigen (PSA) and prostate acid phosphate (PACP) analysis were done following the procedure outlined in literature [14]. Relative organ weight determination was calculated as the ratio of the excised organ and the final weight of each rat and the mean taken for each group. Statistical analysis results were expressed by one way analysis of variance (ANOVA) using statistical package for social sciences (SPSS) 17 software.

3. RESULTS AND DISCUSSION

A graph of serum PSA concentration in rats for the different groups is shown in Figure 1a. Serum PACP activity concentration in rats for the different groups is shown in Figure 1b. Mean prostatic weight graph in rats for the different groups is shown in Figure 1c.

The elemental analysis data shows that the metal complex was prepared. The result of the elemental analysis was in good agreement with the calculated values of 1:1 metal to ligand combination. This is as a result of the close percentage values of the carbon, hydrogen, nitrogen, and the metal obtained to those calculated from the formulae of the compounds. This shows that in aqueous solution, the mode of interactions

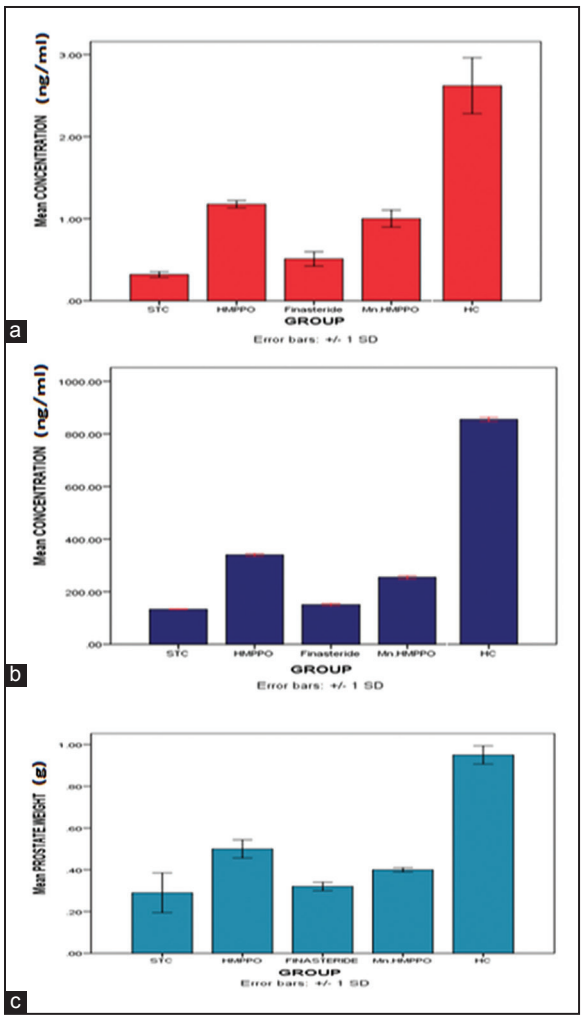

Figure 1: (a) Serum prostate-specific antigen concentration in rats for the different groups, (b) serum prostatic acid phosphatase activity concentration in rats for the different groups, (c) mean prostatic weight graph in rats for the different groups.

between the Mn(II) ion and the HMPPO is in the mole ratio of 1:1. The conductivity measurements of the metal complex in acetone (10^{-3} M) gave 7.2 Siemens mol^{-1} cm^{-1}. This value is lower than 30 Siemens mol^{-1} cm^{-1}, showing that it is a non-ionic compound [14,15]. The lower the conductivity value, the more the covalent. The solubility data showed that the complex is hydrophobic. The complex was insoluble in water. The complex showed high degrees of solubility in ethylacetate, acetone, DMSO, THF, dioxane, and pyridine. These solvents have lone pairs of electrons, which they must have donated to the complexes to complete the octahedron in the complexes, thereby reducing further the ionic character of the complex, thus an increase in the solubility of the complexes in those solvents.

3.1. IR Spectra

Broadbands centered between 3483.56 and 3551.07 cm^{-1} in the IR of the complex have been assigned to the νOH of the adduct water molecules coordinated to the central metal ion or residing in the crystal lattice of the complexes [16]. The νC=O stretching bands of HMPPO were observed as a very strong peak at 1743 cm^{-1} [9], but that of Mn(HMPPO).H_2O was shifted upfield (1705 cm^{-1}). This observation indicates the involvement of the C=O group in the chelation process, hence the formation of C=O→M bonding system. In addition, the C=N band of HMPPO was observed as a strong peak at 1618.33 cm^{-1} [9], but those of the complex was shifted to 1606.76 cm^{-1}, this indicates the involvement of the C=N in bonding system. The metal complex showed strong absorption bands around 1151.54-1228.70 cm^{-1} region. This has been assigned to C-H vibration frequency resulting from bending vibrations in the molecule. The vibration frequency modes observed at 1064.74 cm^{-1} have been assigned to C-H in-plane deformation of the phenyl ring in the complex. The comparison of the spectra of the ligand with those of the metal complex showed that there is little or no shift in the above frequencies, indicating that the π-system of the unsubstituted phenyl ring of the free ligand moiety is not involved in the coordination with Mn(II) ions. Vibration frequency 1300.07-1452.54 cm^{-1} has been assigned to $–CH_3$ bending vibration. These values are present both in HMPPO [9] and Mn(II) complexes. The presence of bands between 600 and 400 cm^{-1}, which are typical of 1,3-diketonates, has been suggested as due to bonding to metals through the oxygen atom of the ligand [11]. These weak bands in the IR spectra of Mn(HMPPO).H_2O complex appeared at 530.44 and 572.88 cm^{-1}. This band is absent in the IR of the ligand [9].

In our previous publication [9], the ligand HMPPO showed ^1H NMR resonance peak at 3.65 ppm for –OH group. This shows that the enolic form of the ligand was prepared. This chemical shift was absent in the spectra of the Mn(II) complex, indicating complexation via deprotonation of the enolic hydrogen of the ligands.

The signals of methyl and methylene protons were observed as multiplets at 0.82-1.17 ppm for the Mn(II) complex. On the other hand, the signals of the phenyl protons were observed at chemical shifts 7.33, 7.72 ppm.

From our previous publication [9], analyses of the ^{13}C NMR spectral data indicated that the carbon resonance spectra of C=O was observed at a chemical shift value of 174.50 ppm in HMPPO ligand. This functional group shifted downfield (194.02 ppm) in Mn(II) complex. This suggested the involvement of C=O in complexation. The aromatic carbons were observed at 119.53-148.98 ppm. The carbon atoms of methyl and methylene were observed at 14.01-17.92 ppm.

Based on the elemental analyses, IR spectrum, and NMR characterization, the complexes conformed to the general molecular formula $ML_n.xH_2O$, where n is the number of the ligand, L being the ligand HMPPO, and x is the number of water molecules in all the complexes. The proposed structure of the complex is shown in Figure 2.

3.2. Biological Activity

PSA is a glycoprotein localized within the prostate gland [17]. It is found in cytoplasmic granules and vesicles, the endoplasmic reticulum, vacuoles and secretory granules, and in lysosomal dense bodies in columnar or cuboidal epithelium of acini but not in acinar cells. It appears to belong to the *Kallikrein* family of serine proteases [18], with 57% structural similarity. It is 93% protein and 7% carbohydrate of mainly hexose, hexoseamine, and sialic acid [19]. PSA is believed to be synthesized in the rough endoplasmic reticulum, stored in the vesicle and vacuole, and released in the glandular lumina by exocytosis [20]. Because of its tissue specificity, PSA is used as a marker for BPH and prostate cancer. PSA is a single

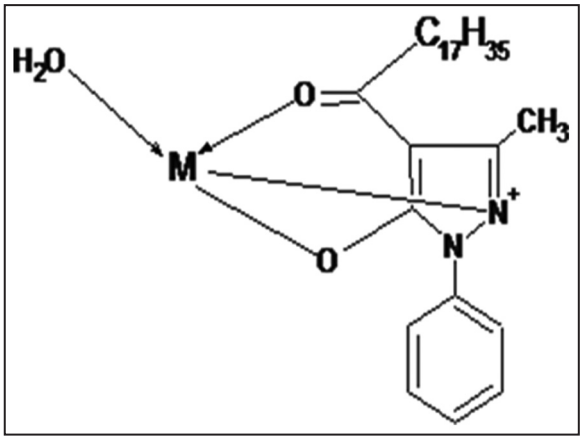

Figure 2: Proposed structure of [Mn(HMPPO).H_2O] complex where M=Mn.

polypeptide and occurs both in normal and malignant prostatic tissues and in the gland of men with BPH, but not in any other human tissue [20]. PSA is a secretory product of prostatic cells and will increase when the prostatic cells increase in number. PSA value can be used to predict the enlargement of the prostate because high PSA value correlates with an increase in prostate mass [7].

PACP could be used as a marker to detect prostate disorders in human or animals [21]. However, the discovery of PSA has resulted in a shift from PACP to PSA. Elevated PACP level in animals treated with DHT and estradiol have been reported, [22] and this may be due to hyperplasia of the prostate gland. Together with PSA, PACP can give useful information about prostatic disease, especially BPH prostate cancer.

The result shows a significant increase ($p<0.05$) in PSA value of "HC" group receiving only hormonal induction with neither the ligand nor the complex administration relative to all the other groups. However, there was no significant difference ($p>0.05$) between the groups receiving HMPPO and [Mn.HMPPO.H_2O]. Serum prostatic acid phosphatase activity showed a significant increase ($p<0.05$) in "HC" group receiving only hormonal induction with neither the ligand nor the complex administration relative to all the other groups. There was no significant difference ($p>0.05$) between HMPPO and [Mn.HMPPO.H_2O]. Relative prostate weight was significantly increased ($p<0.05$) in "HC" group compared to the other groups. There was no significant difference ($p>0.05$) between HMPPO and [Mn.HMPPO.H_2O].

4. CONCLUSION

The analytical data showed that the compositions or stoichiometries of the complex formed between the ligand and the metal ions are in the mole ratios of 1:1, with a molecule of water of crystallization from aqueous solution. The complex conforms to the general molecular formula [Mn.HMPPO.H_2O]. The conductivity measurements of the metal complex in acetone (10^{-3} M) gave value lower than 30 Siemens mol^{-1} cm^{-1}, indicating that it is a non-ionic compound. PSA, PACP, and prostate size are important markers for benign prostate hyperplasia. The result of this study showed that the complex has an inhibitory effect on the development of BPH, since the complex administered to the rats resulted in a marked difference in both PSA and PACP values and prostate size compared to group "HC" without [Mn.HMPPO.H_2O].

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***Bibliographical Sketch**

Mr. Sokwaibe Chinweotuto, E, is from Okwu village in Elu-Elu Old Umuahia Autonomous community in Umuahia South local Government of Abia State, Nigeria. He obtained a B.Sc. Hons Degree in Pure and Industrial Chemistry in 2009 at Nnamdi Azikiwe University, Awka. A professional Diploma in Education from Alvan Ikoku Federal College of Education, Owerri, Nigeria in 2015 and an M.Sc degree in Inorganic Chemistry from Michael Okpara University of Agriculture, Umudike, Nigeria. He has contributed so much to the society through journal publications and has also co-authored some text books in both Physical and General Chemistry. He is currently a lecturer in the Department of Chemistry, Alvan Ikoku Federal College of Education, Owerri, Nigeria.